Chapter one

Introduction

1.1 Introduction

Medical imaging is the technique, process and art of creating visual representations of the interior of a body for clinical analysis and medical intervention. Medical imaging seeks to reveal internal structures hidden by the skin and bones, as well as to diagnose and treat disease.(Dhawan , 2003).

Medical ultrasonography uses high frequency broadband sound waves in the megahertz range that are reflected by tissue to varying degrees to produce (up to 3D) images. This is commonly associated with imaging the fetus in pregnant women. Uses of ultrasound are much broader, however. Other important uses include imaging the abdominal organs, heart, breast, muscles, tendons, arteries and veins. While it may provide less anatomical detail than techniques such as Computerize Tomography (CT) or Magnetic Resonance Imaging (MRI), it has several advantages which make it ideal in numerous situations, in particular that it studies the function of moving structures in real-time, emits no ionizing radiation, and contains speckle that can be used in elastography. Ultrasound is also used as a popular research tool for capturing raw data, that can be made available through an ultrasound research interface, for the purpose of tissue characterization and implementation of new image processing techniques. The concepts of ultrasound differ from other medical imaging modalities in the fact that it is operated by the transmission and receipt of sound waves. The high frequency sound waves are sent into the tissue and depending on the composition of the different tissues; the signal will be attenuated and returned at separate intervals. A path of reflected sound waves in a multilayered structure can

be defined by an input acoustic impedance (ultrasound sound wave) and the Reflection and transmission coefficients of the relative structures. It is very safe to use and does not appear to cause any adverse effects. It is also relatively inexpensive and quick to perform. Ultrasound scanners can be taken to critically ill patients in intensive care units, avoiding the danger caused while moving the patient to the radiology department. The real time moving image obtained can be used to guide drainage and biopsy procedures. Doppler capabilities on modern scanners allow the blood flow in arteries and veins to be assessed.(Dhawan , (2003).

Diagnostic sonography (ultrasonography) is an ultrasound-based diagnostic imaging technique used for visualizing internal body structures including tendons, muscles, joints, vessels and internal organs for possible pathology or lesions. In physics, 'ultrasound' refers to sound waves with a frequency too high for humans to hear. Ultrasound images (sonograms) are made by sending a pulse of ultrasound into tissue using an ultrasound transducer (probe). The sound reflects and echoes off parts of the tissue; this echo is recorded and displayed as an image to the operator. Many different types of images can be formed using ultrasound. The most well-known type is a B-mode image, which displays a two-dimensional cross-section of the tissue being imaged. Other types of image can display blood flow, motion of tissue over time, the location of blood, the presence of specific molecules, the stiffness of tissue, or the anatomy of a three-dimensional region. Compared to other prominent methods of medical imaging, ultrasonography has several advantages. It provides images in real-time (rather than after an acquisition or processing delay), it is portable and can be brought to a sick patient's bedside, it is substantially lower in cost, and it does not use harmful ionizing radiation.(Ellis,et al, 2000).

Sonography can be enhanced with Doppler measurements, which employ the Doppler effect to assess whether structures (usually blood) are moving towards or away from the probe, and its relative velocity. By calculating the frequency shift of a particular sample volume, for example flow in an artery or a jet of blood flow over a heart valve, its speed and direction can be determined and visualised. This is particularly useful in cardiovascular studies (sonography of the vascular system and heart) and essential in many areas such as determining reverse blood flow in the liver vasculature in portal hypertension. The Doppler information is displayed graphically using spectral Doppler, or as an image using color Doppler (directional Doppler) or power Doppler (non directional Doppler). This Doppler shift falls in the audible range and is often presented audibly using stereo speakers: this produces a very distinctive, although synthetic, pulsating sound. *(*Bose,et al,2009).

Doppler ultrasound has become increasingly important in investigating abdominal vascular disease. Both the coeliac and superior mesenteric vessels have been studied in detail in response to physiological stimuli such as feeding and exercise. Dubbins has reported its use in diagnosing renal artery stenosis, and a few anecdotal reports have discussed the potential role of ultrasound in investigating both superior mesenteric artery and coeliac artery stenosis. (Dhawan P, A, 2003).

At ultrasound scanning combined with the pulsed Doppler technique we can non-invasively monitor alterations in blood flow velocity**.** (Ellis,et al, (2000).

Duplex ultrasound (DU) provides a simple, portable, reproducible, and non-invasive assessment of blood flow. Measurement of luminal diameter and blood velocity allows estimation of blood flow and peripheral

resistance, and the detection of arterial occlusive disease. This method has gained widespread acceptance as a reliable tool in many vascular beds such as the carotid, aorto-iliac, periopheral limb, and renal functions. Owing to its size and anatomic position, the superior mesenteric artery (SMA) is also accessible to DU (Ellis,et al, 2000). Applied to the superior mesenteric artery (SMA), the method confirms that blood flow in this vessel increases in response to a meal. The vasoactive components seem to be influenced by the digestive products of the diet, so that the effect of a meal on splanchnic blood flow may depend on the intraluminal digestion. The intake of food results in an increase in superior mesenteric artery blood flow (SMABF) in man after liquid meals. The relative contribution of different food components to the postprandial mesenteric blood flow has, however, not been thoroughly investigated up to now in humans. There are extensive data from animal experiments. From these, it is clear that the hydrolytic products of food digestion are primarily responsible for the food induced hyperaemia (Perko, 20).

1.2 Study problem:

Superior Mesenteric Artery (SMA) is one of the important arteries in the gastrointestinal tract that supplies the duodenum, whole of the small bowel, and the right half of the colon by blood flow which is reflects motor, secretary, and absorptive activities. All of these activities increase after a meal and, consequently, induce a great increase in (SMA) circulation to provides nutrients and oxygen to its supplying organs, carries away the absorbed substances, and removes waste products from these organs. Due to ingestion of meal, there was changing in blood supply of SMA which is associated with changing in diameter and blood volume of SMA. These changes was varies according to the type of meal

that was ingested. The alteration in SMA from fasting to postprandial state may affect in the activities of that organs supplied by SMA which may stimulate problems and induce pathology. The estimation blood volume during fasting and postprandial status it clarify status of estimation of pathology.

1.3 Importance of the study:

- There are no data or recent studies was done in the effects of meals on SMA in the human in any Arab countries as far as researcher Knew, in spite of spread of bad dietary habits in these countries. So the conducted study may clarify the effect of meals on SMA in healthy human subjects.
- Increasing in Gastro Intestinal Tract (GIT) vascular disorders which is associated with bad dietary habits.

1.4 Objectives of the study

1.4.1 General objective

The main objective is to study SMA for intake of different types of meals, in increased blood flow in the superior mesenteric artery and determine the relative potencies of the major nutrient stimuli in healthy human subjects.

1.4.2 Specific objectives

- To determine the change in diameter of SMA postprandially.
- To evaluate the variation in blood velocity and blood volume in SMA according to different types of meals.
- To correlate between the blood flow indices and time taken after meal

Chapter two

Background and previous study

2.1 Anatomy and Physiology of Vessels

Arteries and veins have walls made of three principal layers. Each layer is called a coat or tunic. The outer layer (tunica externa or adventitia) consists of strong connective tissue which acts as a specular reflector (a broad, smooth echogenic interface). The middle layer (tunica media) consists of smooth muscle and elastic fibers. The muscle contractions propel the blood along the vessel; whereas, the elastic fibers enable vessel expansion due to surges in blood pressure. The inner layer (tunica intima) consists primarily of epithelial tissue which acts as a smooth lining so blood will encounter the least possible resistance to blood flow. Although arteries and veins have the same three layers, each layer is thicker in the arteries. (Buirwin,2002)

Blood flow in an artery is faster than in a vein since the flow is dependent upon the strength of the heart's contractions. Arteries have thicker muscle layers and can propel blood more effectively than the thinner walled veins. Blood flow is reduced whenever it has to flow against the influence of gravity; conversely, it is increased whenever it flows with the forces of gravity. This is the principle behind lowering a person's head between their knees whenever the patient feels faint. Blood flow is increased to their brain in this position. The empty atria of the heart provide a vacuum that also pulls the venous blood toward the heart. Arteries and veins are influenced by the actions of the heart. Real time imaging enables the viewer to see the sharp arterial pulsations and to differentiate these slight reductions in lumen diameter from the wavy pulsing action of veins as their lumen open and close. Arteries are not influenced by respiration

since the arterial blood is under high pressure and the arterial walls are thick and strong, thus enabling the vessel to withstand any alterations in intra abdominal pressure caused by breathing. (Buirwin,2002)

2.2 Superior Mesenteric Artery(SMA) Anatomy

2.2.1 The superior mesenteric artery:

The superior mesenteric is a large artery that springs from the front of the abdominal aorta opposite the first lumbar vertebra about a quarter of an inch below the celiac artery. (Reuter S R, et al, 1986). It originates from the aorta approximately 1 cm below the coeliac trunk, at the level of the L1/2 intervertebral disc **(Fig. 2:1)**. It lies posterior to the splenic vein and the body of the pancreas, and is separated from the aorta by the left renal vein. (Gray's Anatomy,2008). It descends to the front of the third part of the duodenum, where it enters the mesentery; and then, in the root of the mesentery, it runs downward and to the right with a slight curve whose convexity is toward the left; and it terminates in the right iliac fossa, near the end of the ileum, by anastomosing with a branch of the ileo-colic arter. (Reuter, et al, 1986).

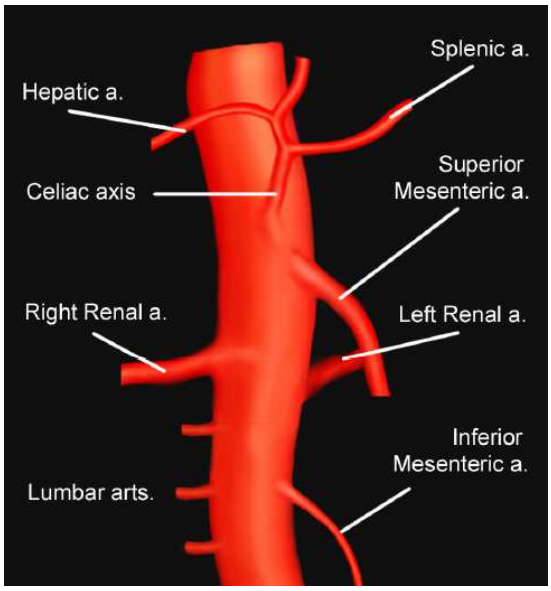


Fig. 2.1: showed the superior mesenteric artery originates from the aorta (Reuter et al 1986) .

2.2.2 Mesenteric Arteries supply:

The mesenteric arteries supply blood to the large and small intestines. The SMA supplies blood to the whole length of the small intestine, the cecum, the ascending colon and about one-half of the transverse colon. The Inferior Mesenteric Artery (IMA) arises approximately 3 cm cephalad to the aortic bifurcation and supplies the left half of the transverse colon, the descending and iliac portions of the colon, the sigmoid colon, and the most of the rectum. Because the distal vascular bed being supplied by the mesenteric arteries is gut, the perfusional requirements vary based on whether the intestines are fasted or fed. In the fasting state, when the gut is metabolically quiescent, the normal pattern of blood flow is characterized by high resistance; only enough blood is needed to maintain viability of the intestines themselves. After eating (or after a high-calorie meal in the vascular testing setting), the flow pattern changes to one of lower resistance **(Fig:2.2)**. The intestines areactively processing nutrients in the gastrointestinal tract and, therefore, they need more blood to support the digestive process. The additional blood volume delivered to the gut also allows for more efficient transport of nutrients away from the intestines to the other parts of the body. Spectral waveforms obtained from the mesenteric arteries reflect the varying resistivity present in the fasted or fed gut.(James &. Baun, 2010).

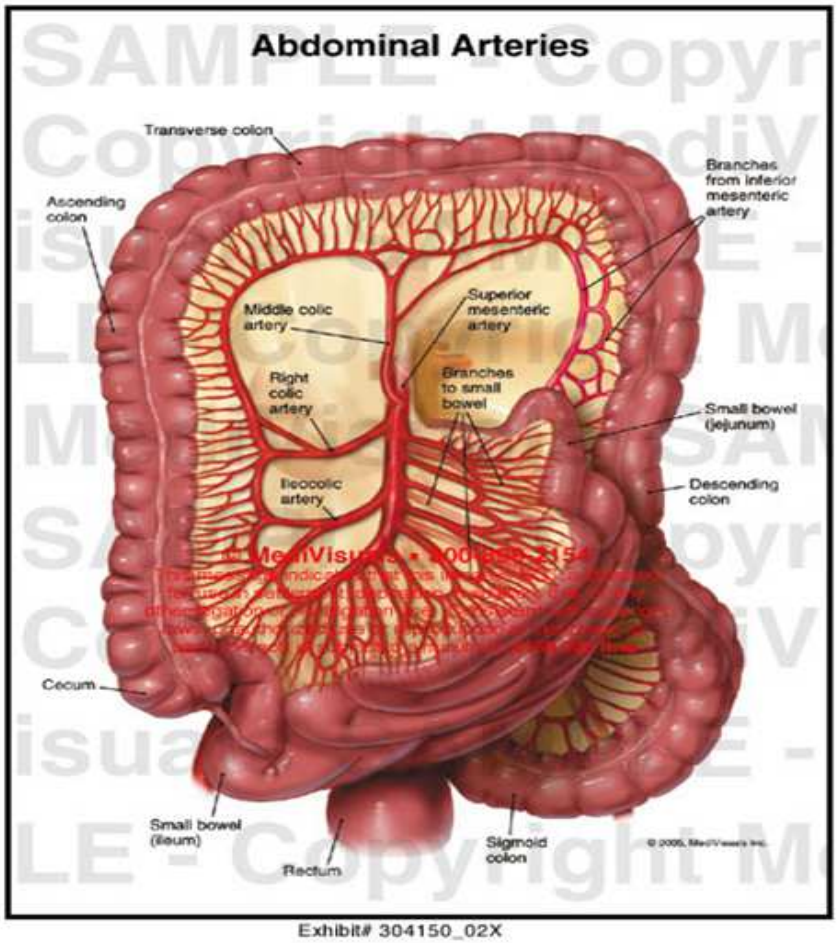
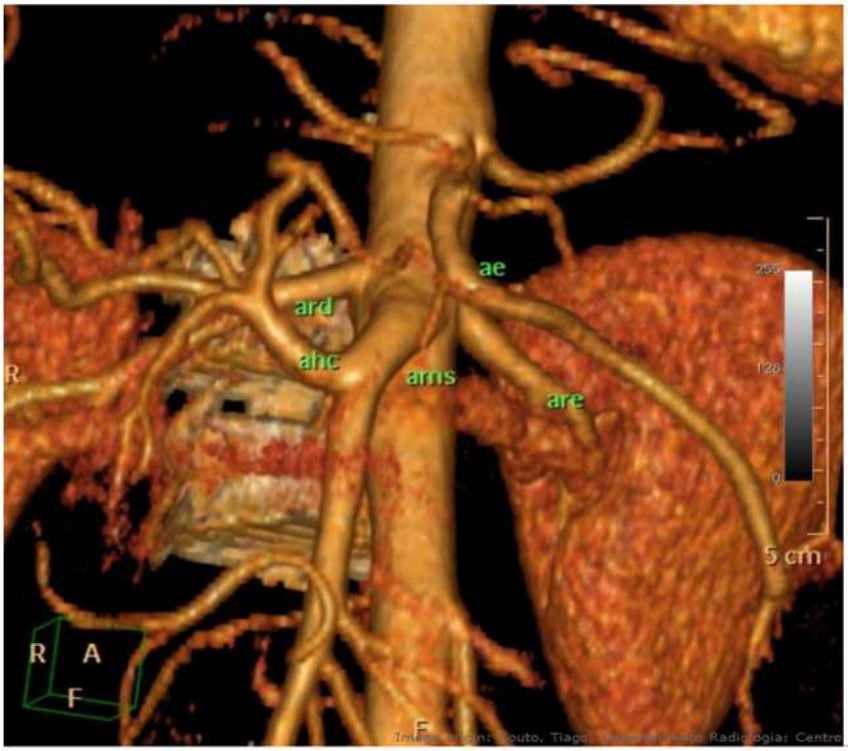


Figure. 2.2: Blood supply to small and large intestine with superior and inferior mesenteric, ileocolic, and right colic arteries

2.2.3 Mesenteric Arteries Relations:

Throughout its course, the superior mesenteric artery is surrounded by the superior mesenteric plexus of sympathetic nerves, which springs from the lower part of the celiac plexus; and it is accompanied by its vein, which lies close along its right side (Kaufman J A, Lee M J, 2004). At its origin, it is behind the body of the pancreas, and is closely related to the two large veins that cross the front of the aorta from the left to right – namely, the splenic vein, which passes above its origin, and the left renal vein, which passes below it. As the artery begins its descent, it crosses in front of the left renal vein, and, after escaping from behind the body of

the pancreas, it crosses in front of the uncinate process of the pancreas to reach the third part of the duodenum. Beyond that point, its posterior relations are the same as those of the root of the mesentery (Petscavage & Maldjian 2007).



http://eurorad.org/eurorad/view_figure.php?pubid=9662&figid=28593&nr=1&lang=en

Figure: 2.3: Abdominal aorta branches. SMA - Superior mesenteric artery; SA - Splenic artery; CHA - Common hepatic artery; RRA - right renal artery; LRA - left renal artery.

2.2.4 Mesenteric Arteries Branches:

The branches of the superior mesenteric artery are numerous and are accompanied by veins and by offsets of the superior mesenteric plexus of nerves. They supply the lower parts of the head of the pancreas and duodenum, the jejunum, and ileum, and vermiform appendix, thececum, and the ascending and transverse colon. The jejunal and ileal branches arise from its convex or left border; the others form its concave border (Iezzi et al 2008).

The jejunal and ileal branches – twelve or more in number – run obliquely toward the intestine between the layers of the mesentery, dividing and anastomosing to form a series of arterial arcades; smaller branches spring from the first series of arcades and divide and unite to form a second series, and, in the lower part of the mesentery, three or four tiers of arcades are formed. Numerous small arteries spring from the last series of arcades, and, alternating with one another, pass round opposite sides of the gut, gradually sinking deeper into the substance of its walls (Saad ,etal,2005). The middle colic artery arises as the superior mesenteric escapes from behind the body of the pancreas. It enters the transverse mesocolon at once, and runs downward and forward in it, dividing into right and left branches. Secondary and tertiary branches arise from these, forming variable arterial arcades, but much less richly than in the case of the arteries to the small intestine. The ultimate branches supply the transverse colon; and, towards its ends, they anastomose with branches of the right and left colic arteries. Since the middle colic vessels lie in the transverse mesocolon, they are posterior relations of the stomach (Saad et al, 2005).

The inferior pancreatico-duodenal artery is a small vessel that arises immediately before the superior mesenteric reaches the duodenum. It runs to the right between the head of the pancreas and the third part of the duodenum, giving branches to both; and these branches anastomose with branches of the superior artery of the same name (Winston, et al, 2007).

The right colic artery is variable in its level of origin, or it may be absent altogether. It runs toward the right, behind the peritoneumacross the duodenum or other structures on the posterior wall according to its level of origin. It ends by dividing into ascending and descending branches from which small arteries spring to supply the ascending colon; the

ascending branch passes over the lower part of the right kidney to supply also the right flexure and first part of the transverse colon and anastomose with a branch of the middle colic; the descending branch anastomoses with a branch of the ileo-colic. When the right colic artery is absent, its place is taken by a large ascending branch of the ileo-colic which anastomoses directlywith the middle colic (Koop, et al,2004).

The ileo-colic artery arises in common with the right colic or a little lower down. It runs toward the right behind the peritoneum; and it also ends by dividing into ascending and descending branches which give twigs to the ascending colon. The descending branch, besides supplying the colon, sends, branches to the cecum, a branch to the vermiform appendix, and a branch to the lowest part of the ileum which anastomoses with the end of the superior mesenteric artery (Iezzi , et al 2008).

The appendicular artery enters the lowest part of the mesentery, whence it passes into the mesentery of the appendix and runs in its medial free margin to reach the appendix (McNulty, et al, 2001).

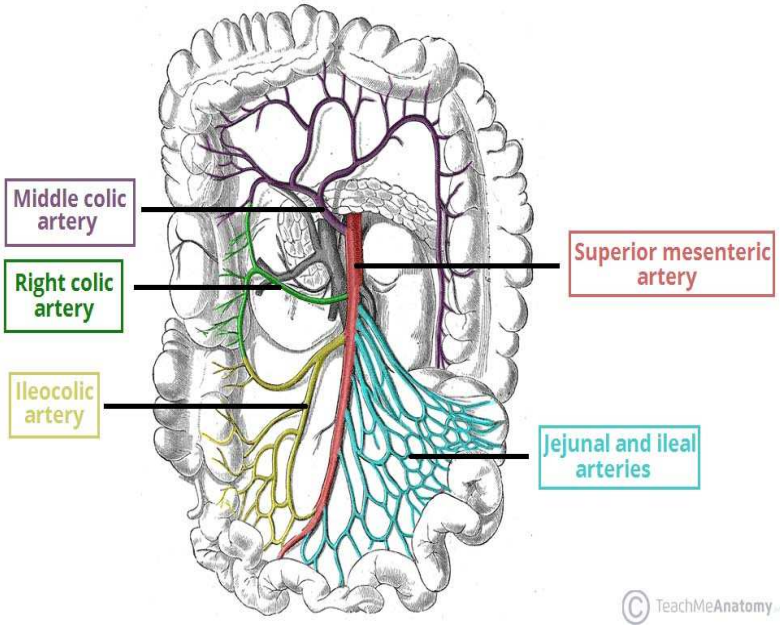


Figure:2.4 a diagram shows the superior mesenteric artery and its branches.

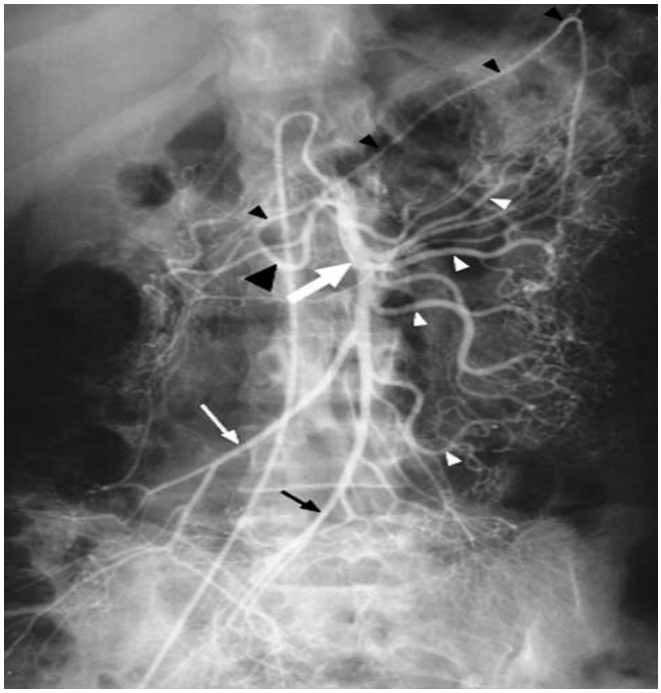


Figure:2.5 : Selective superior mesenteric artery (SMA; large white arrow) arteriogram shows the middle colic artery (large black arrowhead) divides into left (small black arrowheads) and right branches. Multiple jejunal (Iezzi , et al 2008).

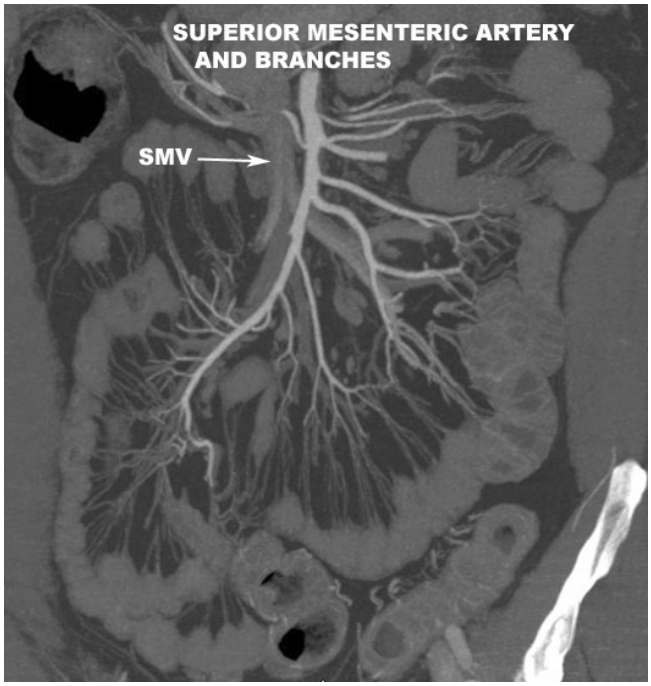


Figure:2.6 : CTA of the superior mesenteric artery and its branches (Saad et al, 2005).

2.2.5 Mesenteric Arteries Variants

As noted above, the SMA can have a great number of variants related to arteries typically seen from the celiac axis. The SMA may originate from the celiac axis (Figure:2.7) or provide any combination of hepatic arteries, or accessory gastric, splenic, or pancreatic vessels. The “normal” SMA anatomy may be present in as many as 68 % of cases (Figure:2.8a). The ileocolic artery appears to be the most consistent structure from the SMA. The other vessels have some degree of variability. Although normally a separate branch, the middle colic artery, can share a common trunk with the right colic artery (middle colic-right colic trunk, (Figure:2.8b) in up to 52 % of cases, representing the most common variant. If not involved in aberrant anatomy with the middle colic artery, the right colic artery may be an independent branch of the SMA (38 %) or a branch of the ileocolic artery in 8 % of cases (Figure:2.8c). There may be an accessory right colic in 8–10 % of cases. Less commonly, the middle and right colic artery are absent (<10 % of the time), or the middle colic may send a large branch to the splenic fl exure. In very rare cases, the middle colic artery may be a branch of the celiac artery (Valentine , 20003).

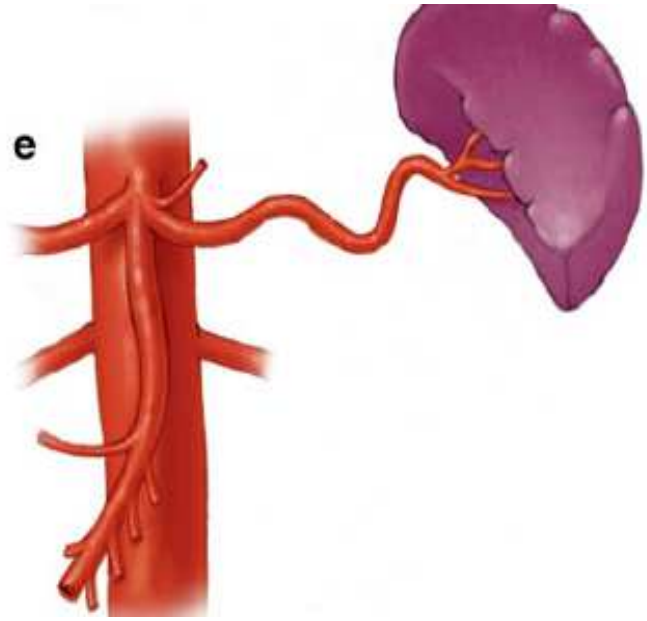


Fig:2.7: Celiacomesenteric axis – combined celiac and superior mesenteric artery origin (By permission of Mayo Foundation for Medical Education and Research. All rights reserved) (Randall R., 2015).

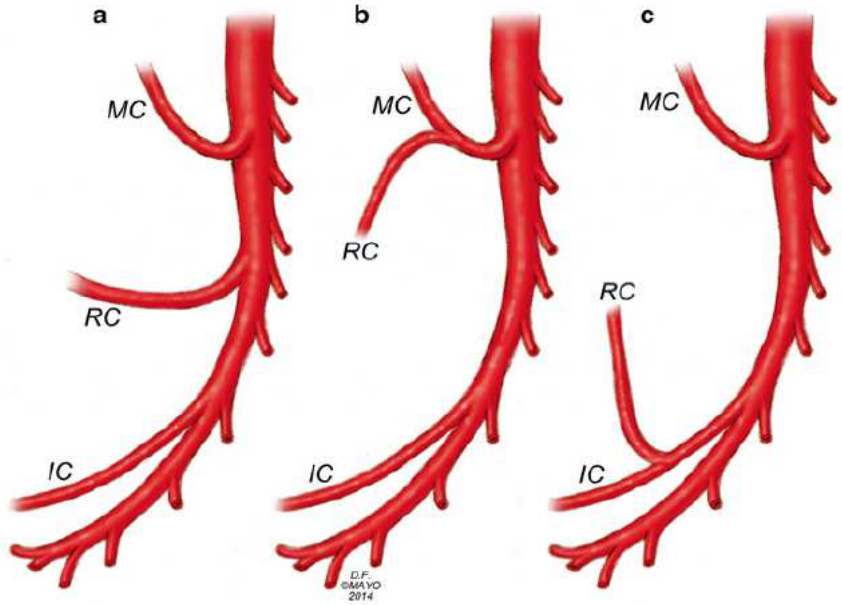


Fig. 2.8 : Superior mesenteric artery anatomic variants. IC ileocolic artery, RC right colic artery,MC middle colic artery. (**a**) normal anatomy with separate IC, RC, and MC origins. (**b**) Combine MC and RC origin and separate IC origin. (**c**) Common RC and IC origin and separate MC origin (By permission of Mayo Foundation for Medical Education and Research. All rights reserved) (Randall R., 2015).

2.3 Superior Mesenteric Artery(SMA) Physiology

2.3.1 Mesenteric Arteries Physiology

During fasting conditions, 20-25% of the total arterial circulation is distributed to the three major splanchnic arteries; the celiac artery (CA), the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA). The splanchnic arteries receive approximately 800 ml (CA), 500 ml (SMA) and 50 ml (IMA) blood/minute, respectively (Kolkman et al 2003). The arterial branches first reach the serosal layer of the gut, then the muscular layer, and finally terminate in the mucosal branches. In the presence of hypotension, local vasodilation ensures adequate perfusion within a wide pressure range (Bulkley ,et al,1987). Two other mechanisms to ensure oxygen supply is increased oxygen extraction and internal intestinal redistribution of blood flow to the metabolically more demanding mucosa (Haglund et al,1999). The CA arises from the aorta in a nearly perpendicular angle (figure 1), just below the diaphragm, and supplies the stomach, the duodenum, the spleen and the liver through the left and right gastric arteries, the gastroduodenal artery, the hepatic artery and the splenic artery, respectively. The organs supplied by the CA have an interdependent collateral blood flow making them less vulnerable to peripheral arterial occlusion. The CA and SMA share a collateral blood flow, with great individual differences, through the superior and inferior pancreaticoduodenal arteries (Figure:2.9). The marginal artery of Drummond, running in the mesentery alongside the colon, is supplied by both the SMA and IMA and constitutes a collateral circulation for the large intestine. The meandering mesenteric artery, also called the Arcade of Riolan, is a collateral vessel important in chronic pathologic redistribution of mesenteric arterial circulation (Gourley,et al, 2005). This collateral vessel connects the middle and left colic artery and runs in a

tortuous fashion in the left upper quadrant of the abdomen. The artery of Drummond and the meandering mesenteric artery can attenuate ischemia in the small or large intestine caused by isolated peripheral occlusion of the SMA or IMA. However, there is little if any collateral circulation to the small intestine in a healthy person, leaving this organ highly vulnerable to acute arterial occlusion of the SMA. The IMA arises from the mid section of the abdominal aorta and supplies the descending colon, the sigmoideum and the proximal part of the rectum. As mentioned above, IMA and SMA share a collateral blood flow through the meandering mesenteric artery. There is also a collateral blood flow from the left internal iliac artery for the tissues supplied by the IMA, as well as a rich collateral blood-flow between the left and right internal iliac arteries.

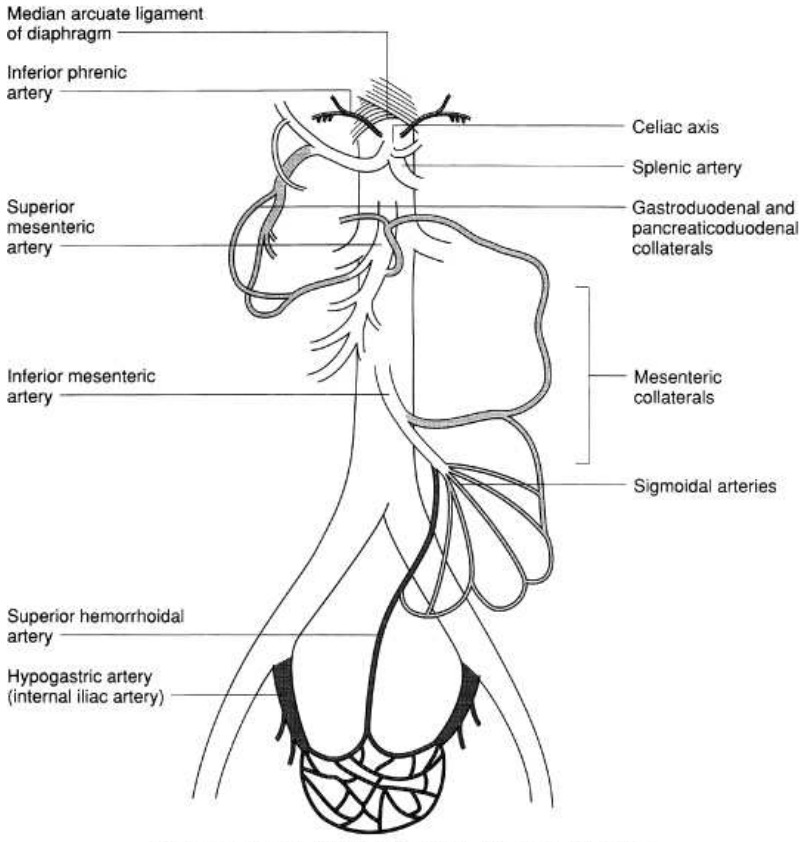


Fig:2.9 Mesenteric Arterial Circulation

2.3.2 Mesenteric Arteries Pathophysiology

Due to the metabolic and anatomical conditions mentioned above, the mucosal layer is the most susceptible to intestinal ischemia. In response to ischemia, the intestine responds by basal shunting of the circulation to the base of the villi in order to minimize oxygen consumption and preserve bowel integrity (Haglund , 1987). As a consequence, in ischemic conditions, the mucosal layer is sacrificed first in order to preserve bowel integrity. The intestinal tissue responds to ischemia by arteriolar smooth muscle relaxation and metabolic response. If the blood flow decreases below 30ml/100g tissue, oxygen consumption drops markedly in order to prevent necrosis (Bulkley , 1985). Hypovolemia in the patient with acute occlusive ischemia can further exacerbate oxygen delivery by activation of the renin-angiotensin axis (Reilly, , 1997) and thus promoting vasoconstriction of the mesenteric arterial circulation. If ischemia prevails, development of transmural infarction occurs and eventually, bowel wall integrity is compromised. If untreated, this leads to peritonitis, septic shock and death.

2.3.3 Mesenteric Arteries Pathogenesis

2.3.3.1 Arterial embolism to the SMA

Due to the oblique angle of the SMA relative to the aorta, the SMA is susceptible to arterial embolism which is most commonly dislodged in the proximal part of the SMA, sparing the jejeunal branches (Bergan , 1967). The sources of emboli are often of cardiac origin due to infarction, atrial fibrillation, cardiac thrombus or cardiac valvular disease. In this group of patients, there are rarely signs of previous abdominal angina and the onset of pain and symptoms is sudden. There may be other manifestations of arterial embolic disease such as a previous history of ischemic stroke and/or emboli to other organs. Synchronous emboli are

present in 2/3 of the patients (Acosta , 2005). Embolism can also be secondary to aortic thrombus, arterial catheterization, or paradoxical venous thromboembolism through a patent foramen ovale.

2.3.3.2 Arterial thrombosis of the SMA

Patients with arterial thrombosis of the SMA share the common features associated with atherosclerotic disease such as hypertension, hyperlipidemia, diabetes and smoking. They often present with a previous history of myocardial infarction and/or stroke (Järvinen,1994 & Björck , 2002). There may be symptoms of previous abdominal angina with diarrhoea, post-prandial pain and involuntary weight loss. The thrombosis often occurs in an area with turbulent flow, i.e the proximal SMA, or in the ostial SMA due to calcification of the aorta. In thrombotic occlusions, several SMA branches may be occluded, but the extent of infarction is dependent on collateral circulation and possibly hypoxic preconditioning (Mallick, 2004).

2.3.4 Mesenteric Arteries Pathology

2.3.4.1 Mesenteric arterial ischaemia:

Ischaemic damage may occur in the bowel secondary to partial or complete occlusion of the major mesenteric arteries. Complete occlusion, especially if acute in origin, usually leads to haemorrhagic infarction and gangrene of a variable extent of the bowel, limited to the areas supplied by the artery involved. Such an event leads to localized and subsequently generalized peritonitis, which is often a fatal condition. Infarcted bowel may be rapidly fatal as the bowel lies within the relatively large peritoneal cavity which has a large surface area from which toxins may be absorbed and this is particularly likely in the presence of a secondary paralytic ileus. Such a situation results in severe toxaemia and gross

dehydration, secondary to uid exudate into the peritoneal cavity, which may result in rapid coma and death. For these reasons acute mesenteric ischaemia requires a rapid diagnosis and early treatment to avoid a fatal outcome. Conversely, narrowing of the mesenteric arteries from one cause or another does not usually result in such rapid demise of the patient as collateral circulation may take over the supply of the bowel. This may be sufficient to maintain viability although function may be severely compromised. Such a situation may persist until compromise of the mesenteric vessels is sufficient to lead to symptoms and this may be followed by acute on chronic occlusion and bowel infarction. Again, a timely diagnosisallows treatment to be instituted, which may both treat the symptoms and substantially reduce the risk of impending bowel infarction. Acute intestinal ischaemiaFor the purposes of this article, ischaemia secondary to venous thrombosis will not be considered, rather a sudden symptomatic reduction in arterial intestinal blood flow which is sufciently restricting to result in mesenteric infarction. Vasoconstriction may occur secondary to physiological changes or secondary to drug administration, but these are usually transient and of insufficient duration to result in irreversible damage. Such nonocclusive causes for mesenteric ischaema are usually treated by supportive therapy, treatment of cardiac failure or infusion of vasodilators. In practical terms, acute intestinal ischaemia is a result of a sudden occlusion of the proximal portion of the major mesenteric arterial supply, usually due to thrombosis or embolus. Clinical presentation of such a situation is usually with acute abdominal pain that is of sufficient severity to cause the patient to seek medical advice. Unfortunately, the duration of symptoms does not necessarily seem to correlate with whether there is reversible or irreversible bowel damage. Physical examination may be surprisingly unremarkable

compared with the degree of pain the patient experiences, and is often associated with vomiting and/or diarrhea. It is therefore vital that the diagnosis is borne in mind and the level of clinical suspicion high for this potentially life-threatening condition. The typical patient presenting with this complaint would be a female of around 70 years, with sudden onset of severe and constant abdominal pain. More than half of patients will have vomited and some will have associated diarrhoea. Chronic intestinal ischaemia. A number of conditions may cause chronic intestinal ischaemia from occlusion or severe stenosis of the major visceral arteries. The majority of cases are caused by atherosclerotic involvement of the proximal portions of the coeliac, superior mesenteric or inferior mesenteric arteries. Other conditions, however, may affect the large or small arteries supplying the gut, including neurobromatosis, bromuscular hyperplasia, visceral artery dissection, radiation therapy, connective tissue diseases and other rare causes such as cocaine abuse. The majority is, however, secondary to large vessel stenotic or occlusive disease and it is these conditions which are most amenable to surgical and endovascular therapies. In order to understand and treat obstructive lesions causing bowel ischaemia, it is important to review the anatomy of the splanchnic blood supply and the collateral vessels which may develop. In normality, the majority of the gut, from oesophagus to rectum, is supplied by the three splanchnic arteries, namely the coeliac artery (CA), the superior mesenteric artery (SMA) and the inferior mesenteric artery (IMA). There is, however, the potential for considerable collateralization between the territories of these arteries, which arise from the embryologic foregut, midgut and hindgut. Such collateralization is evidenced by the observation that 27% of patients undergoing aortography prior to peripheral vascular surgery were found to have an

asymptomatic stenosis of 50% or more of either the CA or the SMA (Valentine , et al. 1991). It is quite possible to completely occlude the SMA and the CA and for the bowel to survive entirely on the bloodflow derived from the IMA (the smaller of the three) (Chiene ,1869). Should such a situation arise, then blood derived from the IMA flows through the Arch of Riolan to supply the marginal anastomotic arteries and subsequently on to ll the middle colic and pancreatico-duodenal arteries. This allows blood to supply areas normally supplied by the CA and SMA. If the opposite situation exists where the IMA is occluded then flow from the middle colic artery, derived from the SMA, may supply the distal colon (Figure 1). Whilst theoretically possible for the left colon to receive collateral blood supply from the internal iliac artery, via the haemorrhoidal vessels, in the presence of an occluded IMA it would appear that the principal supplier of collateral flow to the left colon is the SMA (Iliopoulos , et al.1990). In common with other areas, the opening up of the collateral channels is dependent upon the slow progression of stenotic disease in the major mesenteric vessels: the more rapid the progression of the disease, the more likely are symptoms at an earlier stage of the progression. However, it would appear in practical terms that symptoms of intestinal ischaemia are not usually present until at least two of the three major splanchnic arteries have either high grade stenoses or occlusions (Mikkelsen, 1957) (Hansen , 1976). It is, however, important to bear in mind the patient's clinical history in relation to previous gut surgery as bowel resection may interrupt the ability of the bowel to collateralize effectively. In such circumstances the territory of the SMA tends to be vital for collateralization, and compromise of the SMA will cause the early onset of symptoms. Following ingestion of food there is a signi can't change in the haemodynamics of the normal mesenteric

circulation. At rest the duplex waveforms found in the SMA show a triphasic pattern in keeping with a high resistance vascular system *(Moneta , et al. 1988).* After feeding, this pattern changes markedly due to both local and systemic factors. The resulting bowel hyperaemia causes the duplex wave pattern to change to that of a low resistance system, with a high end diastolic velocity. In addition, the peak systolic velocities increase. In the presence of sign can't occlusive disease the ability of the main vascular trunks to satisfy the need for increased flow is insufficient and the situation analogous to angina pectoris exists, where the demand for oxygen delivery from the blood is not met, and the bowel becomes ischaemic. There is a resultant release of anaerobic metabolic byproducts and this result in the typical postprandial pain in patients with chronic intestinal ischaemia. A typical patient presenting with chronic intestinal ischaemia is usually female, is between 40 and 70 years of age, and will usually have undergone extensive investigation for long-standing abdominal pain with weight loss (Olafsson , et al 1989). Early in the progression of mesenteric angina, patients may not necessarily relate their pain to meals, although as the condition progresses this becomes more obvious. The pain typically will develop approximately half an hour after ingestion of a meal and this may last for up to 3 hours after. As the relationship to food becomes progressively more obvious, then patients will tend to 'fear' eating and weight loss becomes a feature of the syndrome, to the extent that advanced malignancy may be suspected as an alternative diagnosis.

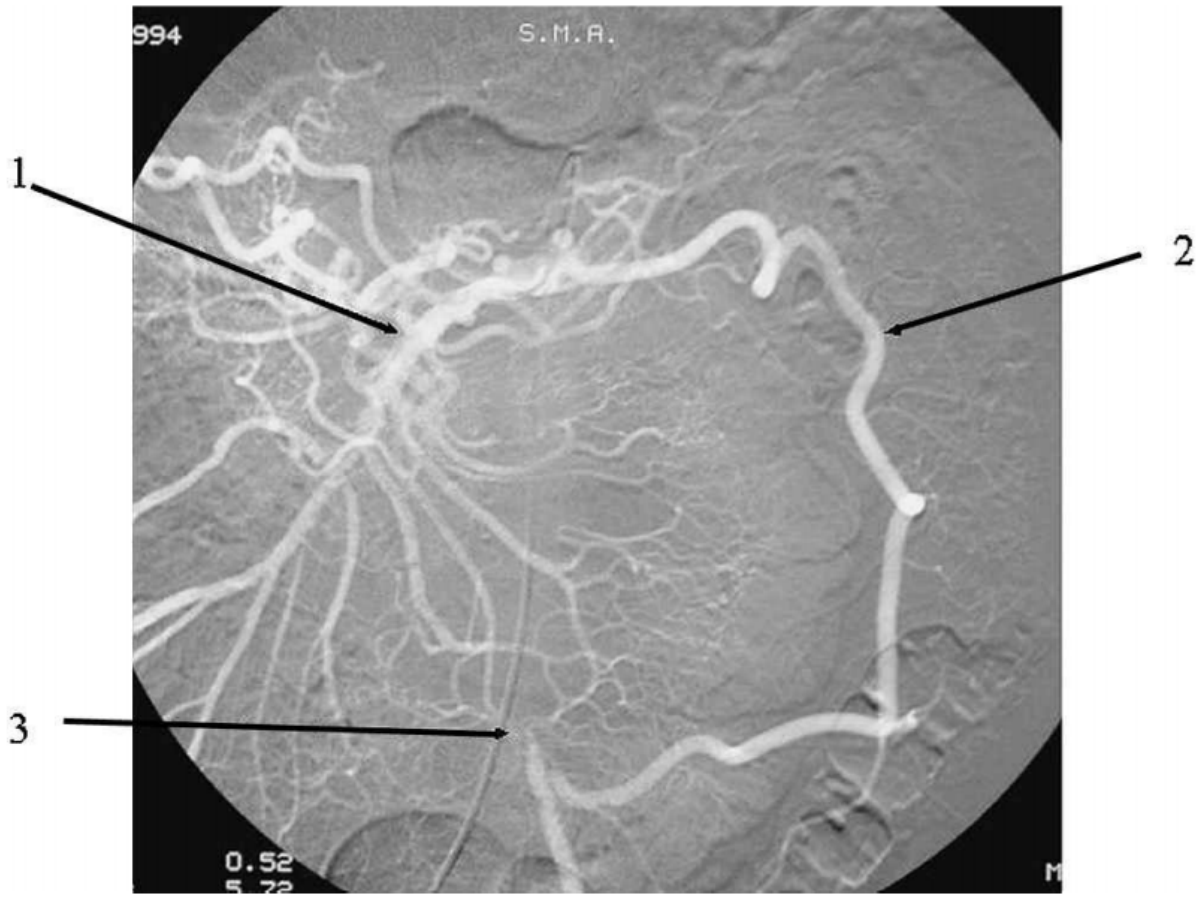


Figure:2.10 Selective SMA (arrow 1) angiogram: contrast lls the marginal artery (arrow 2) to supply the left colon and the IMA territory. The IMA is occluded (arrow 3) (Olafsson , et al 1989).

2.3.4.2 Superior mesenteric artery (SMA) syndrome:

Is a gastro-vascular disorder in which the third and final portion of the duodenum is compressed between the abdominal aorta (AA) and the overlying superior mesenteric artery. This rare, potentially life-threatening syndrome is typically caused by an angle of 6°-25° between the AA and the SMA, in comparison to the normal range of 38°-56°, due to a lack of retroperitoneal and visceral fat (mesenteric fat). In addition, the aortomesenteric distance is 2-8 millimeters, as opposed to the typical 10-20 (Shetty, et al, 2000). However, a narrow SMA angle alone is not

enough to make a diagnosis, because patients with a low Body Mass Index (BMI), most notably children, have been known to have a narrow SMA angle with no symptoms of SMA syndrome (Mehta, 2012). SMA syndrome was first described in 1861 by Carl Freiherr von Rokitansky in victims at autopsy, but remained pathologically undefined until 1927 when Wilkie published the first comprehensive series of 75 patients (Welsch , et al, 2007). According to a 1956 study, only .3% of patients referred for an upper-gastrointestinal-tract barium studies fit this diagnosis, making it one of the rarest gastrointestinal disorders known to medical science (Goin, ,& Wilk, ,1956). Recognition of SMA syndrome as a distinct clinical entity is controversial, due in part to its possible confusion with a number of other conditions (Cohen , Field,& Sachar DB, 1985), though it is now widely acknowledged (Shetty, , et al, 2000). SMA syndrome is also known as Wilkie's syndrome, cast syndrome, mesenteric root syndrome, chronic duodenal ileus and intermittent arterio-mesenteric occlusion (Laffont , et al, 2002). It is distinct from Nutcracker syndrome, which is the entrapment of the left renal vein between the AA and the SMA, although it is possible to be diagnosed with both conditions (Barsoum,2008).

2.4 Superior Mesenteric Artery(SMA) Investigation

2.4.1 Radiological investigation:

2.4.1.1 Radiography

Upright and supine abdominal images are helpful screening tools for detecting free air or bowel obstruction, the findings are usually not specific for mesenteric ischemia. Findings such as thumbprinting (mucosal edema) are occasionally masked by a gasless fluid-filled abdomen (Dhatt, et al 2015).

2.4.1.2 Computed Tomography

CT is the primary imaging modality, and it has been proven to be highly accurate in the diagnosis of mesenteric ischemia; scans sometimes depict the underlying etiology. Typically, CT scans show mesenteric edema with irregular thickening of the wall of the small or large bowel that is greater than 3 mm. Large-vessel disease (superior mesenteric artery/vein [SMA/SMV]; inferior mesenteric artery/vein [IMA/IMV]) is diffuse, whereas small-vessel arterial or venous disease is more likely to be focal (Lee , et al 2008 & Ofer, et al 2008).

2.4.2 Magnetic Resonance Arteriography

Magnetic resonance arteriography (MRA) is occasionally used to evaluate the patency of the superior mesenteric artery (SMA) and inferior mesenteric artery (IMA). However, MRI plays a limited role in the diagnosis of mesenteric ischemia of the small or large bowel. Typically, if additional imaging modalities are needed, ultrasound or angiography is the next step in the workup (Li KC, et al 1997)

2.4.3 Ultrasound

2.4.3.1 Ultrasonography

Ultrasonography is the first-line imaging study for the diagnosis and the postoperative evaluation and follow-up of patients with diseases of the abdominal great vessels. The combination of velocimetry with ultrasound and color methods (color Doppler ultrasound) substantially increased the possibilities for ultrasound in the diagnosis of disorders involving the deep vessels. (Battaglia, et al, 2010)

2.4.3.2 Doppler Ultrasound

Color Doppler (CD) and spectral waveform ultrasonography help in evaluating the patency and adequacy of flow through the celiac artery, SMA, and IMA. Preprandial and postprandial Doppler examinations are typically performed. Sample velocities are assessed proximal to the stenosis, where flow is expected to be normal; at the stenosis, where velocity is maximal; and distal to the stenosis, where velocity is the most turbulent (Baccoli ,et al ,2008).

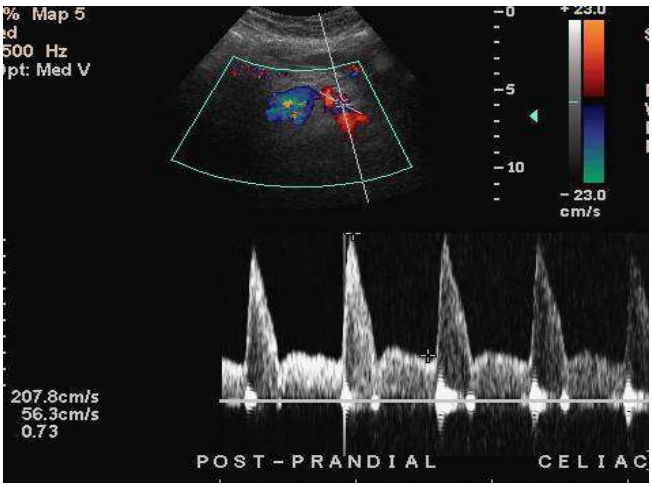


Figure:2.11: Ultrasonographic evaluation of mesenteric ischemia with spectral analysis and color Doppler imaging (Baccoli ,et al ,2008).

2.4.3.3 Doppler Ultrasound Physics

2.4.3.3.1 The Doppler Effect

The Doppler effect is a change in sound frequency when sound is emitted from a moving source. In diagnostic ultrasound techniques the sound is bounced off red blood cells which act as moving sources of ultrasound within a blood vessel. In the diagram below, if the red blood cells are scatters, it can be seen that when the incident sound wave strikes red blood cells moving towards the sound source, the scattered wave returning to the transducer has a shorter wavelength and therefore a

higher frequency. If the incident wave strikes red blood cells moving away from the sound source, the returning wave has a longer wavelength and therefore a lower frequency. The Doppler shift in sound frequency is equal to the difference between the transmitted and received sound frequencies. These shifts in frequency are within the audible range of human hearing. The rise or fall in frequency is directly proportional to the velocity of the moving red blood cells. Therefore, the Doppler shift can demonstrate the presence, velocity and direction of blood flow within a vessel. Continuous wave Doppler devices cannot discriminate specific depth levels whereas pulsed wave Doppler devices can. (D. Buirwin, 2002)

2.4.3.3.2 Color flow Doppler

Referred to as color flow imaging or simply color Doppler (CD) is a method of displaying Doppler shift data as color information in a real time image. CD provides a rapid means of determining the presence and direction of flow and is especially helpful for finding small vessels which cannot be seen on regular real time display. CD compliments pulsed wave Doppler (PD) but does not replace it. CD shows us where there is flow and what direction the flow is in. It can then be used to direct the PD sample volume placement for waveform spectral analysis and velocimetry assessment. The Doppler shifts may be encoded in just about any color but by popular convention, red and blue are the two primary colors used. The primary colors represent flow towards or away from the transducer. The color scale may be reversed so that flow towards the transducer may be displayed as red or blue. Again by convention, arteries are usually displayed in red and veins in blue. The color scale is graded in shades or hues of red and blue. Increasing distance away from the baseline (zero flow) indicates greater Doppler shifts or higher flow

velocity detection which, if exceeded, results in a display artifact known as aliasing. Proper setup and interpretation of CD images is relatively complex and demands a good understanding of Doppler physics and the capabilities and limitations of the equipment. (D. Buirwin, 2002)

2.4.3.3.3 Power Doppler (PD)

Is based on the power in the Doppler spectrum. (Conventional CD is based on frequency shift.) One advantage of PD over CD is an increased sensitivity to slow flow. Its disadvantages are that it can only indicate the presence of flow and not direction or velocity. (*D. Buirwin, 2002*)

2.4.3.4 Doppler Ultrasound Indices

Quantitative indices can be calculated from the peak systolic velocity (V_{peak}), mean flow velocity (V_{mean}), and diastolic velocity (V_{diast}) (***equation*** I & II). These indices are independent of the beam angle and are therefore unaffected by imprecise angle data (*Matthias Hofer, 2001*).

- Gosling pulsatility Index (PI)

$$PI = (V_{peak}-V_{diast})/V_{mean}$$ ……***equation*** *(I)*

- Pourcelot resistance Index (RI)

$$RI = (V_{peak}-V_{diast})/V_{peak}$$ ……***equation*** *(II)*

2.5 Ultrasound Technique

Duplex ultrasound (DU) is recognized as a valuable tool for the assessment of blood flow in many vascular territories. The application of this technique to the superior mesenteric artery (SMA) has increased rapidly throughout the last decade (Perko, 2001).

Doppler sonography is the most affordable method to measure noninvasively the main characteristics of mesenteric blood flow. Doppler sonography spites some well-known drawbacks such as operator dependence so the superior mesenteric artery can now be considered a well-established field for the application of Doppler sonography. Normal parameters of blood flow have been defined and the most relevant changes in conditions such as mesenteric ischemia and Crohn's disease have been documented. Moreover, Doppler sonography can show dynamically the deep changes of blood flow parameters in the superior mesenteric artery after a standard meal. Evaluation of the superior mesenteric artery with Doppler sonography before and after a standardized meal has therefore been proposed as a possible way to detect borderline disease, when changes caused by disease are too small to be visible at fasting and a stressed condition is needed to uncover the alterations (FranCes, et al, 1998).

2.5.1 Patient preparation and Choice of transducer

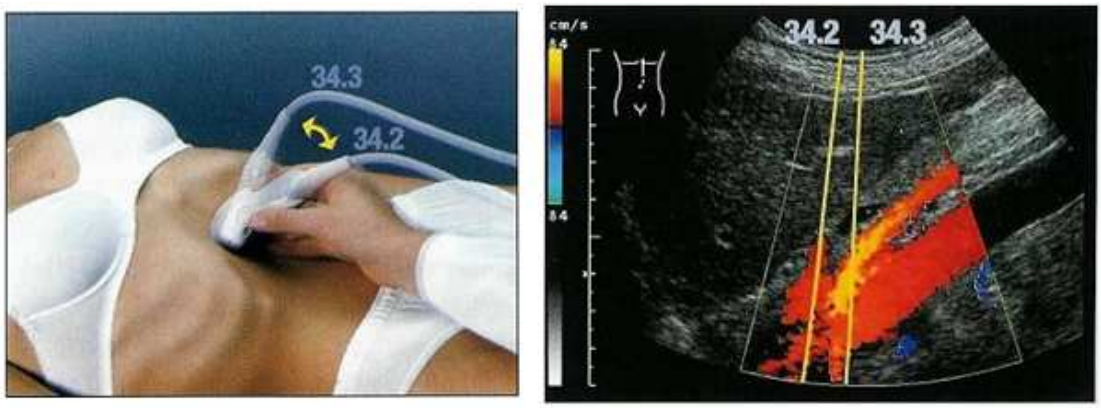
Sonography is performed in the morning, after patients are fasted overnight. The patients are placed in the supine position. As transducer chosen, 3.5-MHz convex transducer with a Doppler emission frequency of 2.8 MHz is used (AU4 Idea; Esaote, Genoa, It aly); pulse repetition frequency is 5 KHz, Doppler angle ranged between 40 and 60,and a high-pass filter of 400 Hz is used to avoid undesirable low-frequency signals arising from movement of the arterial wall (FranCes,et al, 1998).

2.5.2 Scanning technique:

Superior mesenteric and celiac arteries are identified by duplex ultrasound of the upper abdomen, either longitudinally or transversely (Figure 1). The proximal portion of each vessel is scanned initially,

having adjusted the sample volume size to the diameter of the vessel. After this, the vessels are scanned along their visible length. Fasting measurements are made on three occasions. The inferior mesenteric artery is inconsistently visualized (Muller, et al 1991).

Direct color-flow imaging will occasionally make it easier to locate small vessels. But color flow also delays real-time visualization, and moving the transducer around to search for vessels can increase color artifacts. (Figure:2.12(A)) illustrates the search for the correct transverse planes, the location of which is marked with lines in the upper abdominal longitudinal scan (Figure:2.12(B)) (Matthias Hofer, 2001). The SMA is sagittal scan, with high definition zooming and 5 kHz pulse repetition. The Doppler sampling volume is positioned 2–3 cm distally from the vessel origin, before the emergence of its branches and adjusted to comprise the vascular lumen, but without touching its walls. A spectrum of at least five cardiac cycles is obtaining during breath hold, and the mean flow velocity is automatically determined by the equipment. The insonation angle is determined on the real time B-mode image and always maintained below 60°. The measurement of the SMA diameter is utilizes by the equipment for determining the vessel area and automatically calculating the SMA flow volume. With the objective of reducing random errors, each measurement is repeated for three times, and the mean value is considered as the final result (Fabiana, et al,2013).



(A) (B)

Figure:2.12: moving the transducer around to search for SMA (A), location of the upper abdominal longitudinal (B) (Matthias Hofer, 2001).

2.6 Nutrition

Breakfast helps top up the energy stores you have used up each night whilst your body repairs and renews itself. It also gives you energy for your morning activities, whether at work, school, home or out and about. While breakfast is often quoted as 'the most important meal of the day', this may not be strictly true. It's more helpful to say that no meal should be categorised as more important than another, and daily food intake should be considered as a whole. Skipping meals, whether it be breakfast, lunch or dinner, is not advised. Establishing a regular eating pattern has been shown to improve glycaemic control, reduce likelihood of weight gain and curb hunger pangs. However, it is estimated that up to one third of us still regularly miss breakfast. Many of us put this down to time pressures in the morning, but with a little planning, you can find a breakfast choice to suit your lifestyle. Healthy breakfast provides essential nutrients that the body needs, such as fibre, vitamins and key minerals such as calcium and iron. Research has shown that people who eat breakfast have more balanced diets than those who skip it, are less likely to be overweight, lose weight more successfully if overweight, and

have reduced risk of certain diseases such as cardiovascular disease and diabetes. Missing breakfast may increase feelings of hunger later on in the day, resulting in snacking on less healthy foods without necessarily catching up on essential nutrients. Eating breakfast may also help to improve mental performance, concentration and mood – three more good reasons to eat something in the morning (Deshmukh-Tasker, etal 2010).

Breakfast should provide about 20-25% of your daily nutritional requirements, and it's not just about having any breakfast it's about having a healthy breakfast. Breakfast built from the main food groups below will give you an excellent start to the day. Starchy foods such as bread, cereals, rice, potatoes, and pasta provide energy, B vitamins, some iron and fibre. Cereals are a really good choice as well as being quick and easy to prepare, they often are fortified with vitamins, iron and calcium to contribute to your daily nutritional requirements. However be careful to check the labels, as some of these products have added sugar and salt. Porridge, bread, rolls, English muffins, scones, malt loaf, fruit bread, currant buns and bagels all provide good sources of energy, mainly as starchy carbohydrate, that will help kick start your metabolism, and they're all usually low in fat too (Deshmukh-Tasker, etal 2010).

Choose wholegrain varieties whenever possible to ensure a good fibre intake, and try to avoid cereals coated in sugar. Evidence suggests that porridge oats for breakfast may have a positive effect on total cholesterol concentration when compared to skipping breakfast, making porridge a winning choice. If you're pushed for time, try an oat-based rumbler as a 'packed breakfast' the night before, place some porridge oats in a pot and cover with enough low fat milk or yoghurt to soak into the oats, add some fresh or dried fruit on top, sprinkle with a bit of cinnamon for added flavour and store in the fridge. Then, in the morningjust grab from the

fridge with a spoon before you leave the house a perfect breakfast on the go. Fruit and vegetables are good sources of vitamins and fibre. Breakfast is a perfect time to boost your 5-a-day intake. On your cereal, try chopped fresh fruit, like a banana, or some dried, stewed or canned (in juice rather than syrup) fruit , or add half a grapefruit or fruit salad to your usual breakfast. A small glass (150ml) of pure fruit juice also counts as one serving of your 5-a-day. For something different, try a fresh fruit smoothie just blend some fruit of your choice with low fat yogurt or milk. Frozen berries, fruit in season or ripe fruit are all ideal for making smoothies. Alternatively, give vegetables a try at breakfast time, mushrooms, baked beans or tomatoes on toast make a tasty change when you have a bit more time (Deshmukh-Tasker, etal 2010).

Milk and dairy foods give you protein, calcium and B vitamins. Calcium is essential to keep your bones strong and healthy, whatever your age, and a serving of milk on your cereal can give you up to one third of your daily calcium needs. Use low fat milks like skimmed, semiskimmed or 1%. If you don’t like milk on cereal, try a glass of milk on its own or in a smoothie, or have a pot of low fat yogurt instead. Natural yoghurt is delicious topped with fruit and a sprinkle of muesli. If you use milk and other products not made from cows’ milk such as soya, oat, coconut, almond or rice, make sure they are unsweetened and fortified with calcium.

Meat, fish, eggs, beans and other non-dairy sources of protein give you protein, iron and vitamins. These foods are not essential at breakfast, but they can add variety. Try not to have meat at breakfast every day, and choose cooking methods such as grilling or poaching instead of frying in fat. Poached, boiled or scrambled eggs, baked beans, grilled kippers or smoked haddock are healthier options than bacon and sausages, which are

higher in saturated fat. Foods and drinks high in fat and sugar give you energy but are generally low in vitamins, minerals and other nutrients. Limit these foods and choose low fat sunflower, olive or vegetable oil based spreads where possible and spread thinly. Choose low sugar, wholegrain breakfast cereals instead of sugar-coated, refined varieties. Avoid fizzy drinks, biscuits and crisps at breakfast and use fruit to add natural sweetness instead of sugar on your cereal. Remember to include a drink. Water, milk, pure fruit juice, tea and coffee all supply vital fluids. Use low fat milks and ask for 'skinny' coffee when out and about. Being well hydrated also helps you to concentrate better (Deshmukh-Tasker, etal 2010).

2.7 Previous study:

Color Doppler ultrasongraphy has been used in fasting and postprandial state to show the alterations on SMA indices in response to different meals types.

Doppler sonography, with its capacity to explore noninvasively the characteristics of blood flow in many vessels, could be a method to dynamically evaluate the mesenteric blood flow in patients with CD; previous preliminary studies have reported potential usefulness in this field, Francesco Giovagnorio, et al (1998).

With the publication of studies demonstrating the viability of Doppler ultrasonography (US) for the evaluation of the superior mesenteric artery (SMA) flow, there has been an increasing interest in the splanchnic hemodynamics evaluation in CD by means of Doppler US, considering its noninvasiveness, safety, low-cost and capability for quantitative analysis, (Fabiana Paiva Martins (2013) .

Transcutaneous measurement of blood flow in humans using Doppler ultrasound techniques has been accomplished in a number of peripheral

and deeply situated blood vessels such as the brachial artery, carotid arteries, aorta, and fetal blood vessels, (Qamar, et al (1986).

In a novel study **by Stephen, et al,** (1986) they stated that the velocity waveforms from the SMA in the fasting state exhibited re verse diastolic flow, whereas those from the celiac artery exhibited forward flow throughout diastole. No significant postprandial changes were noted in the diameter of either the celiac axis or SMA, and the systolic rise time in each vessel was also unchanged. The velocity waveform in the celiac axis was unchanged postprandially. Reverse velocity in the SMA was reduced or abolished by eating, and this was statistically significant. Peak systolic velocity was unaffected in the celiac artery, and although a modest rise was seen in the SMA, this did not reach statistical significance. Diastolic forward flow was also unchanged in the celiac artery but almost doubled in the SM. This was significant ($p < 0.01$). In summary, celiac artery circulation showed no postprandial changes whereas the SMA showed changes in peak systolic velocity, reverse velocity and flow time, and forward diastolic flow. Of these, the latter reached statistical significance.

In other study **by Sieber, et al,(1991) they** reported that the mean diameter in basal values of SMA did not differ significantly. After food intake, the diameter of the superior mesenteric artery increased significantly ($p<0$ 05) and reached a maximum 60 minutes after food intake. This represents a 12% (range 5-26%) increase over the initial values.

The time averaged velocity of the superior mesenteric artery in the fasting state did not differ significantly in the various experiments. Oral food intake induced a significant increase in the mean time averaged velocity

(p<001), reaching a maximum 30 minutes after eating. This velocity reflects a 130% (range 31-245%) increase over the fasting value.

During the basal period, the computed blood flow was 443 (38) ml/minute. After the test meal, there was an immediate, noticeable increase in mesenteric blood flow and a plateau was reached within 20 minutes of taking the meal . The maximum mesenteric blood flow was 250% higher than in the basal period (range 180- 360%) and was reached within mean (SEM) 38 minutes of eating. Sixty minutes after food increase (area under curve over 60 minutes) of mean (SEM) 53 (6)% over control values (p<005). Again, the increase in mesenteric blood flow was mainly due to increases in velocity rather than diameter.

In a local study that published internationally and cited by many authors **E.Abd Elrahim, et al,** (2016) found the basal superior mesenteric artery blood flow volume (SMABFV) measurements were approximately near to similar in values for different four meals type. The mean basal SMABFV is 285 ml/min for fatty meal, 305 ml/min for carbohydrate meal, 299 ml/min for Cheese and yoghurt meal, and 278 ml/min for jam and Boiled meal, increase in response to the oral intake of four meals. An immediate and marked increase in mesenteric blood flow was observed, and the maximum was reached within 30-60 minutes of taking four meals, this differ with. Thirty minutes after carbohydrate and jam and Boiled meal intake, SMABFV fall towards basal values and was not significantly different from baseline at 105 minutes post pranially. (359ml/min for meal 2, and 433ml/min for meal 4). In contrast sixty minutes after fatty and Cheese and yoghurt meal intake, SMABFV decreased gradually, but was still significantly (p<005) above basal values (656ml/min for meal

1, 779ml/min for meal 4) at the 105 minutes,

In a unique study by **Nami Someya, et al, (**2008) they studied blood flow (BF) responses in superior mesenteric artery (SMA) during and immediately after a meal, (n _ 9) healthy subjects ingested solid food {300 kilocalories(Kcal)} and water ad libitum within 5 min (4.6 _ 0.2 min, means _ SE), and then rested for 60 min in the postprandial state. They found that the baseline values did not differ between experimental and control trials except for BF in the forearm. In the control trial, the MBV, vessel diameter, and BF in CA and SMA, and MAP showed no change throughout the measurement. The MBV in the SMA increased from the baseline 10 min after the end of the meal and reached its peak increase (from 0.28 _ 0.02 to 0.64 _ 0.03 m/s) at 36 _ 4 min after the end of the meal. Postprandial splanchnic vessels' diameters and BF. Vessels' diameters did not show significant change in both arteries. SMA BF in the experimental trial was greater than in the control trial from the start of the meal to the end of the measurement. SMA BF increased from baseline to 15 min after the end of the meal. During the meal, the RIBF in the SMA tended to decrease in the experimental trial than in the control trial (P _ 0.08) and was lower than the control trial after the end of the meal. The RIBF in SMA decreased from baseline to 5 min after the end of the meal throughout the postprandialstate. Splanchnic circulation during meal. Compared with the baseline, the MBV in both CA and SMA showed a greater value immediately after the start of the meal (0.08 _ 0.02 and 0.03 _ 0.01 m/s increase from the baseline, respectively). Compared with the control trial, MBVs in the experimental trial were greater during a meal. The RIMBV started decreasing from baseline within 1 min in the CA and tended to decrease 4 min after the meal in the SMA. The RIMBV in both arteries was lower in the experimental trial than in the control trial.

Sieber et al, (1992) they used Duplex ultrasound w to investigate superior mesenteric artery haemodynamics in six healthy male volunteers, aged 21-27 years (mean 23 years), and they state that an immediate and marked increase in mesenteric blood flow was observed, and the maximum was reached within 45 minutes of taking either meal. Sixty minutes after food intake, SMABF decreased gradually, but was still significantly ($p<005$) above basal values at the end of the experiments. The increase in blood flow was mainly the result of an increase in velocity, as sharp increases ($p<0$ 05) in Time Average Velocity (TAV) were seen after both liquid and solid food intake (Table II). Liquid and solid oral test meals induced small but significant ($p<0$ 05) increases in vessel diameter amounting to 16 (1)% and 13 (2)% (mean (SEM)) respectively.

Andrew , et al (1995) performed fasting and postprandial duplex scanning of 25 healthy control group volunteers, and 80 patients with vascular disease (the Patients were divided into three groups). They were found that the mean fasting SMA Peak Systolic Velocity (PSV) did not differ among controls and groups 1 and 2. Postprandial PSV increased significantly in all groups, but was not different among controls and groups 1 and 2. Mean fasting PSV was significantly higher, and the postprandial increase in PSV significantly lower, in group.3 compared with controls and with groups 1 and 2. The mean fasting and postprandial PSV for the control group was 147 + 30 cm/s and 230 f 55 cm/s, respectively. The mean fasting and postprandial SMA PSVs for the 80 patients with peripheral vascular disease and varying degrees of stenosis. There were no significant differences in fasting and postprandial

PSVs among controls and group 1 patients and group 2 patients (P >0.05). Nor were there significant differences in fasting or postprandial PSVs between patients in groups 1 and 2. Group 3 patients had significantly higher fasting PSVs than did the controls (P <0. 000l) and group 1 patients (P <0.000l) or group 2 patients (P = 0.003) (Figure). The mean maximum percent increase in postprandial SMA PSVs for the control patients was 57%, and was not significantly different from patients in groups 1 and 2. They conclude that postprandial increases in SMA PSVs are blunted in patients with high-grade stenosis, but feeding velocities do not stratify between lesser degrees of stenosis. Both fasting and postprandial PSVs identify high-grade (>70%) stenosis.

Taourel, et al (1998) they assessed the resistance index (RI) in the superior mesenteric artery under fasting and postprandial conditions in 15 healthy subjects and in 27 patients with cirrhosis to determine whether the amount of change in the RI reflects the presence or severity of liver dysfunction. They state there was no difference was found between the baseline RIs in healthy (RI = 0.85) and cirrhotic subjects (RI = 0.84), nor was there a difference in baseline RIs between subgroups of cirrhotic patients according to the severity of liver disease. The RI decreased significantly ($p < 0.05$) after the meal in both the healthy (13%) and cirrhotic (8%) subjects, but the postprandial decrease was significantly less pronounced ($p < 0.05$) in cirrhotic patients than in healthy subjects. Among cirrhotic patients, there was no correlation between the postprandial decrease of the RI and severity of liver disease.

Sidery, et al, (1994) They were studied the differential effect of high fat and high carbohydrate meals on mesenteric blood flow using Doppler ultrasound by measuring Superior mesenteric artery blood flow in eight

healthy men were twice before and after a 2 5 MJ meal (either 74% of the energy as carbohydrate or 71% as fat).

The pattern of the superior mesenteric artery blood flow response was different after the two meals (interaction effect p<0.000l analysis of variance), with a far more sustained response after fat. The time by which half the meal had emptied (t_{50}) was also significantly greater after fat (p<002). Superior mesenteric artery blood flow corresponding to t_{50} was 449 ml/min after carbohydrate and 592 ml/min after fat. There was a significant curvilinear relation between the superior mesenteric artery blood flow response and gastric emptying after carbohydrate (r^2=094) and no relation at all after fat. They conclude that ingestion of meals with a high fat content slows gastric emptying compared with meals with a high carbohydrate content in healthy volunteers. The relation, however, between the volume of meal remaining in the stomach and the mesenteric response was considerably different after the two meals.

Elsamani1, et al, (2016) used color Doppler technique in assessment of superior mesenteric artery for different meals intake. They was state that the basal superior mesenteric artery blood flow volume (SMABFV) measurements were approximately near to similar in values for different four meals type. The mean basal SMABFV is 285 ml/min for grilled goat meat, 305 ml/min for beans meal, 299 ml/min for the cheese with yoghurt meal, and 278 ml/min for the jam with boiled egg meal. An immediate and marked increase in mesenteric blood flow was observed, and the maximum was reached within 30-60 minutes of taking four meals. Thirty minutes after carbohydrate and jam and Boiled meal intake, SMABFV fall towards basal values and was not significantly different from baseline at 90 minutes postpranially. (359 ml/min for meal 2, and 433 ml/min for meal 4). In contrast sixty minutes after fatty and

Cheese and yoghurt meal intake, SMABFV decreased gradually, but was still significantly (p<005) above basal values. The peak systolic velocity PSV of the SMA changed with time to reveal a main effect of four meals types and the alteration of the PSV of the SMA was in the same direction in fatty and Cheese and yoghurt meal s in which the maximum PSV reached at 30 minutes and then decrease gradually toward baseline value. In contrast in carbohydrate, and jam and Boiled meals the maximum PSV reached at 45, 60 minutes consequently in carbohydrate , and jam and Boiled meals , then slightly decrease but still significant at the end of these meals at 90 minutes postprandially The peak systolic velocity PSV of the SMA changed with time to reveal a main effect of four meals types and the alteration of the PSV of the SMA was in the same direction in fatty and 3rd meals in which the maximum PSV reached at 30 minutes and then decrease gradually toward baseline value. In contrast in carbohydrate, and jam and Boiled meals the maximum PSV reached at 45, 60 minutes consequently in carbohydrate , and jam and Boiled meals , then slightly decrease but still significant at the end of these meals at 90 minutes postprandially

Muller, (1992), stated that in 10 patients with postprandial abdominal pain thought likely to be the result of mesenteric ischaemia Doppler ultrasound examinations of the superior mesenteric and coeliac arteries were performed both after fasting and a standard meal of 800 kcal. Compared with control values Doppler waveform analysis suggested seven abnormal vessels. Two patients had abnormal fasting superior mesenteric artery waveforms manifested by very high peak systolic velocities together with spectral broadening (one also had evidence of celiac artery stenosis), and one patient had normal velocities but an abnormal signal and evidence

of proximal superior mesenteric stenosis was supported by colour Doppler imaging and confirmed by angiography. Postprandialiy, two patients showed very high peak systolic and end diastolic velocities in the superior mesenteric artery (one had had a normal fasting waveform signal) and one in the coeliac artery, suggestive of vascular stenosis, while one patient showed a fall in peak systolic velocity. The diagnosis of mesenteric ischaemia in two of these patients was supported by digital subtraction angiography and abdominal computed tomography. Doppler ultrasound may be a useful non-invasive investigation for patients with postprandial abdominal pain that

helps to select patients for angiography. Patients with tight vascular stenosis may have abnormal fasting Doppler waveform patterns but in symptomatic patients further information may be obtained after the haemodynamic stress of feeding. Additional information to enhance the diagnostic sensitivity of the test may be obtained by colour Doppler imaging.

Hornum, , et al, (2006) reported that the Blood flow in the superior mesenteric artery (SMA) increases after a meal due to a vasoactive effect of the decomposed food. In exocrine pancreatic insufficiency, the digestion of food is compromised. We used duplex ultrasound to test the hypothesis that blood flow in the SMA after a meal increases less in patients with pancreatic insufficiency than in control persons. We studied 16 patients with chronic pancreatitis, eight of them with exocrine insufficiency, and eight healthy volunteers. The resistive index (RI) in the SMA was determined before and after a liquid meal. The RI reflects the downstream circulatory resistance, giving a precise description of mesenteric hyperaemia. Both groups of patients with chronic pancreatitis unexpectedly had lower fasting RI than controls,

0.818 and 0.815 vs 0.851, p=0.028 and p=0.0030, respectively. Postprandialy there was significantly less decrease in RI (less increase in flow) in patients with exocrine insufficiency than in controls, 0.055 vs 0.099, p=0.0047. There was a significant trend for a less pronounced postprandial decrease in RI with more impaired pancreatic function (p=0.0036). Our study thus demonstrates a reduced postprandial increase in SMA flow in patients with exocrine pancreatic insufficiency, and suggests an increased fasting SMA flow in chronic pancreatitis. Further studies are needed to evaluate the possible role of the test-meal-induced shift in RI in the SMA and of a lower-than-normal fasting RI in the diagnosis and monitoring of chronic pancreatitis.

In a rare study by Quarto , et al, (2002) they reported that intestinal involvement is frequently observed in systemic sclerosis (SSc) and is associated with malnutrition and a decreased survival rate. Vascular lesions are claimed to underlie and precede these changes. The aim of this study was to establish whether a reduced mesenteric blood flow was present in SSc patients with no signs or symptoms of small bowel involvement. Superior mesenteric artery (SMA) blood flow in the fasting state was measured by colour Doppler ultrasonography in 27 SSc patients and in 25 controls. The effect of a balanced liquid meal on mesenteric blood flow was measured in six matched patients and controls.

In fasting SSc patients, there were reductions in mean SMA diameter ($P<0.001$), blood flow (213±92 *vs* 398±125 ml/min in controls, $P<0.0001$) and pulsatility index (3.49±1.0 *vs* 4.13±0.97 in controls,$P<0.07$). In both groups, the meal increased basal flow values and the differences between controls and patients in the fasting state were not significant.

Dauzat, et al (1994) they reported that the haemodynamic effects of a meal on the splanchnic and hepatic circulation were evaluated in 30 healthy volunteers, using Doppler ultrasonography. The resistance index (RI) of the superior mesenteric artery and of the left and right intrahepatic arteries, the portal vein blood flow as well as the ratio between maximal velocity in the left and right intrahepatic arteries and the adjacent portal vein were measured initially, then 15, 30, 45, and 60 min after the ingestion of a standard balanced liquid meal. Postprandial haemodynamic changes were maximal 30 min after the meal; at that time, mesenteric artery RI decreased significantly [mean -11% (SEM 14%)] whereas portal vein blood flow increased markedly [mean +79% (SEM 14%)]; a significant increase in hepatic artery RI was observed in both liver lobes. The ratio between maximal velocities of the intrahepatic artery and the intrahepatic portal vein was reduced significantly; this ratio decreased more markedly in the right lobe of the liver. These findings would suggest that there was an adaptation of hepatic artery to portal vein blood flow after a meal. The subsequent increase in intrahepatic portal vein flow velocity was found to be greater in the right lobe of the liver.

Qamar, et al, (1988) they were used Transcutaneous Doppler ultrasound to measure superior mesenteric artery blood flow in 12 healthy volunteers in the fasting state and serially for 1 h after the ingestion of isocaloric and isovolaemic carbohydrate, fat, and protein liquid meals. The superior mesenteric artery blood flow increased significantly within 5 min of the end of each meal. The maximal responses were not significantly different but were reached at different times: carbohydrate, 64% at 15 min; fat, 60% at 30 min; and protein, 57% at 45 min. The response to the fat meal was significantly slower than the response to the

carbohydrate, and the response to protein was slowest of all. In a further group of 20 fasted normal subjects no significant change in superior mesenteric artery blood flow occurred after drinking 400 ml of distilled water at room temperature. In seven of these subjects, drinking 400 ml of distilled water at 4 degrees C also did not affect mesenteric blood flow. These results indicate that the chemical nature of the meal and not the volume per se is a significant factor determining postprandial mesenteric hyperaemia.

Chapter three

Materials and methods

3.1 Ultrasound Examination and equipment :

This study was performed using Siemens ACUSON X300 PREMIUM EDITION KT- LM 150XD with (C6-2) by an experienced sinologist (Germany) ultrasound scanner which was available at the areas of study. This machine allows a real-time cross-sectional images (i.e., B-mode echo) to be displayed simultaneously with real-time Doppler spectral display and sound. The scanner drive convex Doppler scan probes produce a frequency of 3.5 MHz-pulsed Doppler frequency was used; also they were connected with printing facility through digital graphic printer.

3.2 Design of the study:

This study is prospective analytic study deal with the ultrasound procedures to study superior mesenteric artery for different meals intake using color Doppler. Data collection sheet which was designed to include all variables.

3.3 Population of the study:

The population of this study were Saudi volunteers presented to our department. Selection of participation was done through simple random sampling.

3.4 Sample size and type:

This study consisted 100 of healthy participants include just males, their ages between 20 to 50 years were selected carefully after approved from the clinician that they were healthy and they have no history of cardiovascular disorder or diabetic and hypertensive, and according to the

positive evidence completely health, among the outflow of them in to ultrasound departments at collage of applied medical sciences, Taif University, KSA.

3.5Exclusion criteria

Any un healthy volunteers or volunteers above intended age.

3.6 Duration and place of the study:

This study was carried out in the period from December 2013 up to April 2016. All volunteers were scanning in Taif city in collage of applied medical sciences, Taif University, KSA.

3.7 Technique:

The volunteers were told to prepare themselves carefully for the scan by abstaining from food for at the least 8 hours prior to ultrasound scanning. Usually the scanning was carried out with the participants in supine position. A coupling agent gel was used to ensure good acoustic contact between the transducer and the skin. After informing the patients about the procedure, the area of interest in the abdomen was completely evaluated in at least two scanning planes. Surveys were used to set correct imaging techniques, to rule out pathologies and to recognize any normal variants. The volunteers were scanned by Duplex ultrasound in order to assess changes in SMA for Blood Flow Volume (BFV), Diameter (DM), (RI), (PI), (PSV), and EDV. All indices were measured after four standard meals which were prepared by experienced nutritionist. The four meals were different in calories and contents, as shown in the Table (3.1). The first meal was Grilled goat meat (fatty) (250 gram, 545 Kcal), the second meal was Beans (carbohydrate) (270 gram, 477 Kcal), the third meal was Cheese and yoghurt (360 gram, 633 Kcal), and the fourth meal was jam and Boiled egg (210 gram, 555 Kcal).

Table No (3.1): Calories for meals and their contents analysis						
Contents	**Gm**	**Energy**	**Fat**	**CHO**	**Protein**	**Fiber**
Grilled goat meat	150	234	15.3	-	27.6	-
White bread	100	311	0.7	66.9	9.2	0.3
Total	**250**	**545**	**16**	**66.9**	**36.8**	**0.3**
Beans (foul medames)	170	166.6	1.19	29.24	15.52	3.4
White bread	100	311	0.7	66.9	9.2	0.3
Total	**270**	**477.6**	**1.89**	**96.14**	**24.72**	**3.7**
Cheese	60	194.4	15	1.42	16.4	-
Yoghurt	200	128	7.4	8.8	6.6	-
White bread	100	311	0.7	66.9	9.2	0.3
Total	**360**	**633.4**	**23.1**	**77.12**	**32.2**	**0.3**
Jam	60	169.8	0.06	41.82	0.82	0.18
Boiled egg	50	74.5	5.4	0.15	10.8	-
White bread	100	311	0.7	66.9	9.2	0.3
Total	**210**	**555.3**	**6.16**	**108.87**	**20.82**	**0.48**

All volunteers were given one meal every day for duration of four days. Data sheet was prepared before scanning to include the different measurements during scan. The investigations were carried out under resting conditions with the volunteer lying in the supine position. The entire abdomen was also examined with a focus on any possible abnormal finding by B-mode ultrasonography. Each volunteer was scanned in fasting state, then postprandial, first immediately after 5 mins and then 6 fold with interval time of 15 mins continuously up to 90 mins. The SMA was identified and the DM and BFV measured 1 to 2 cm distally to the origin and proximally to the first side branches. The measurements performed while the volunteers held their breath. The angle between the incident Doppler beam and the long axis of the vessel was kept at less than 65°.

3.8 Data collection

The data were collected from measurements of different indices in data sheet which prepared specially for this task. Also ultrasound images were collected after each scan as documentation for that scan.

3.9 Data and analysis:

Microsoft office Excel 2007 was used to analyze data after entering all measurements readings. Mean max and standard deviations were calculated. Soon after that figures were used to display the results. The associations between the different variables of the results and the (SMA) measurement are tested using SPSS version 16.

3.10 Ethical Consideration

Special consideration was given to the right to confidentiality and anonymity of all survey participants. Anonymity was achieved by using numbers for each survey participant that will provide link between the information collected and the participants. In addition confidentiality was censured by making the collected data accessible only to the researchers.

Chapter four
Results

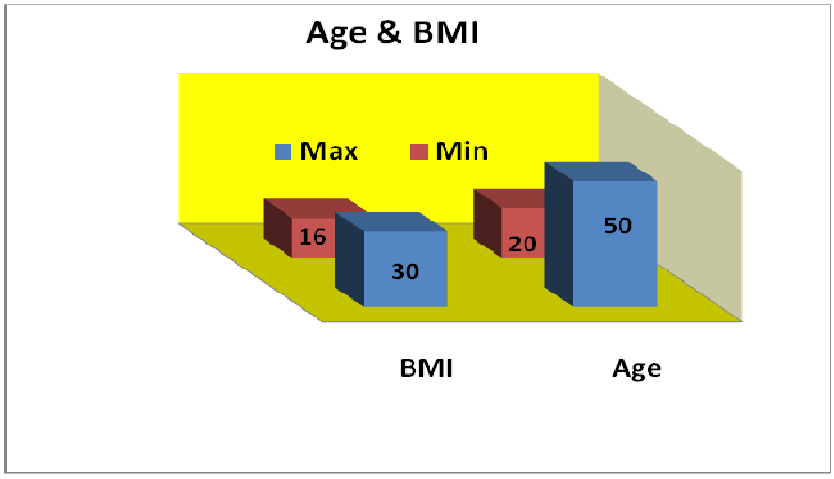


Figure: 4.1: Distribution of BMI and Age

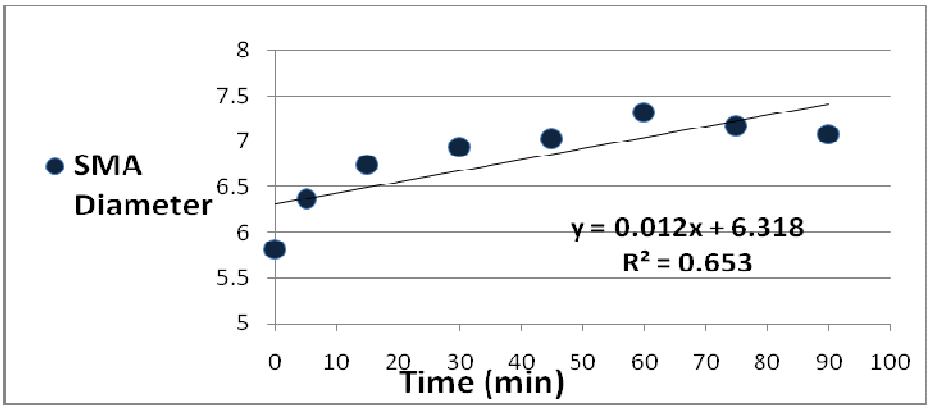


Figure:4.2: Fatty meal correlation of SMA diameter versus time

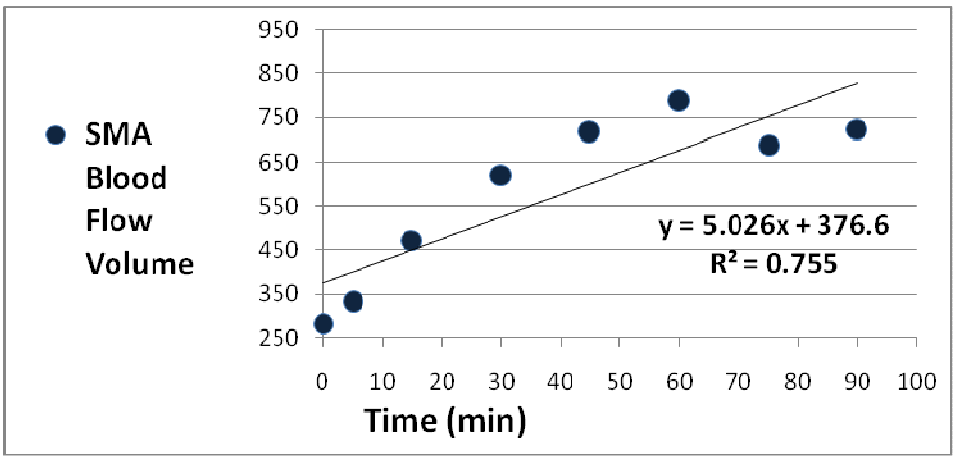


Figure:4.3: Fatty meal correlation of SMA blood flow volume versus time

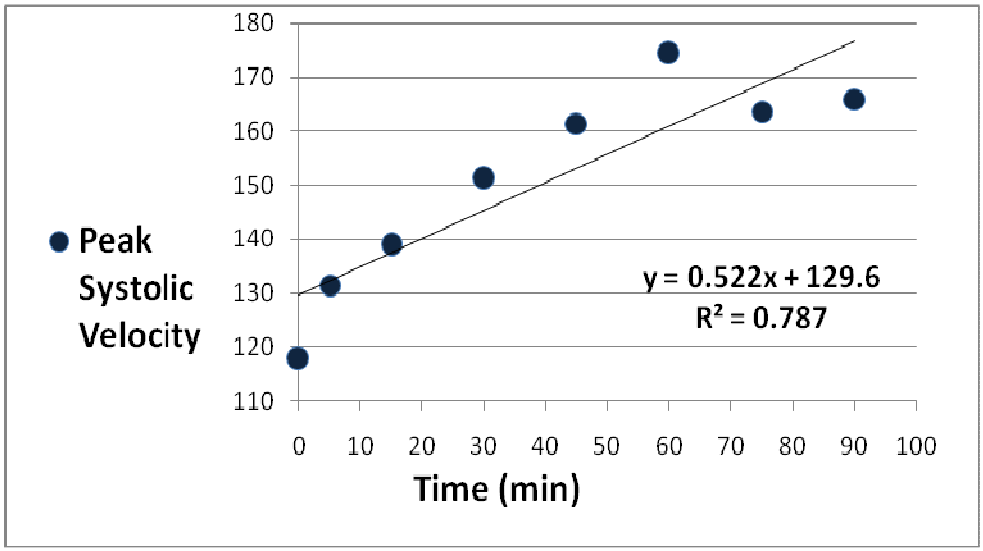


Figure :4.4: correlation for the fatty meal , peak systalic velocity versus time

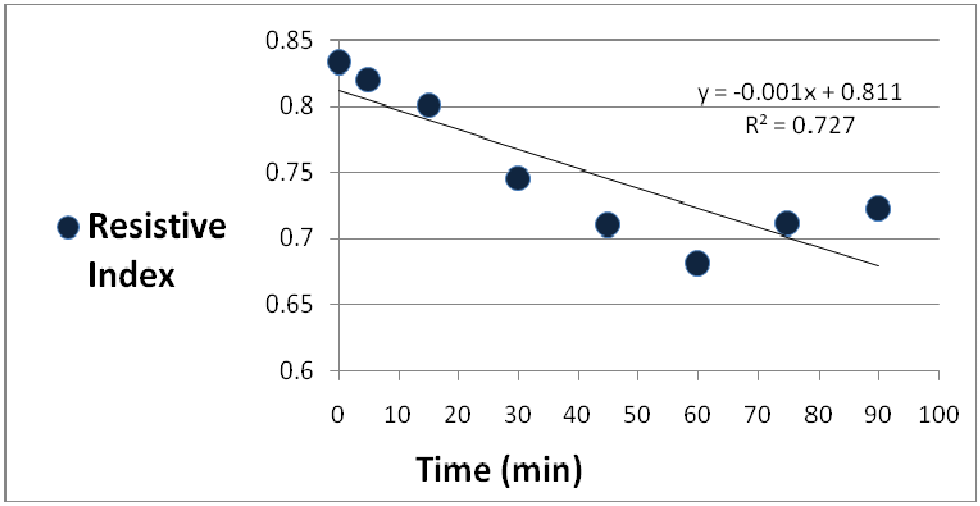


Figure:4.5: Fatty meal correlation of resistive index versus time

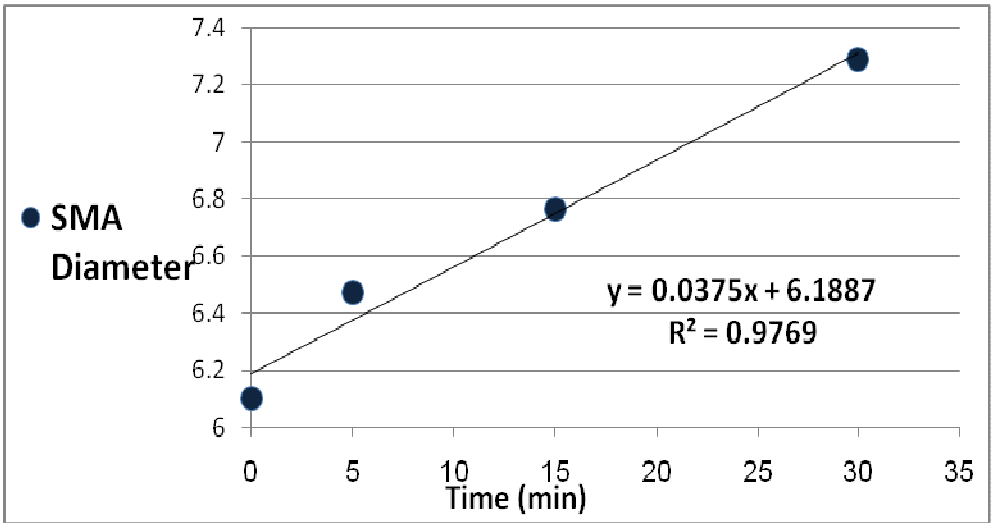


Figure:4.6: Carbohydrate meal correlation of SMA diameter versus time (0-30 mins)

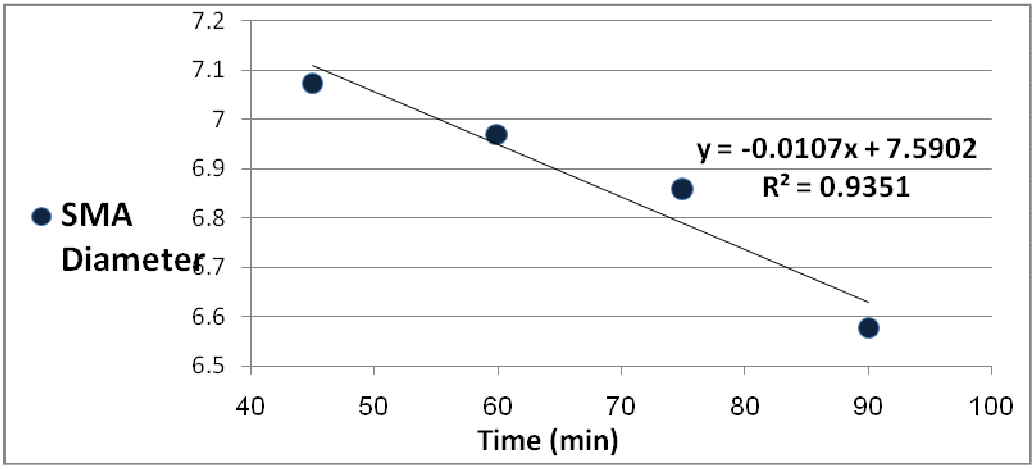


Figure:4.7: Carbohydrate meal Correlation of SMA diameter versus time (30-90 mins)

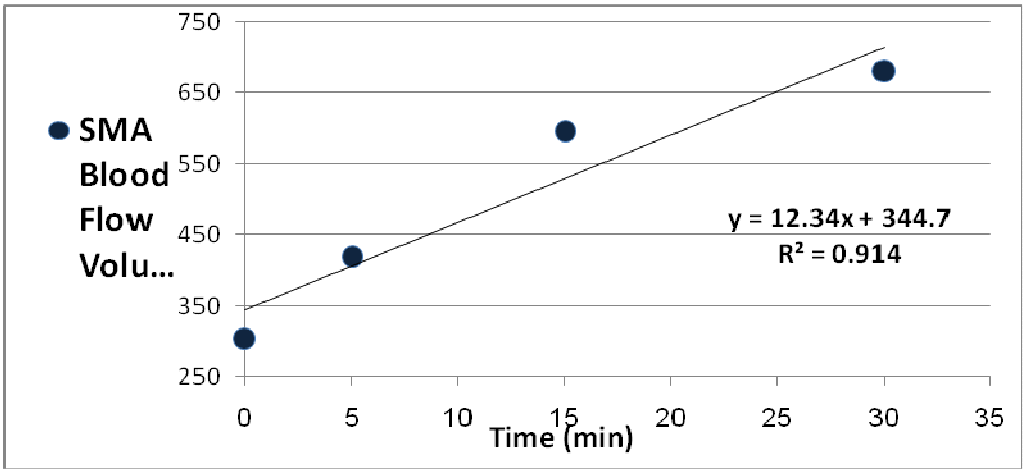


Figure:4.8: Carbohydrate meal correlation of SMA blood flow volume versus time (0-30 mins)

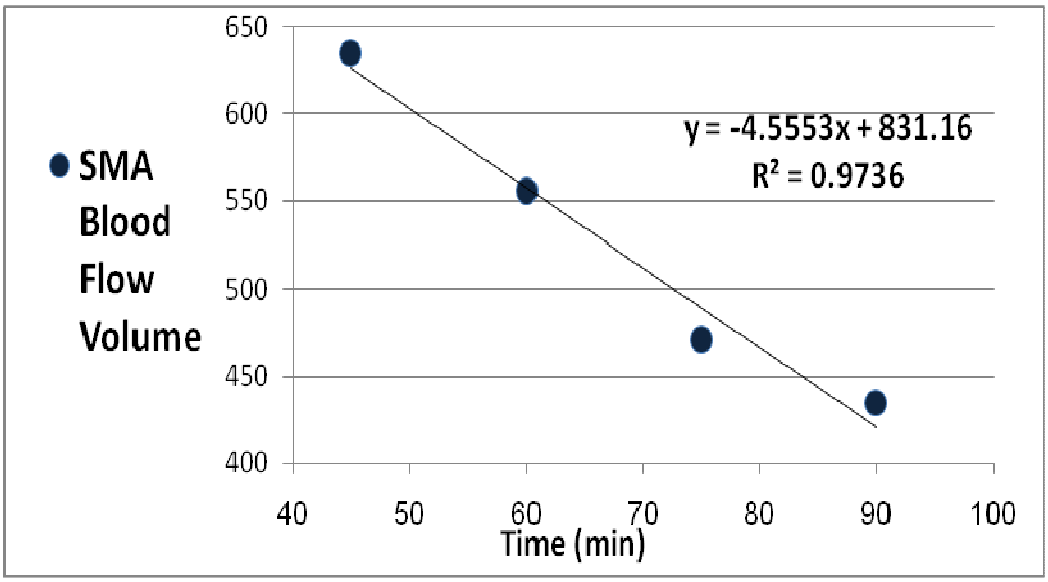


Figure:4.9: Carbohydrate meal correlation of SMA blood flow volume versus time (30-90 mins)

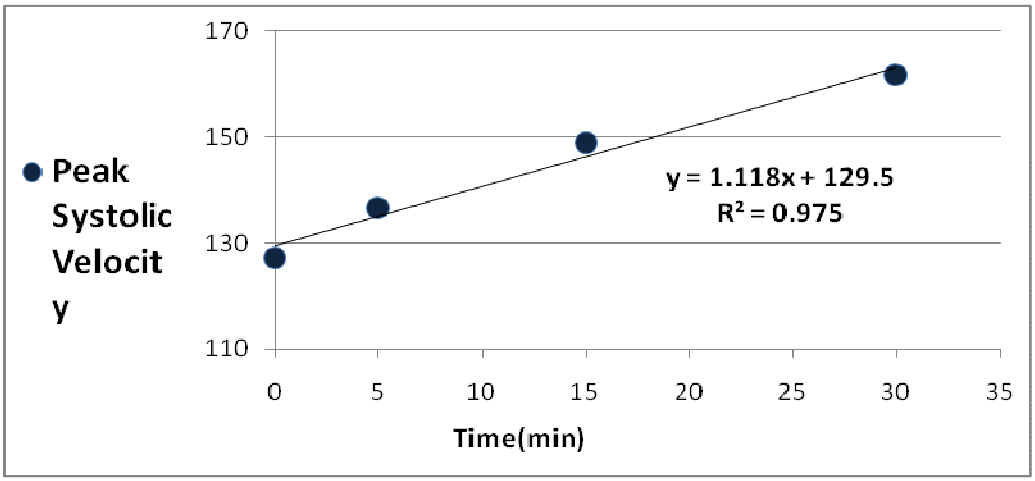


Figure:4.10: Carbohydrate meal correlation of peak systolic velocity versus time (0-30 mins)

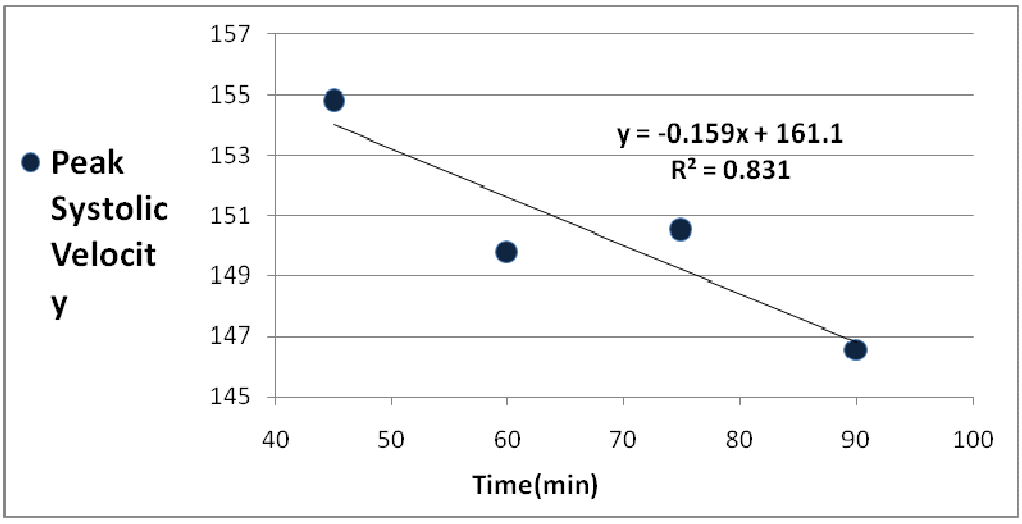


Figure:4.11: Carbohydrate meal correlation of peak systolic velocity versus time (30-90 mins)

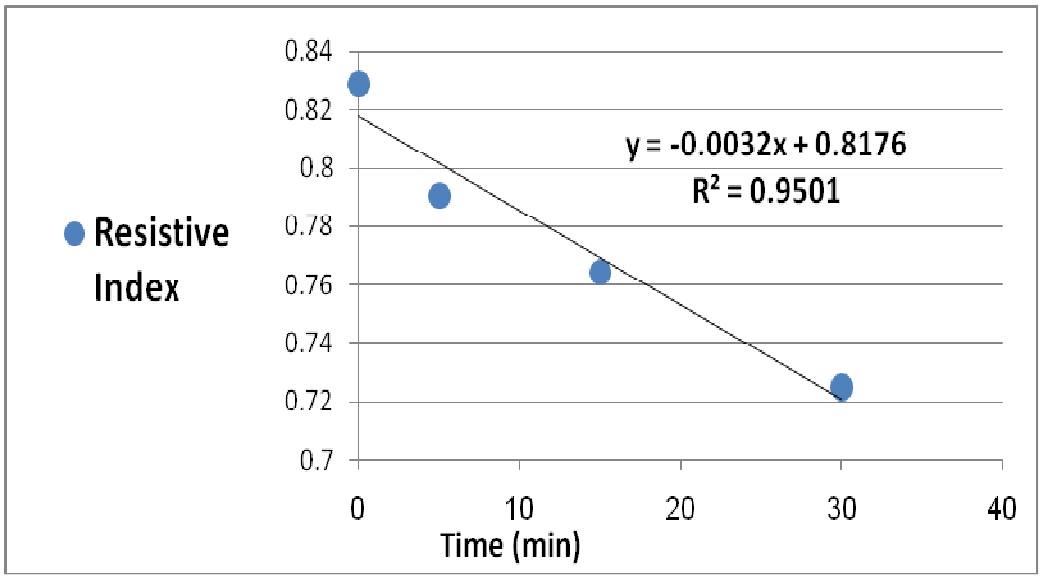


Figure:4.12: Carbohydrate meal correlation of resistive index versus time (0-30 mins)

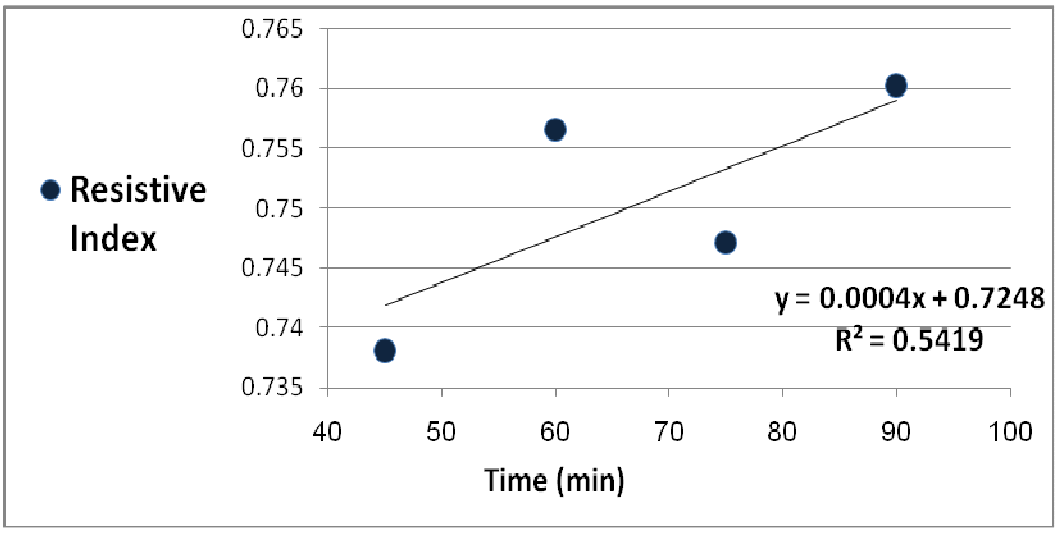


Figure:4.13: Carbohydrate meal correlation of resistive index versus time (30-90 mins)

Table:4.1: Paired sample t test of SMA diameter in fasting state & postprandial in versus time in fatty meal.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	SMADiameterFasting - SMADiameter5min	-.5490	.3261	.0326	-.6137	-.4843	-16.834	99	.0000
Pair 2	SMADiameterFasting - SMADiameter15min	-.9320	.3203	.0320	-.9956	-.8684	-29.096	99	.0000
Pair 3	SMADiameterFasting - SMADiameter30min	-1.1250	.4061	.0406	-1.2056	-1.0444	-27.702	99	.0000
Pair 4	SMADiameterFasting - SMADiameter45min	-1.2110	.3967	.0397	-1.2897	-1.1323	-30.529	99	.0000
Pair 5	SMADiameterFasting - SMADiameter60min	-1.5150	.4482	.0448	-1.6039	-1.4261	-33.802	99	.0000
Pair 6	SMADiameterFasting - SMADiameter75min	-1.3540	.4403	.0440	-1.4414	-1.2666	-30.755	99	.0000
Pair 7	SMA Diameter Fasting - SMADiameter90min	-1.2650	.3968	.0397	-1.3437	-1.1863	-31.880	99	.0000

Table:4.2: Paired sample t test of SMA blood flow velocity in fasting state & postprandial in versus time in fatty meal

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	SMABloodFlowVolumeFasting - SMABloodFlowVolume5min	-47.6600	36.8717	3.6872	-54.9761	-40.3439	-12.926	99	.0000
Pair 2	SMABloodFlowVolumeFasting - SMABloodFlowVolume15min	-189.4100	75.8424	7.5842	-204.4588	-174.3612	-24.974	99	.0000
Pair 3	SMABloodFlowVolumeFasting - SMABloodFlowVolume30min	-338.5900	103.7529	10.3753	-359.1768	-318.0032	-32.634	99	.0000
Pair 4	SMABloodFlowVolumeFasting - SMABloodFlowVolume45min	-432.7800	127.5403	12.7540	-458.0868	-407.4732	-33.933	99	.0000
Pair 5	SMABloodFlowVolumeFasting - SMABloodFlowVolume60min	-504.5300	134.9183	13.4918	-531.3007	-477.7593	-37.395	99	.0000
Pair 6	SMABloodFlowVolumeFasting - SMABloodFlowVolume75min	-406.1100	118.2742	11.8274	-429.5782	-382.6418	-34.336	99	.0000
Pair 7	SMABloodFlowVolumeFasting - SMABloodFlowVolume90min	-440.5300	128.8504	12.8850	-466.0967	-414.9633	-34.189	99	.0000

Table:4.3: Paired sample t test of SMA peak systolic velocity in fasting state & postprandial in versus time in fatty meal.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	PeakSystolicVelocityFasting - PeakSystolicVelocity5min	-13.7700	13.4223	1.3422	-16.4333	-11.1067	-10.259	99	.0000
Pair 2	PeakSystolicVelocityFasting - PeakSystolicVelocity15min	-21.1600	14.0229	1.4023	-23.9424	-18.3776	-15.090	99	.0000
Pair 3	PeakSystolicVelocityFasting - PeakSystolicVelocity30min	-33.4800	14.9811	1.4981	-36.4526	-30.5074	-22.348	99	.0000
Pair 4	PeakSystolicVelocityFasting - PeakSystolicVelocity45min	-43.6900	14.1783	1.4178	-46.5033	-40.8767	-30.815	99	.0000
Pair 5	PeakSystolicVelocityFasting - PeakSystolicVelocity60min	-56.5700	14.3444	1.4344	-59.4162	-53.7238	-39.437	99	.0000
Pair 6	PeakSystolicVelocityFasting - PeakSystolicVelocity75min	-45.9300	17.2054	1.7205	-49.3439	-42.5161	-26.695	99	.0000
Pair 7	PeakSystolicVelocityFasting - PeakSystolicVelocity90min	-48.2300	15.0238	1.5024	-51.2110	-45.2490	-32.102	99	.0000

Table:4.4: Paired sample t test of SMA resistive index in fasting state & postprandial in versus time in fatty meal.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	Resistive Index Fasting - ResistiveIndex5min	.01230	.01118	.00112	.01008	.01452	11.003	99	.0000
Pair 2	Resistive Index Fasting - ResistiveIndex15min	.03150	.01493	.00149	.02854	.03446	21.095	99	.0000
Pair 3	Resistive Index Fasting - ResistiveIndex30min	.08690	.02585	.00258	.08177	.09203	33.621	99	.0000
Pair 4	Resistive Index Fasting - ResistiveIndex45min	.12240	.02633	.00263	.11718	.12762	46.490	99	.0000
Pair 5	Resistive Index Fasting - ResistiveIndex60min	.15220	.02729	.00273	.14679	.15761	55.777	99	.0000
Pair 6	Resistive Index Fasting - ResistiveIndex75min	.12200	.02778	.00278	.11649	.12751	43.917	99	.0000
Pair 7	Resistive Index Fasting - ResistiveIndex90min	.11070	.02746	.00275	.10525	.11615	40.313	99	.0000

Table:4.5: Paired sample t test of SMA diameter in fasting state & postprandial in versus time in carbohydrate meal

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	SMA Diameter Fasting - SMA Diameter 5min	-0.37	0.3252	0.03252	-0.43453	-0.30547	-11.377	99	.0000
Pair 2	SMA Diameter Fasting - SMA Diameter 15min	-0.662	0.40819	0.04082	-0.74299	-0.58101	-16.218	99	.0000
Pair 3	SMA Diameter Fasting - SMA Diameter 30min	-1.188	2.05885	0.20588	-1.59652	-0.77948	-5.77	99	.0000
Pair 4	SMA Diameter Fasting - SMA Diameter 45min	-0.972	0.42094	0.04209	-1.05552	-0.88848	-23.091	99	.0000
Pair 5	SMA Diameter Fasting - SMA Diameter 60min	-0.868	0.38661	0.03866	-0.94471	-0.79129	-22.451	99	.0000
Pair 6	SMA Diameter Fasting - SMA Diameter 75min	-0.755	0.3927	0.03927	-0.83292	-0.67708	-19.226	99	.0000
Pair 7	SMA Diameter Fasting - SMA Diameter 90min	-0.476	0.4313755	0.0431376	-0.5615943	-0.3904057	-11.034469	99	.0000

Table:4.6: Paired sample t test of SMA blood flow volume in fasting state & postprandial in versus time in carbohydrate meal.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 5min	-115.79	78.394663	7.8394663	-131.3452	-100.2348	-14.770138	99	.0000
Pair 2	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 15min	-293.27	99.449988	9.9449988	-313.00304	-273.53696	-29.489194	99	.0000
Pair 3	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 30min	-378.04	105.44795	10.544795	-398.96316	-357.11684	-35.850861	99	.0000
Pair 4	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 45min	-332.1	102.6613	10.26613	-352.47023	-311.72977	-32.349092	99	.0000
Pair 5	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 60min	-252.99	103.93952	10.393952	-273.61386	-232.36614	-24.340116	99	.0000
Pair 6	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 75min	-167.99	113.40608	11.340608	-190.49223	-145.48777	-14.813139	99	.0000
Pair 7	SMA Blood Flow Volume Fasting - SMA Blood Flow Volume 90min	-132.67	111.7734	11.17734	-154.84827	-110.49173	-11.86955	99	.0000

Table:4.7: Paired sample t test of SMA peak systolic velocity in fasting state & postprandial in versus time in carbohydrate meal.

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	Peak Systolic Velocity Fasting - Peak Systolic Velocity 5min	-9.46	6.58345	0.65835	-10.7663	-8.1537	-14.369	99	.0000
Pair 2	Peak Systolic Velocity Fasting - Peak Systolic Velocity 15min	-21.69	1.29E+01	1.28721	-24.2441	-19.1359	-16.85	99	.0000
Pair 3	Peak Systolic Velocity Fasting - Peak Systolic Velocity 30min	-34.52	13.8144	1.38144	-37.2611	-31.7789	-24.988	99	.0000
Pair 4	Peak Systolic Velocity Fasting - Peak Systolic Velocity 45min	-27.7	16.8184	1.68184	-31.0371	-24.3629	-16.47	99	.0000
Pair 5	Peak Systolic Velocity Fasting - Peak Systolic Velocity 60min	-22.68	16.7476	1.67476	-26.0031	-19.3569	-13.542	99	.0000
Pair 6	Peak Systolic Velocity Fasting - Peak Systolic Velocity 75min	-27.43	16.5256	1.65256	-30.709	-24.151	-16.598	99	.0000
Pair 7	Peak Systolic Velocity Fasting - Peak Systolic Velocity 90min	-19.49	16.4012	1.64012	-22.7444	-16.2356	-11.883	99	.0000

Table:4.8: Paired sample t test of SMA resistive index in fasting state & postprandial in versus time in carbohydrate meal

Paired Samples Test									
		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	of the Difference Lower	of the Difference Upper			
Pair 1	Resistive Index Fasting - Resistive Index 5min	0.0383	0.01959	0.00196	0.03441	0.04219	19.546	99	.0000
Pair 2	Resistive Index Fasting - Resistive Index 15min	0.0645	0.02844	0.00284	0.05886	0.07014	22.679	99	.0000
Pair 3	Resistive Index Fasting - Resistive Index 30min	0.1039	0.04107	0.00411	0.09575	0.11205	25.299	99	.0000
Pair 4	Resistive Index Fasting - Resistive Index 45min	0.091	0.04294	0.00429	0.08248	0.09952	21.195	99	.0000
Pair 5	Resistive Index Fasting - Resistive Index 60min	0.0724	0.04015	0.00402	0.06443	0.08037	18.03	99	.0000
Pair 6	Resistive Index Fasting - Resistive Index 75min	0.082	0.03181	0.00318	0.07569	0.08831	25.775	99	.0000
Pair 7	Resistive Index Fasting - Resistive Index 90min	0.0688	0.03503	0.0035	0.06185	0.07575	19.642	99	.0000

Chapter five

Discussion, conclusion, and recommendation

5.1 Discussion

The current study was conducted in Taif city in order to assess the SMA using ultrasonography to demonstrate the effects of different meals response on the SMA.

From the previous results' chapter researcher found that first meal and second meal have semi similar correlation results for different SMA US indices. Also the third meal and the fourth meal have almost the same correlation results. So researcher discussion executed just on The first meal which was Grilled goat meat meal (545 Kcal) and the majority of meal composed from fats, and the second meal which was Beans meal (477 Kcal) and the majority of meal composed from carbohydrates as shown in chapter thee table:31, that had different correlation in order to rich the discussion and not lost the efforts.

Figure 4-1 in the previous chapter showed the BMI and age from this figure researcher found that little part of the sample was overweight according to report by National institutes of health (NHLBI. 2013).

The first and second meals were investigated for 100 health volunteers by Doppler ultrasound as explained in the previous chapter (Results) . Different graphs were derived with various correlation coefficient shown in each figures.

Normal values could be easy derived from the equation and coefficient values (R^2). In each figure, mean value was taken for different SMA indices and then correlated to various time.

Up to the knowledge of the researcher, little literature were published worldwide regarding normal measurements values or figures pre and post certain meals for SMA indices , or even there were no established data

that were verified by any scientific ultrasound committee or society concerning the results values of this study. The little amount of literature and previous studies that were obtained by researcher had less sample number.

SMA Diameter

The mean DM in basal values of SMA did not differ significantly for first and second meals, after intake of two meals, increased in SMADM in response to intake of meals were shown in the figures:(4.2, and 4.6), An immediate and slow increased in SMADM was observed, and the maximum was reached within 60 to 30 minutes of taking fatty and carbohydrate meal respectively. For fatty meal at 90 mins after intake SMADM slightly decreased neared to peak value but till significantly at ($p<0$ 05) was shown in table:4.1. This was agree with study by **Sieber, et al,(1991)** which is reported that the mean diameter in basal values of SMA did not differ significantly.

After food intake, the diameter of the superior mesenteric artery increased significantly ($p<0.05$) and reached a maximum 60 minutes after food intake. In the other hand the SMADM for second meal was increased in response to intake of carbohydrate meal and reached maximum value at 30 mins significantly ($p<0.05$) as shown in figure:4.6, and then decreased to value near the baseline but still significant in figure:4.7. In the case of carbohydrate meal, there was change in DM of SMA after intake of meal from 0 to 90 mins, this change was significantly using paired t-test at $p=0.05$ with $p<0.0001$ shown in table:4.5.

Table:4.1 showed the t-test for SMA Diameter versus time for fatty meal, there is change in SMA Diameter in fasting and postprandial state. This change was significantly using paired sample t-test at P= 0.05 with P <0.0001. The SMADM was slightly increased after two meals but was

still significantly. This increased was resulting in an increasing in SMA BV and BFV, which were seen after meal intake. At sixty minutes SMADM decreased gradually, but was still significantly (p<005) above basal values till to 90 mins.

These results were agree with **Sieber et al, (1992)**, they found that the increase in blood flow was mainly the result of an increase in velocity, as sharp increases (p<0 05) in Time Average Velocity (TAV) were seen after both liquid and solid food intake. Liquid and solid oral test meals induced small but significant (p<0 05) increases in vessel diameter.

The correlation of the Mean DM of SMA for the first meal versus time (0- 90 mins) was shown in Figure:4.2, and from this figure normal value for the DM of the SMA can be easily estimated at any time from 0 to 90 mins before or after the first meal taken using the formula showed in the figure ($y = 0.012 * time + 6.318$), where y was normal value for the DM of the SMA, and (6.318) was the baseline value for the DM of the SMA at fasting state.

Also the normal value for BFV, PSV, and RI of the SMA can be estimated at any time before or after the first meal taken using the formulas showed in the figures **:4.3, 4.4, and 4.5** ($y = 5.026 * time + 376.6$), ($y = 0.522 * time + 129.6$) , **and** ($y = 0.001 * time + 0.811$), where y was normal value for BFV, PSV, and RI of the SMA respectively. This normal values of SMA for DM, and RI in neared to values that were reported by Wilhelm Schäberle, (2004), he found that the normal values for DM, and RI of SMA including preprandial and Postprandial measurements by Duplex parameters was (5-8 cm), and (0.75-0.9) of SMADM for DM, and RI respectively.

SMABFV:

Basal superior mesenteric artery blood flow volume (SMABFV) measurements were approximately near to similar in values for first and second meals. The mean basal SMABFV is 376 ml/min for fatty meal, 344 ml/min for carbohydrate meal, and increase in response to the oral intake of two meals is shown in [Fig:4.3 & Fig:4.7]. this was matched with **Elsamani1, et al, (2016),** They was state that the basal superior mesenteric artery blood flow volume (SMABFV) measurements were approximately near to similar in values for different four meals type.

Figure 4-3 showed correlation of the first meal Mean Blood Flow Volume (MBFV) of SMA versus time (0-90mins). Also the MBFV of SMA in case of fatty and carbohydrate meals, there was change in SMABF after intake of meals. This changes were significant using paired t-test at $p=0.05$ with $p<0.0001$ shown in tables:(4.2, and 4.6).

An immediate and marked increase in mesenteric blood flow was observed and the maximum was reached within 30-60 minutes of taking two meals, and This was agree with **E.Abd Elrahim, et al, (2016),** they found there were an immediate and marked increase in mesenteric blood flow was observed, and the maximum was reached within 30-60 minutes of taking four meals, this differ with. Thirty minutes after carbohydrate and jam and Boiled meal intake, SMABFV fall towards basal values and was not significantly different from baseline at 105 minutes post pranially. (359ml/min for meal 2, and 433ml/min for meal 4). In contrast sixty minutes after fatty and Cheese and yoghurt meal intake, SMABFV decreased gradually, but was still significantly ($p<005$) above basal values (656ml/min for meal1, 779ml/min for meal 4) at the 105 minutes.

The above metion results were also agree to some extend agree with **Sidery, et al, (1994),** they stated that fasted superior mesenteric artery

blood flow was 346 ml/min before the high carbohydrate meal and 351 ml/min before the high fat meal.

A peak blood flow of 611 ml/min was achieved 20 minutes after ingestion of the high carbohydrate meal. In contrast, a peak blood flow of 715 ml/min was reached 40 minutes after fat .on the other hands this differ with **(Someya, et al, 2008)** they found that the baseline values did not differ between experimental and control trials except for BF in the forearm. Forty five minutes after second meal intake, SMABFV fall towards basal values and was not significantly different from baseline at 90 minutes post pranially (425ml/min for carbohydrate meal) [Fig:4.3]. In contrast sixty minutes after fatty meal intake, SMABFV decreased gradually, but was still significantly ($p<005$) above basal values (712 ml/min for fatty meal) [Fig:4.8] at the 90 minutes, this is agree with (Quarti et al, 2002), they found that, the mean SMA diameter was significantly lower in patients than in healthy control ($p < 0.001$), in the two groups, the mean blood velocity was not significantly difference between the two groups, whereas mean SMA blood follow was significantly lower in patients. Also it was consistent to some extent with the (Qamar, et al, (1988) they found that, the maximal responses were not significantly different but were reached at different times: carbohydrate, 64% at 15 min; fat, 60% at 30 min, and also the response to the fat meal was significantly slower than the response to the carbohydrate. These results indicate that the chemical nature of the meal and not the volume persist a significant factor determining postprandial mesenteric hyperaemia. On the other hand, Dauzat, et al (1994) were agrees to some extent with this study, they were measured splanchnic and hepatic circulation in 30 healthy volunteers, using Doppler ultrasonography initially, then 15, 30, 45, and 60 min after the ingestion

of a standard balanced liquid meal, and found that the Postprandial haemodynamic changes were maximal 30 min after the meal; at that time, mesenteric artery RI decreased significantly.

As the first meal almost composed from fats, and the second meal almost composed from carbohydrates mention in chapter three Table:3.1, the peak value of SMADM reached at 60 mins after fatty meal and 30 mins after carbohydrate meal, this due to delaying intestinal absorption in case of fats comparing to carbohydrate.

This agree with **Sidery, et al, (1994)**, they reported that there were a significant curvilinear relation between the superior mesenteric artery blood flow response and gastric emptying after carbohydrate (r^2=094) and no relation at all after fat. They conclude that ingestion of meals with a high fat content slows gastric emptying compared with meals with a high carbohydrate content in healthy volunteers. The relation, however, between the volume of meal remaining in the stomach and the mesenteric response was considerably different after the two meals.

SMAPSV:

The mean fasting SMA Peak Systolic Velocity (PSV) did not differ among first and second meals (129.6 cm/sec and 129.5 cm/sec) respectively as shown in figures:(4.4, and 4.10). Postprandial PSV increased in two meals was significantly using paired t-test at p=0.05 with p<0.0001 shown in tables:(4.3, and 4.7). There PSVs of SMA for two meals was immediate and markedly increased postprandially to reached maximum values at 60 mins (174cm/sec), and 30 mins (163 cm/sec) respectively, this increased was flowed by increasing in SMADM in response to intake of meals as motioned above, this was agree with **E.Abd Elrahim, et al, (2016),** they found that the fatty and 3rd meals in which the maximum PSV reached at 30 minutes and then

decrease gradually toward baseline value. In contrast in carbohydrate , and jam and Boiled meals the maximum PSV reached at 45, 60 minutes consequently in carbohydrate , and jam and Boiled meals , then slightly decrease but still significant at the end of these meals at 105 minutes postprandially. Also this was differ with (M. J. Perko, 2001), they found that all types of food (mixed, carbohydrate, fat and protein) produce increases in blood velocity and diameter of the artery and as a consequence, elevate SMA blood flow. By contrast, water or isotonic NaCl do not influence DU parameters. Alterations in flow parameters are most pronounced about 60 min after intake of a mixed meal. The diastolic systemic blood pressure selectively decreases reflecting peripheral vaso-relaxation which, together with increases in heart rate, elevates cardiac output. In the SMA peak systolic velocity doubles but the diastolic velocity increases 10-fold, reflecting the high sensitivity of this parameter to changes in peripheral resistance. The diameter of the SMA also increases, resulting in an increase in mesenteric blood flow. Figure:(4.4, and 4.11) showed slightly decreased in SMAPSV for fatty and carbohydrate meals, the decreased after 60 mins was neared to the maximum value and still significant for fatty meal, and gradually decrease after 30 mins towards the baseline but also still significant at the end of these meals at 90 minutes postprandially, this was matched with **Elsamani1, et al, (2016),** they reported that the PSV of the SMA changed with time to reveal a main effect of four meals types and the alteration of the PSV of the SMA for fatty and 3rd meals was decrease gradually toward baseline value. In contrast for carbohydrate , and jam and Boiled meals there was slightly decrease in the PSV of the SMA but still significant at the end of these meals.

SMARI:

In order to increased in SMADM and SMABF the Resistive Index RI in the SMA tended to decrease in fatty and carbohydrate meals, and the maximum decrease in mean RI for fatty meal reached at 60 minutes after intake meal, then start to increase but still significant. In contrast the maximum decrease in mean RI for carbohydrate meal was reached at 30 minutes after meals after intake of meal, also then increased gradually toward the value that nearly the baseline was shown in Figure (4.5, and 4.9).

This was agree with **Dauzat, et al (1994),** they stated that the SMARI decreased significantly and persistently after meal. The decreased in SMARI for two meals was significant using paired t-test at p=0.05 with p<0.0001 shown in tables:(4.4, and 4.8). increased still significant (P = 0.60) after end of the two meals at 90 minutes. Also This was matches with Taourel, *et al.* (1998), they found that The RI of the SMA decreased significantly (p <0.05) after the meal in both healthy subjects (13%) and cirrhotic patients (8%). The postprandial decrease was significantly less pronounced (p < 0.05) in patients with cirrhosis than in healthy subjects. And confirmation of previous studies, **Taourel, et al (1998),** they state that the RI was decreased significantly (p < 0.05) after the meal in both the healthy (13%) and cirrhotic (8%) subjects, but the postprandial decrease was significantly less pronounced (p < 0.05) in cirrhotic patients than in healthy subjects. In the other hands the health subject was appeared singnificant decreased in RI pastprandially, and this matched with **Hornum, , et al, (2006), they stated that the** both groups of patients with chronic pancreatitis unexpectedly had lower fasting RI than controls, 0.818 and 0.815 vs 0.851, (p=0.028 and p=0.0030, respectively). Postprandialy there was significantly less decrease in RI

(less increase in flow) in patients with exocrine insufficiency than in controls, 0.055 vs 0.099, p=0.0047. There was a significant trend for a less pronounced postprandial decrease in RI with more impaired pancreatic function (p=0.0036).

5.2 Conclusion:

- Researcher concluded that the different meals components have different effects on SMA circulation ,that may reflecting in the human health.
- Researcher revealed that the normal values of SMA for DM, BFV, PSV, and RI were estimated from each correlation factor found in the specific figure, and this can be easily applicable and taken as the reference values for each normal health subjects.
- Researcher concluded that the fatty meals had slowing in digestion more than carbohydrate.
- Also researcher concluded that the peak value of SMA for DM, BFV, PSV, and RI were found in case of first meal at 60 mins after intake of meal, and in case of second meal at 30 mins after intake of meal.

5.3 Recommendation:

- Researcher recommended more studies in this field for other abnormal patients.
- Also researcher advices more extended studies as this the first study conducted in Sudan or even middle east.
- The estimated values of SMA for different indices, may used as guidance to clarify the pathology which may resulted by effects of the meals in SMA.

Appendix

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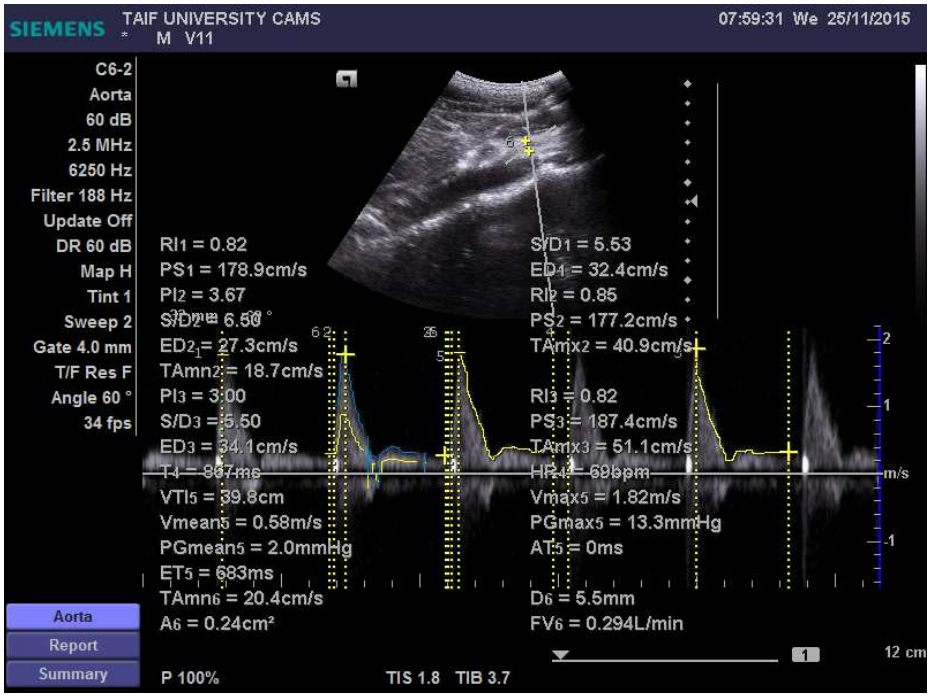

Image No (1) *Duplex sonogram of the SMA showing resistive index (0.82) for fatty meal at fasting state with other parameter*

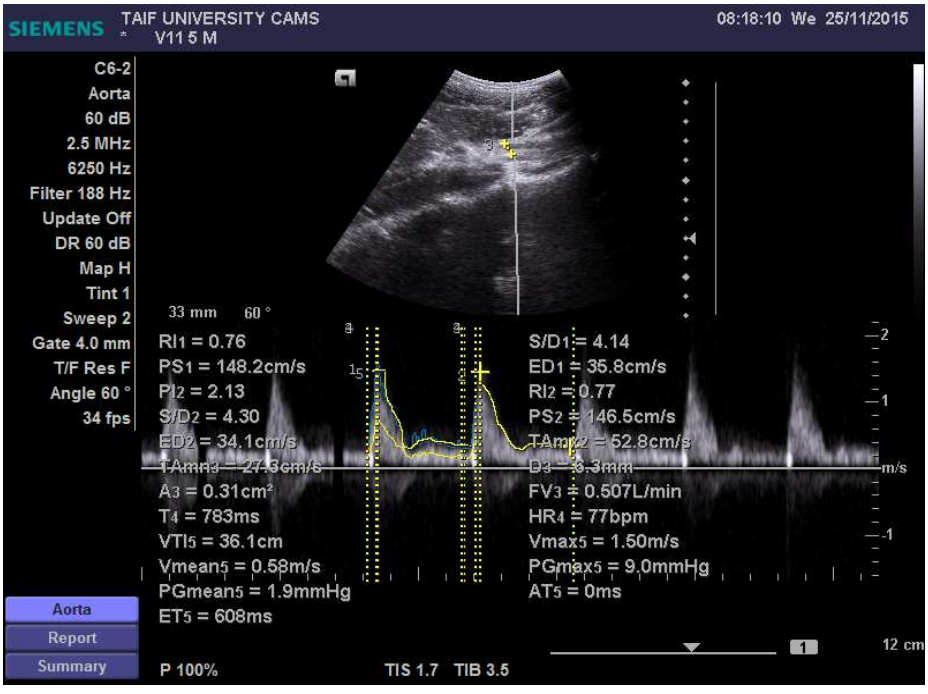

Image No (2) *Duplex sonogram of the SMA showing resistive index (0.76) for carbohydrate meal after 15 min with other parameter*

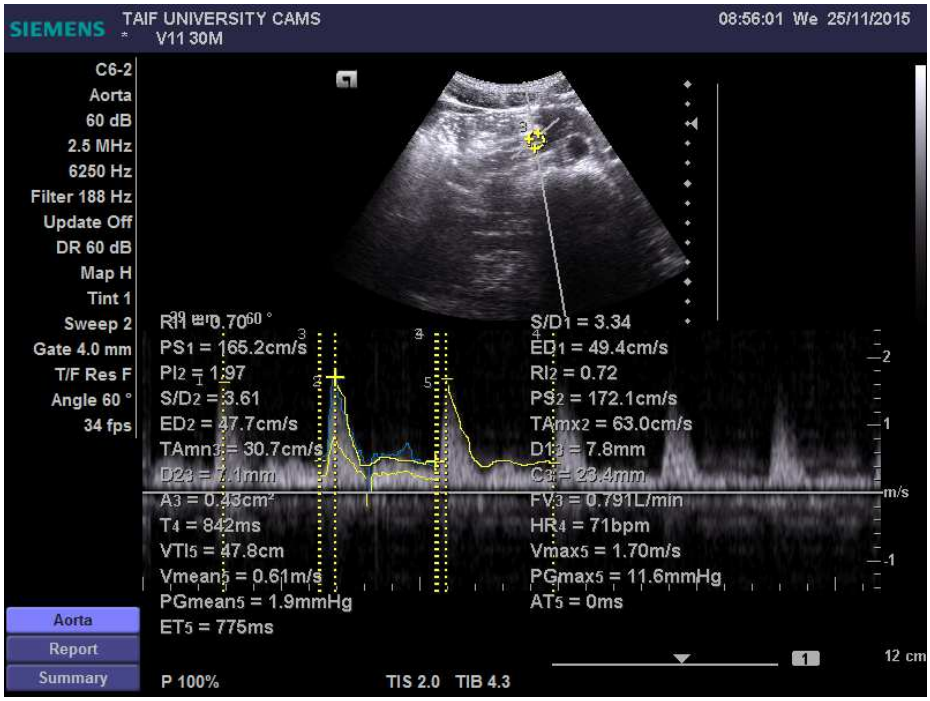

Image No (3)*Duplex sonogram of the SMA showing showing resistive index (0.70) for carbohydrate meal after 30 min with other parameter*

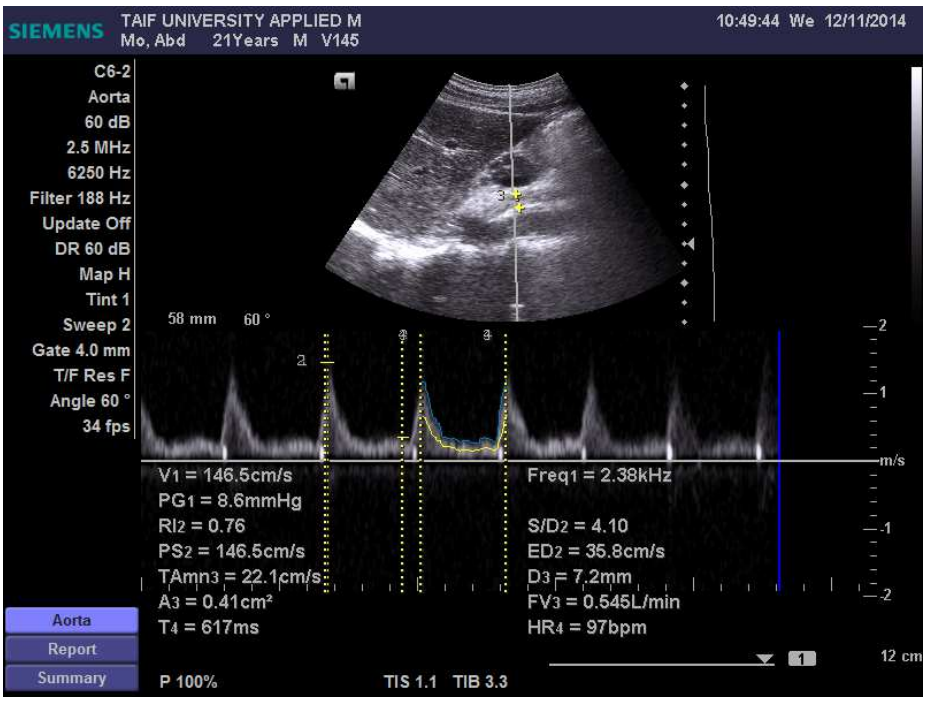

Image No (4) *Duplex sonogram of the SMA showing peck systolic velocity (145.5 cm/s) for fatty meal at fasting state with other parameter*

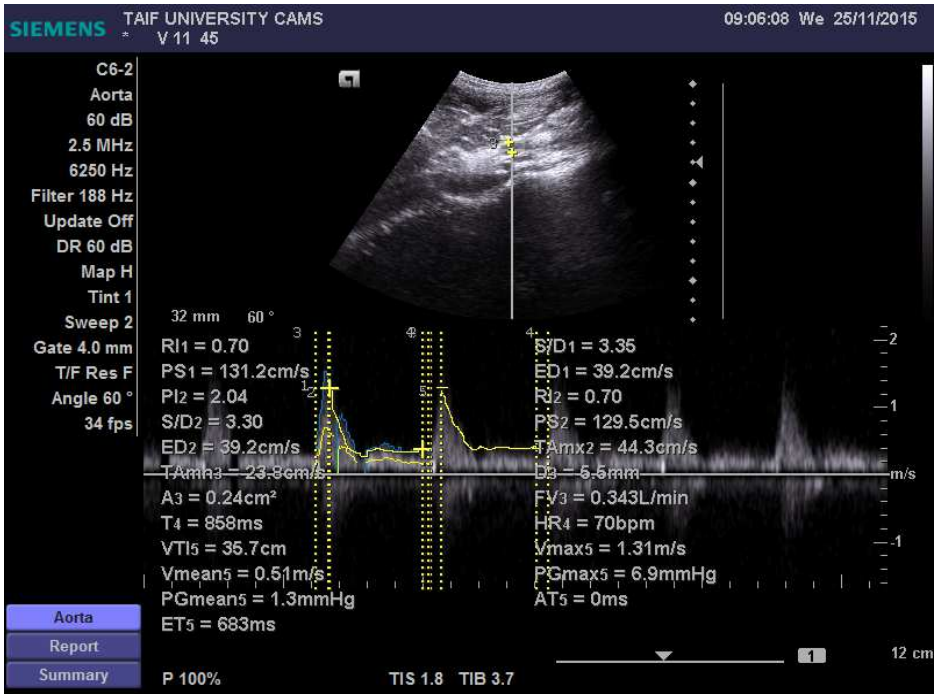

Image No (5) *Duplex sonogram of the SMA showing resistive index (0.70) for fatty meal after 45 min with other parameter*

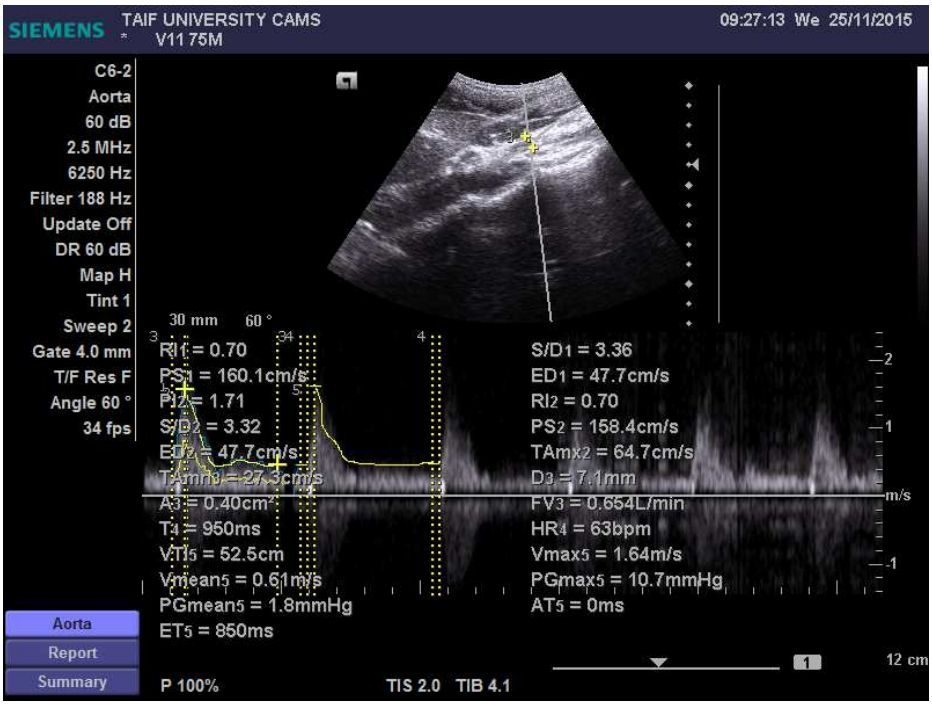

Image No (6) *Duplex sonogram of the SMA showing pulsitilty index (1.71) for carbohydrate meal after 75 min with other parameter*

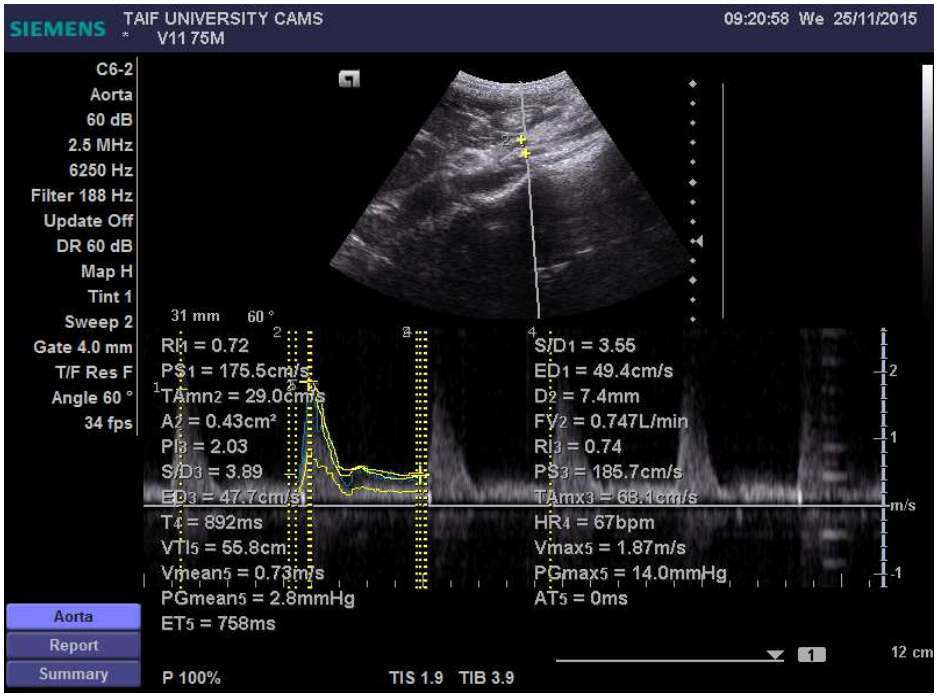

Image No (7) *Duplex sonogram of the SMA showing end diastolic velocity (49.4cm/s) for carbohydrate meal after 75 min.*

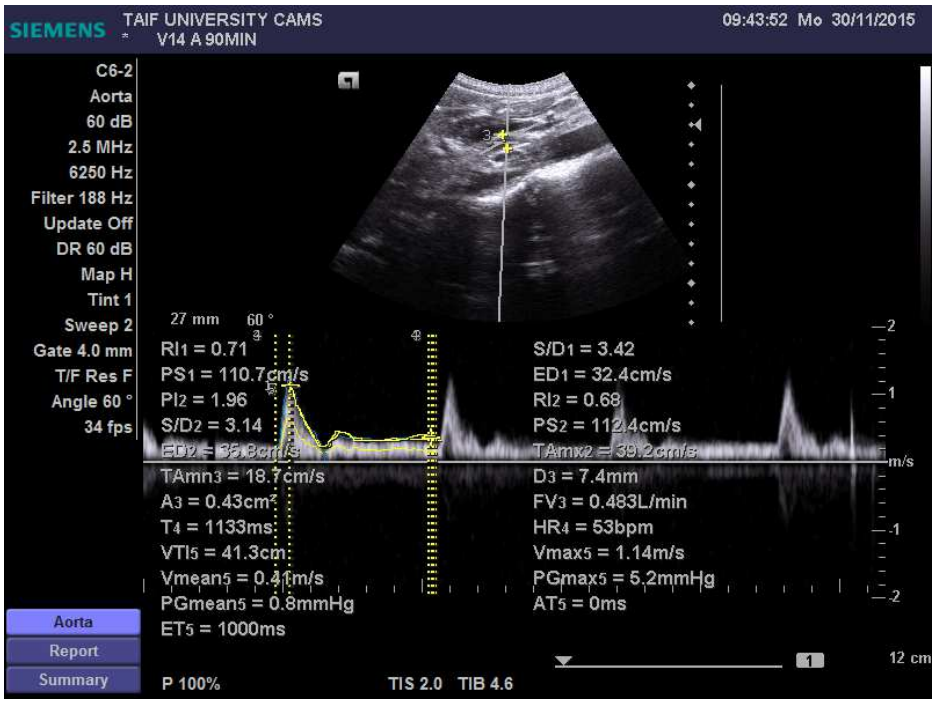

Image No (8) *Duplex sonogram of the SMA showing SMA diameter (7.4 mm) for fatty meal after 90 min with other parameter*

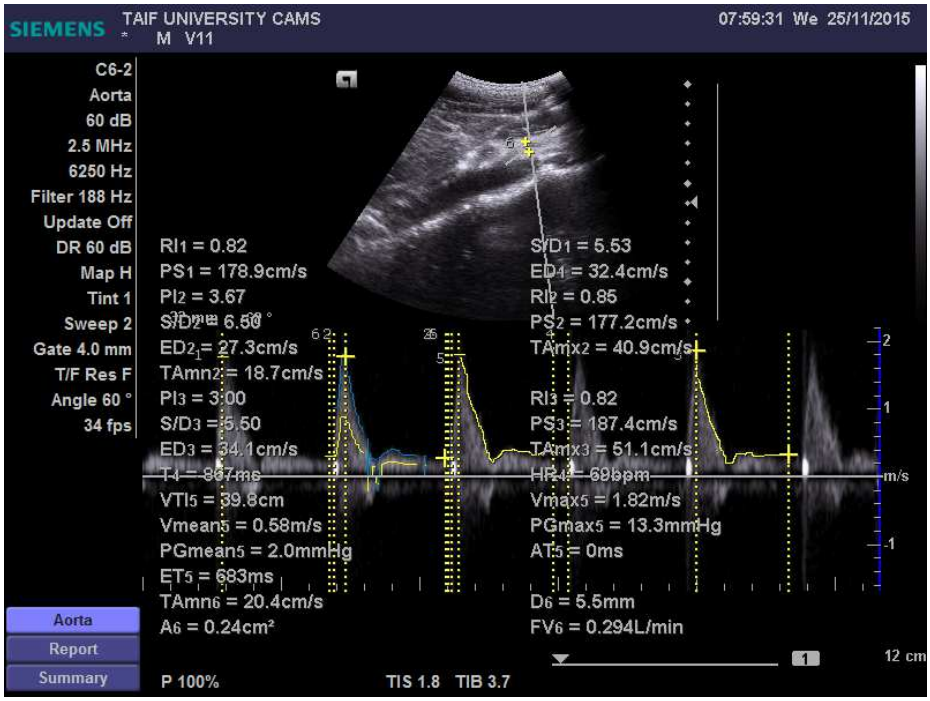

Image No (9) *Duplex sonogram of the SMA showing SMA blood flow volume (0.294 L/min) for fatty meal after 15 min with other parameter*

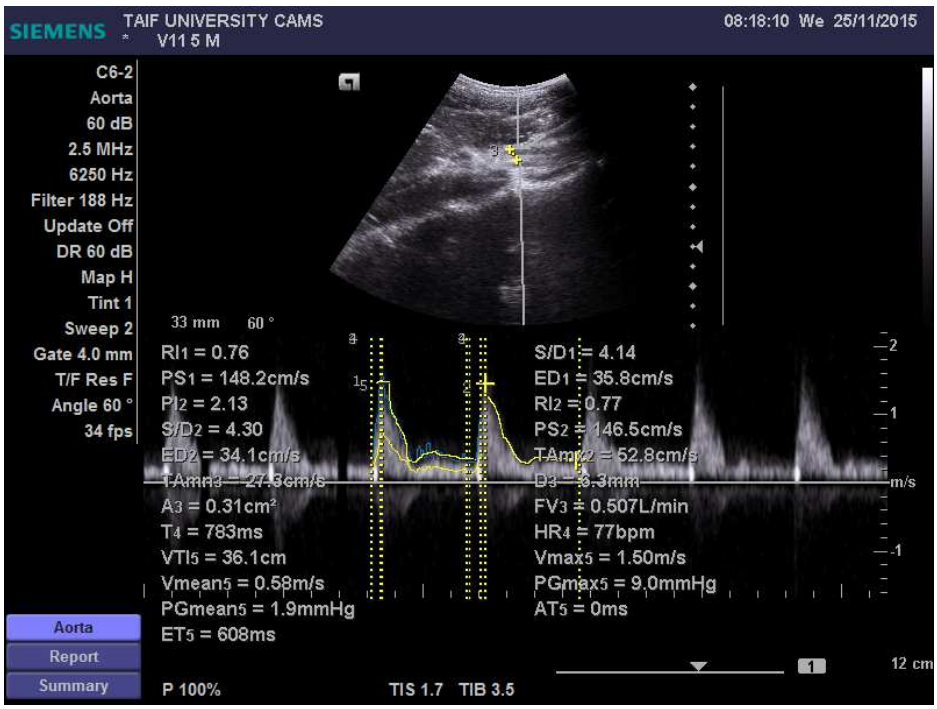

Image No (10) *Duplex sonogram of the SMA showing resistive index(0.76) for carbohydrate meal after 15 min with other parameter*

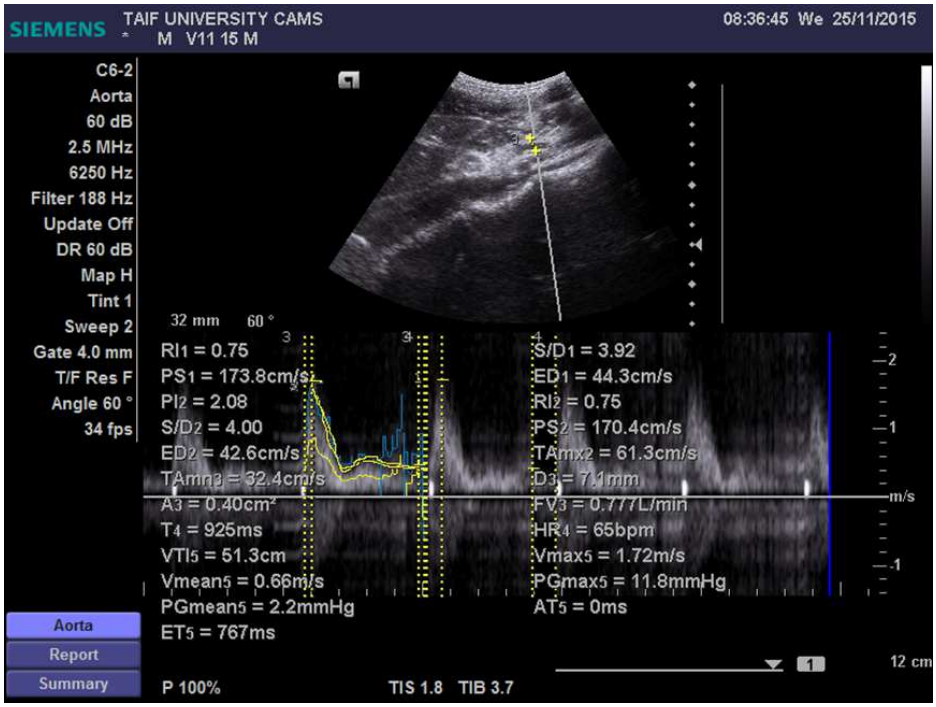

Image No (11) *Duplex sonogram of the SMA showing resistive index(0.75) for fatty meal after 15 min with other parameter*

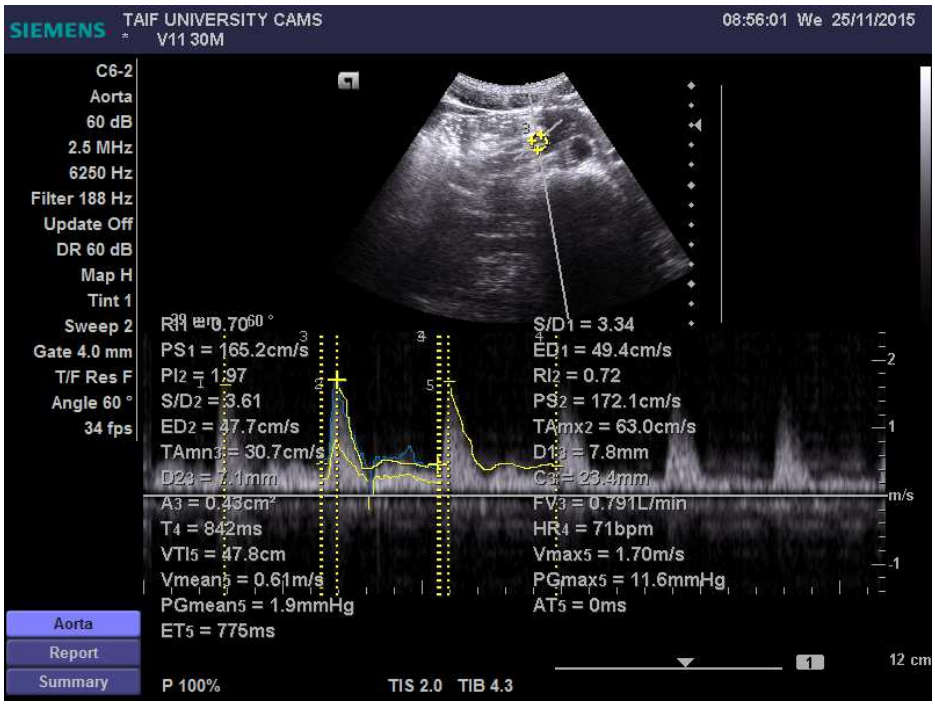

Image No (12) *Duplex sonogram of the SMA showing resistive index(0.70) for carbohydrate meal after 15 min with other parameter*

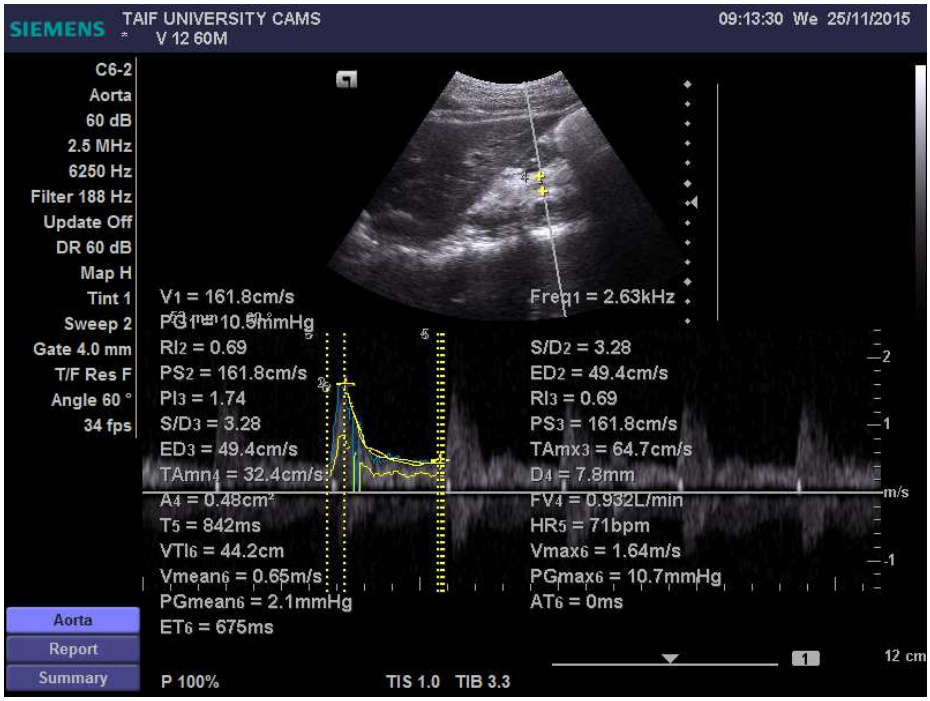

Image No (13) *Duplex sonogram of the SMA showing peak systolic velocity (161.8cm/s) for fatty meal after 60 min with other parameter*

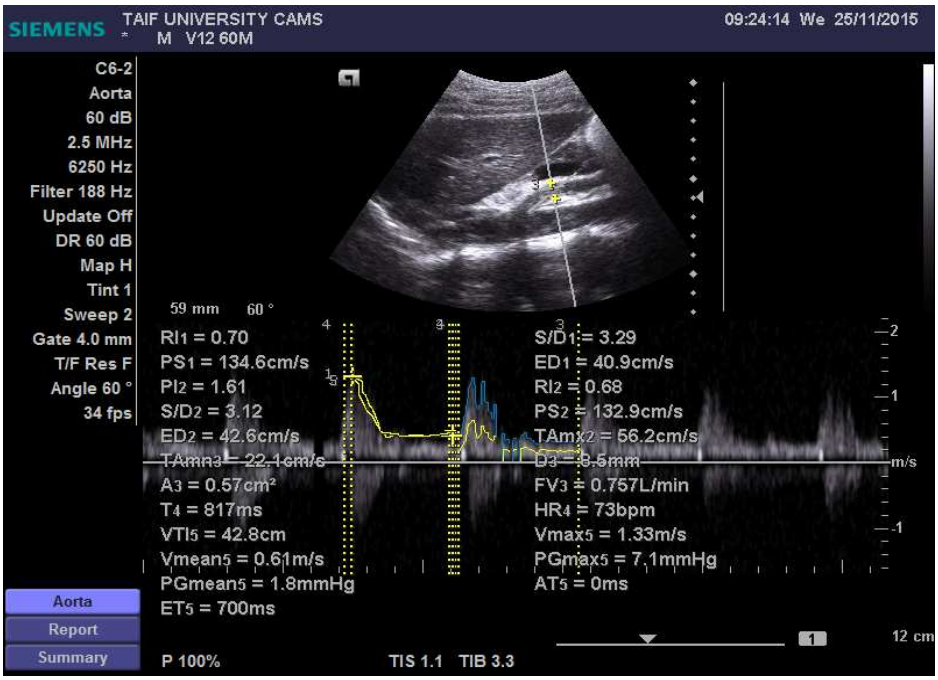

Image No (14) *Duplex sonogram of the SMA showing mean velocity (61 cm/s) for fatty meal after 60 min with other parameter*

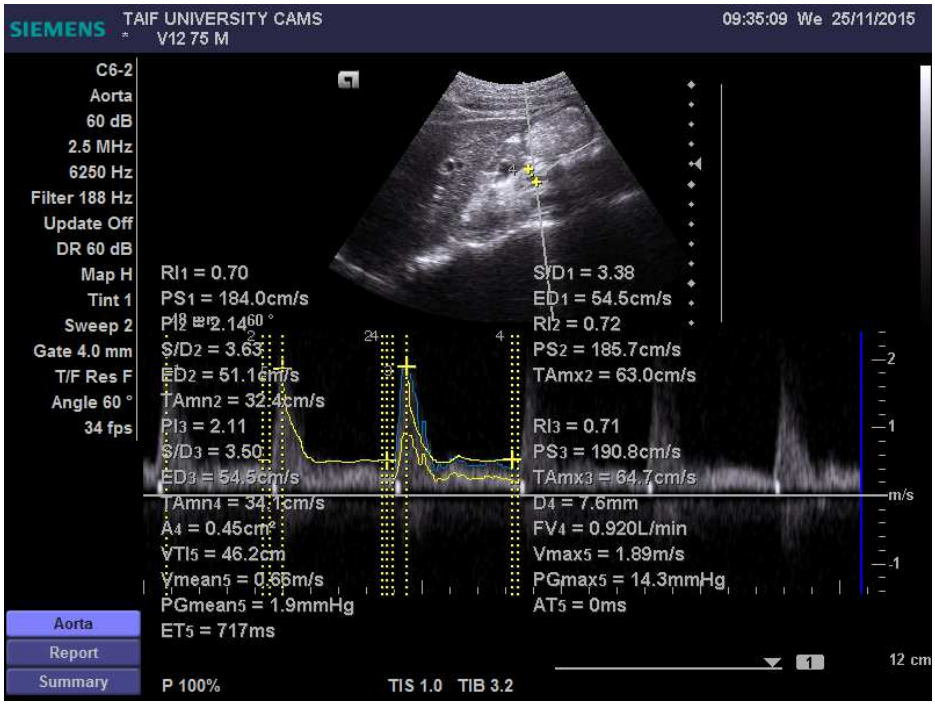

Image No (15) *Duplex sonogram of the SMA showing end diastolic velocity (54.5cm/s) for carbohydrate meal after 75 min.*

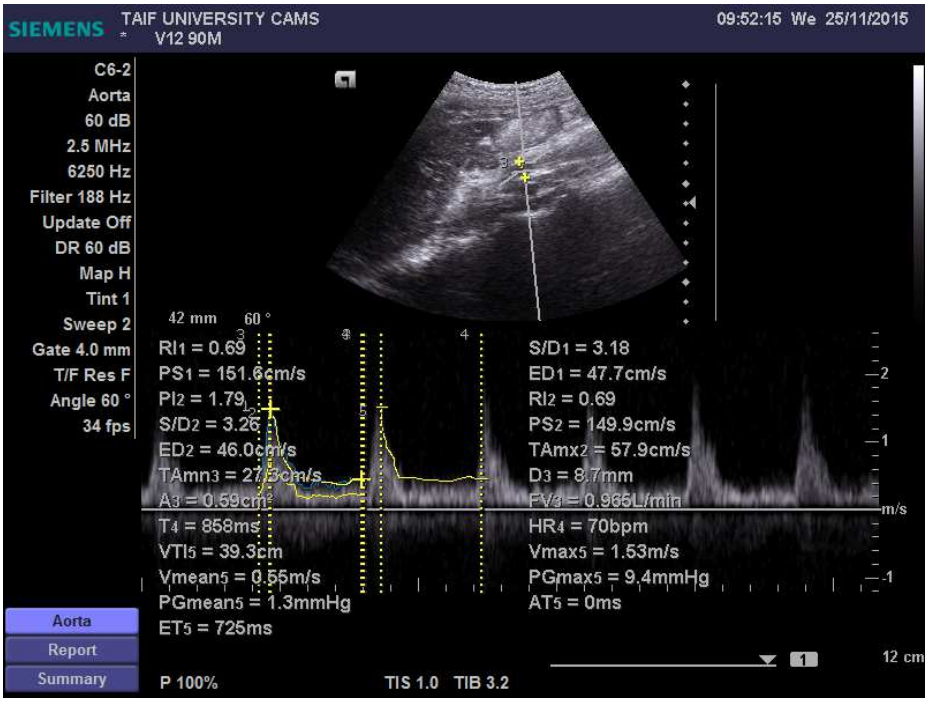

Image No (16) *Duplex sonogram of the SMA showing peak systolic velocity(151.6cm/s) for carbohydrate meal after 15 min.*

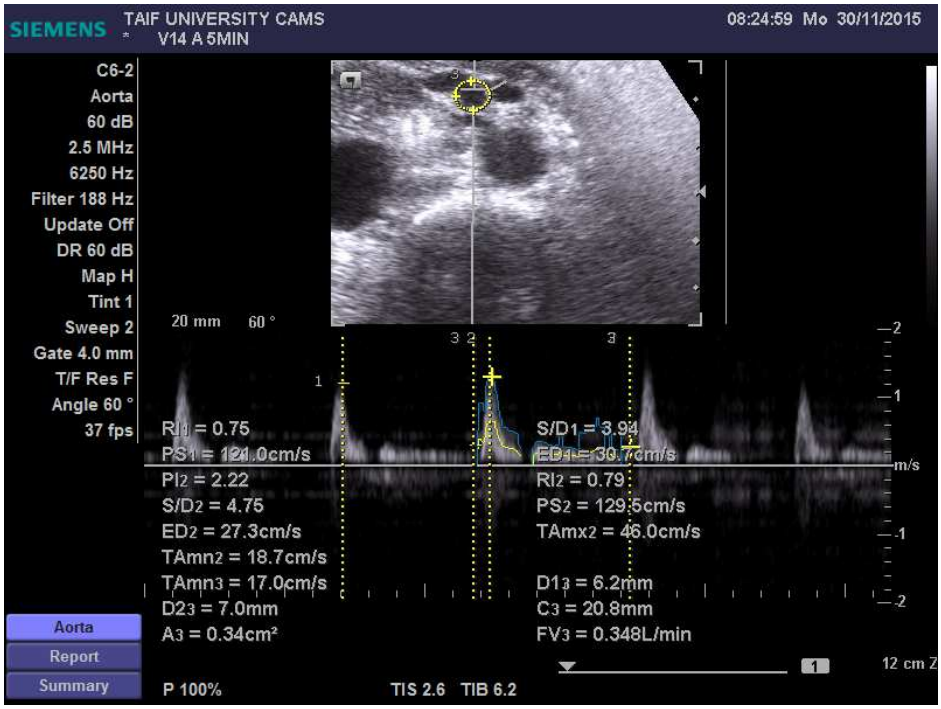

Image No (17) *Duplex sonogram of the SMA showing SMA diameter (7.0mm) for carbohydrate meal after 5 min with other parameter*

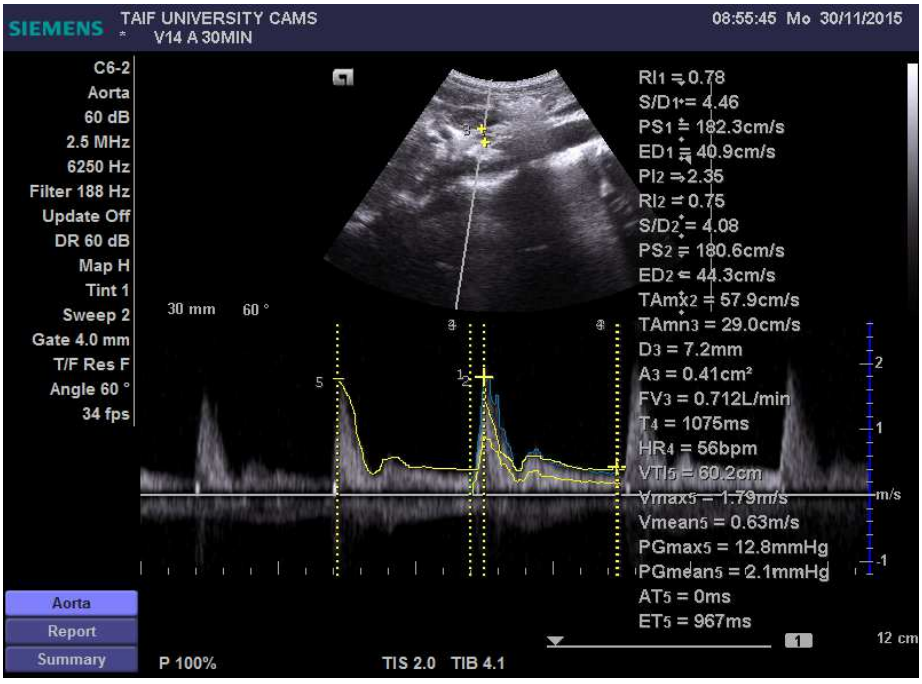

Image No (18) *Duplex sonogram of the SMA showing SMA blood flow volume (0.712 L/min) for carbohydrate meal after 30 min*

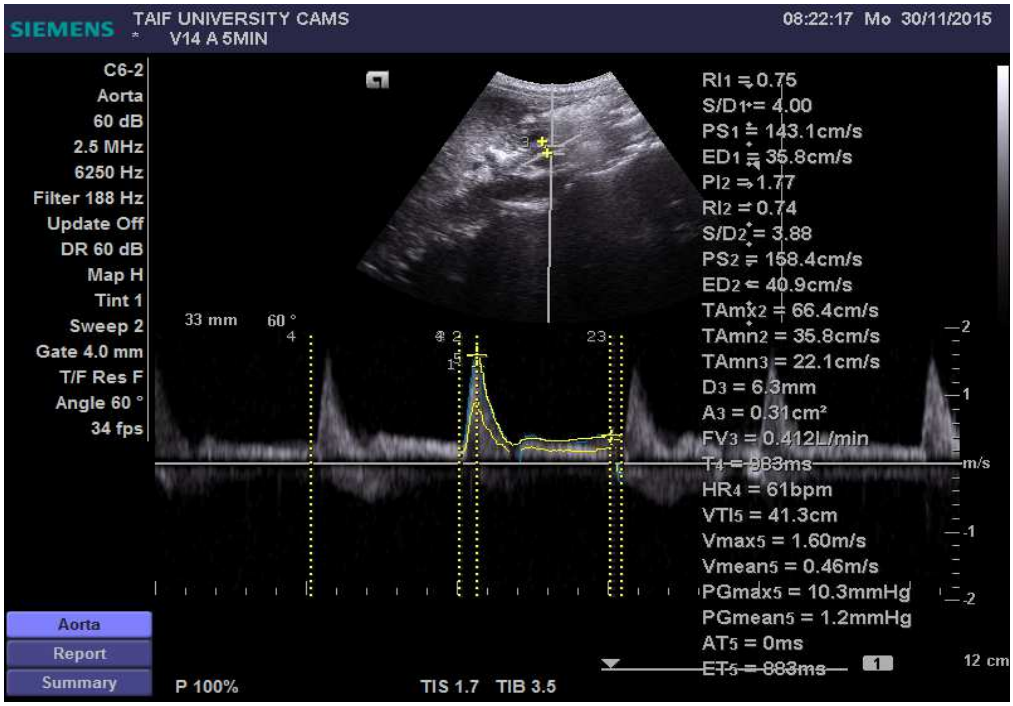

Image No (19) *Duplex sonogram of the SMA showing peak systolic velocity (143.1cm/s) for fatty meal after 5 min with other parameter*

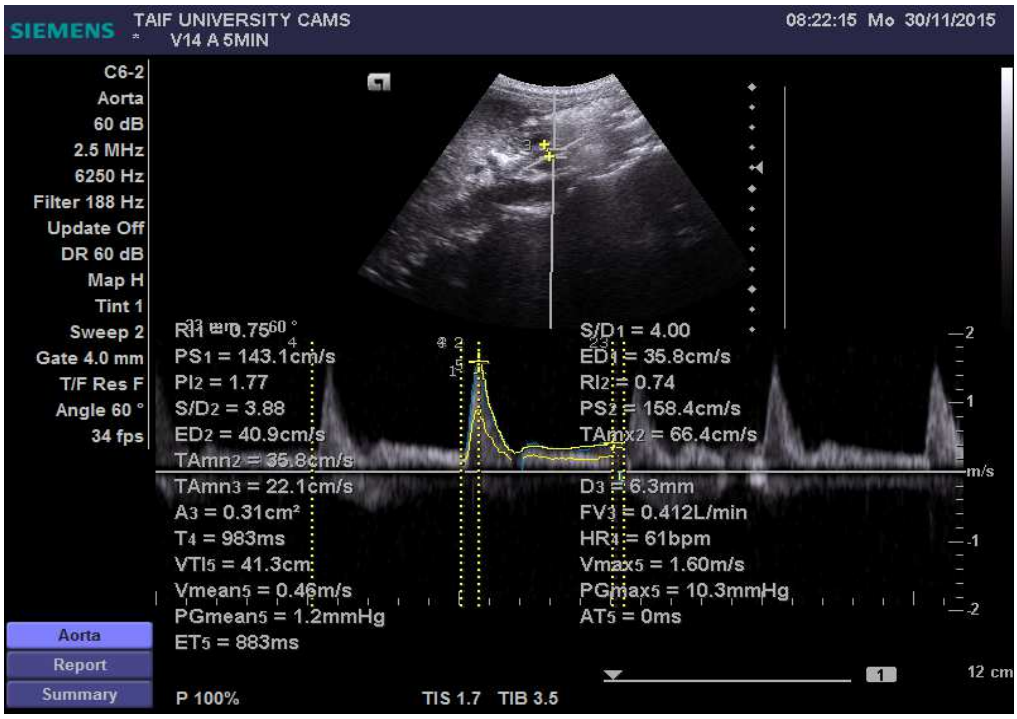

Image No (20) *Duplex sonogram of the SMA showing end diastolic velocity (35.8cm/s) for fatty meal after 5 min with other parameter*

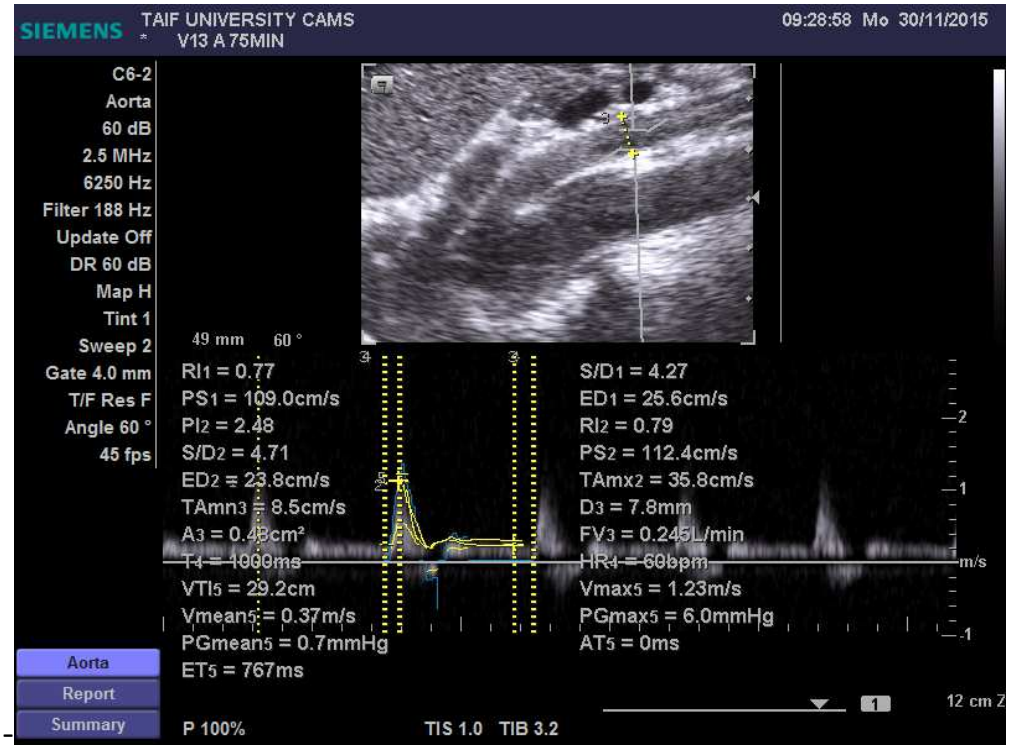

Image No (21) *Duplex sonogram of the SMA showing SMA diameter (7.8mm) for fatty meal after 75 min with other parameter*

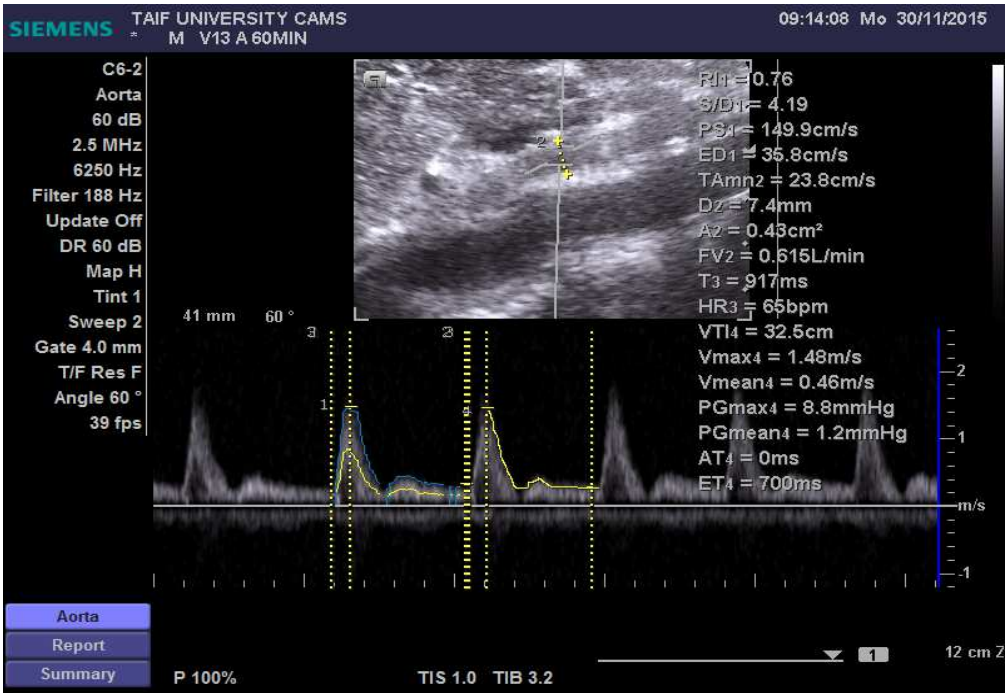

Image No (22) *Duplex sonogram of the SMA showing peak systolic velocity (149.9 cm/s) for fatty meal after 60 min with other parameter*

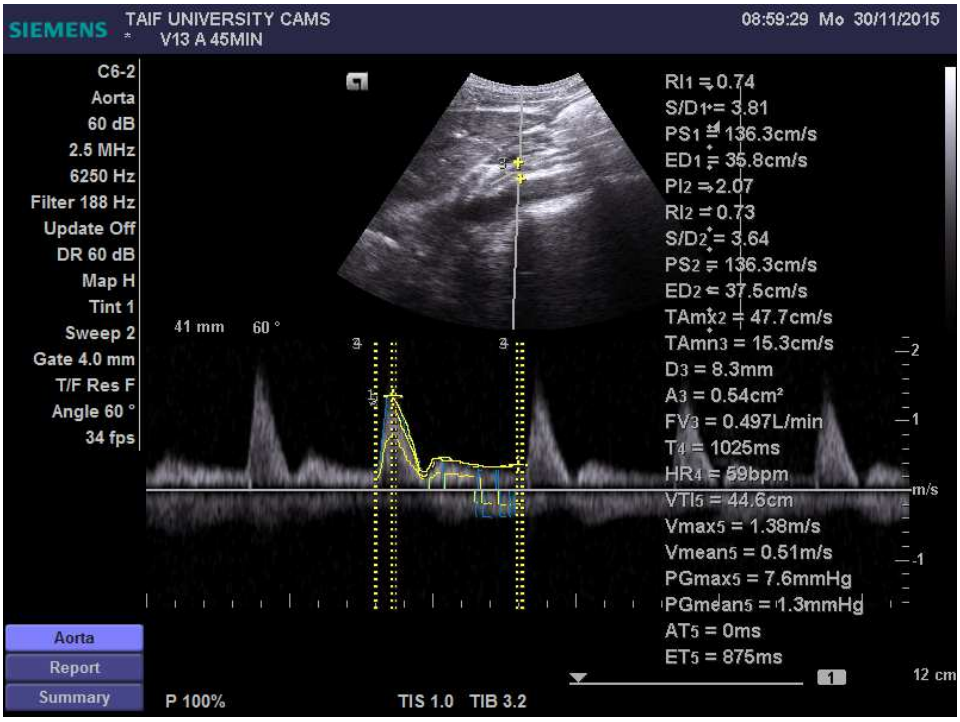

Image No (23) *Duplex sonogram of the SMA showing mean blood velocity (0.51 m/s) for fatty meal after 45 min with other parameter*

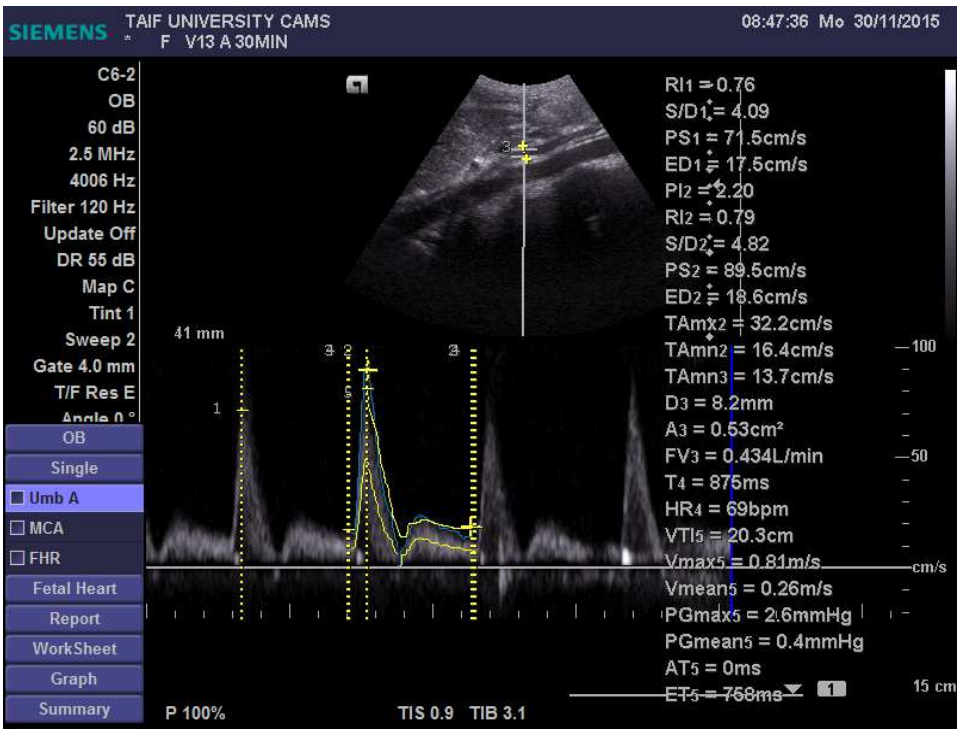

Image No (24) *Duplex sonogram of the SMA showing resistive index (0.76) for fatty meal after 30 min with other parameter*

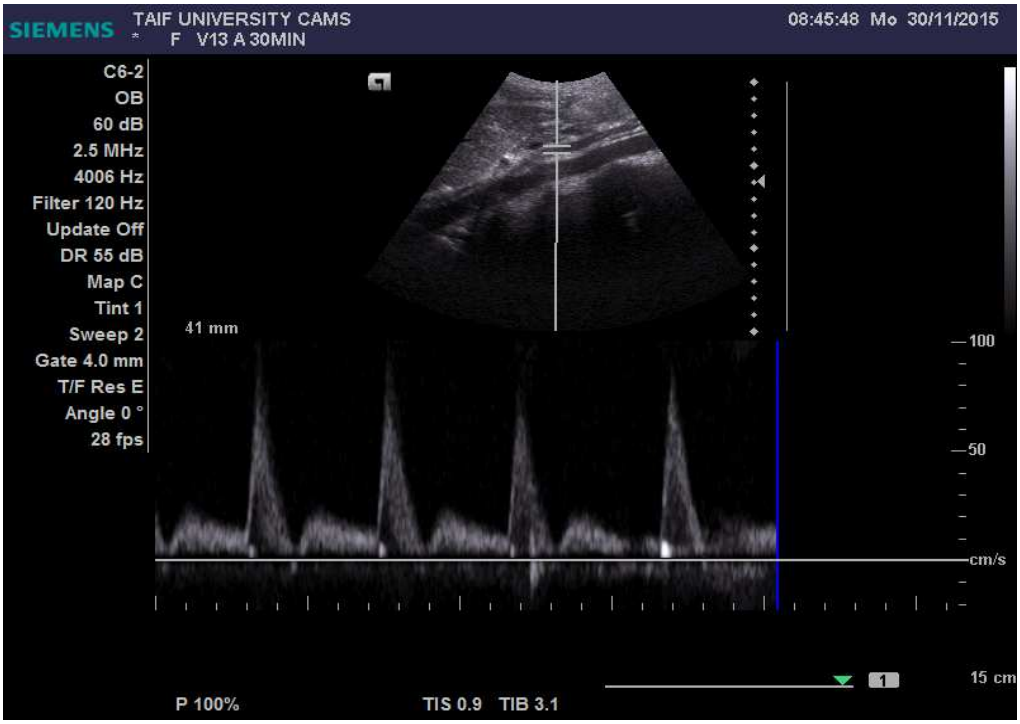

Image No (25) *Spectral Doppler of the SMA showing increasing of blood flow volume for fatty meal after 30 min*

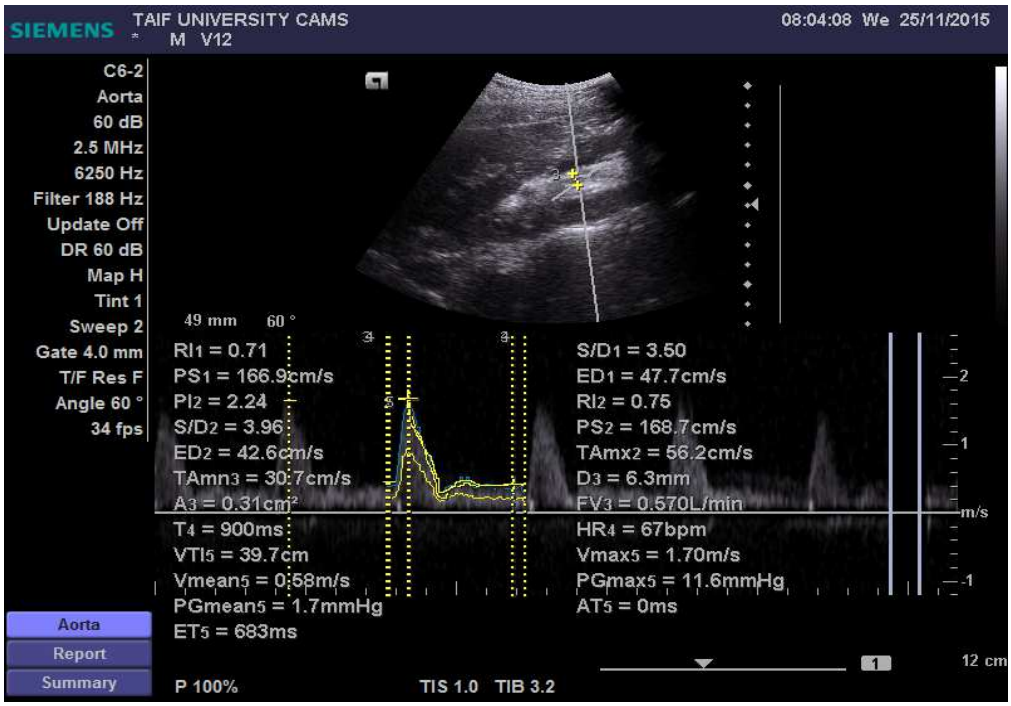

Image No (26) *Duplex sonogram of the SMA showing peak systolic velocity (166.9 cm/s) for carbohydrate meal after 15 min*

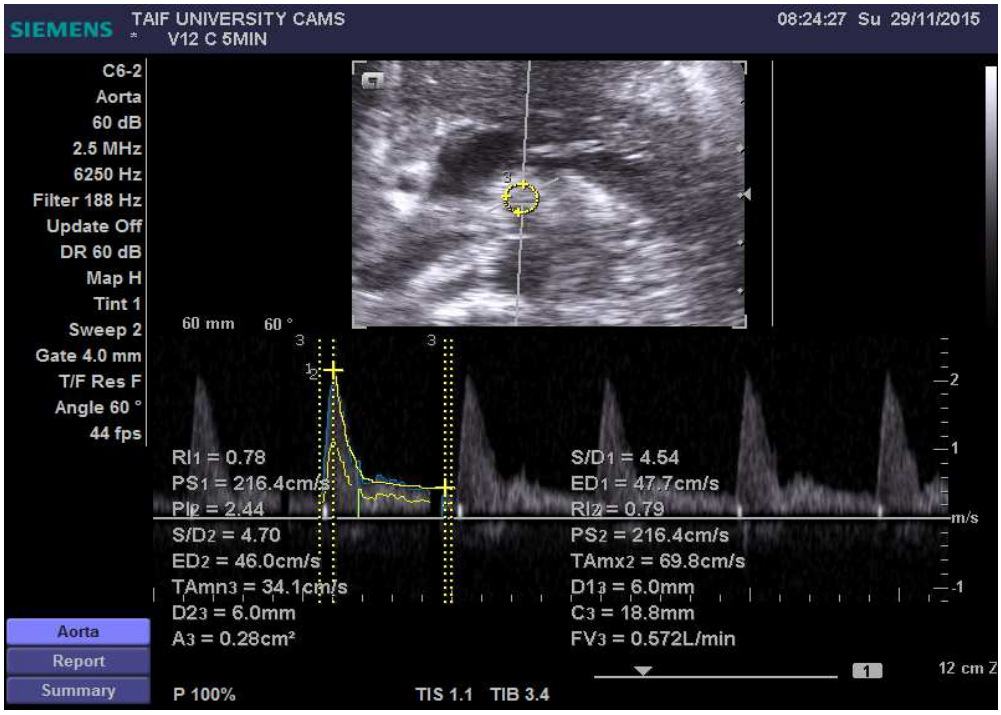

Image No (27) *Duplex sonogram of the SMA showing blood flow volume(0.572 L/min) for carbohydrate meal after 5 min*

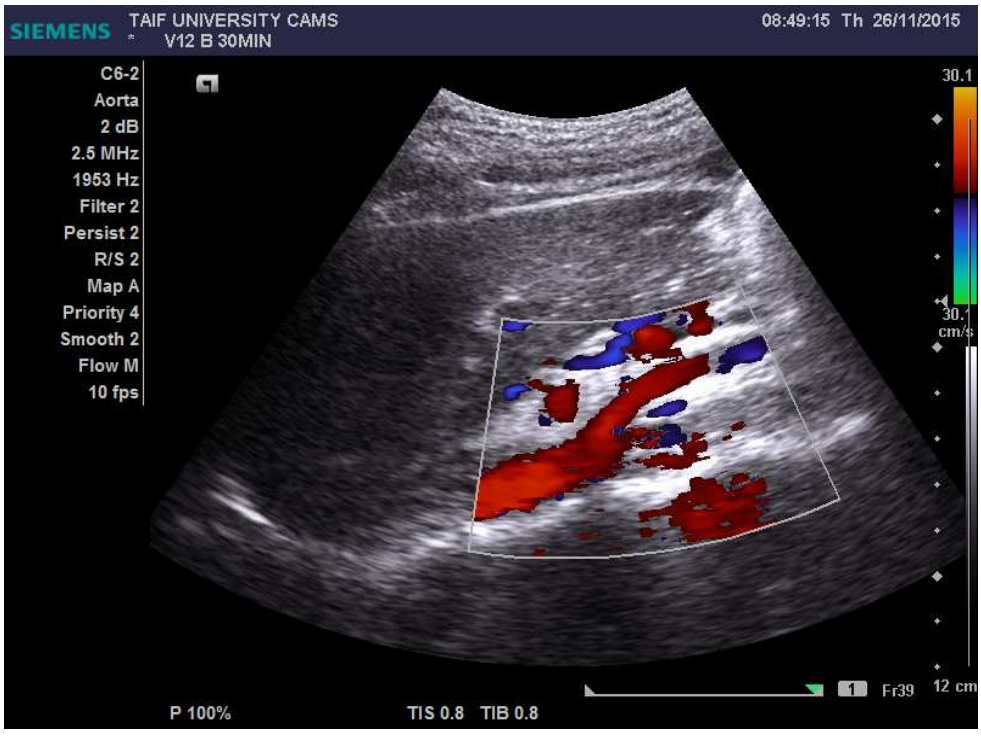

Image No (28) *Color Doppler of the SMA showing SMA blood flow for fatty meal after 30 min*

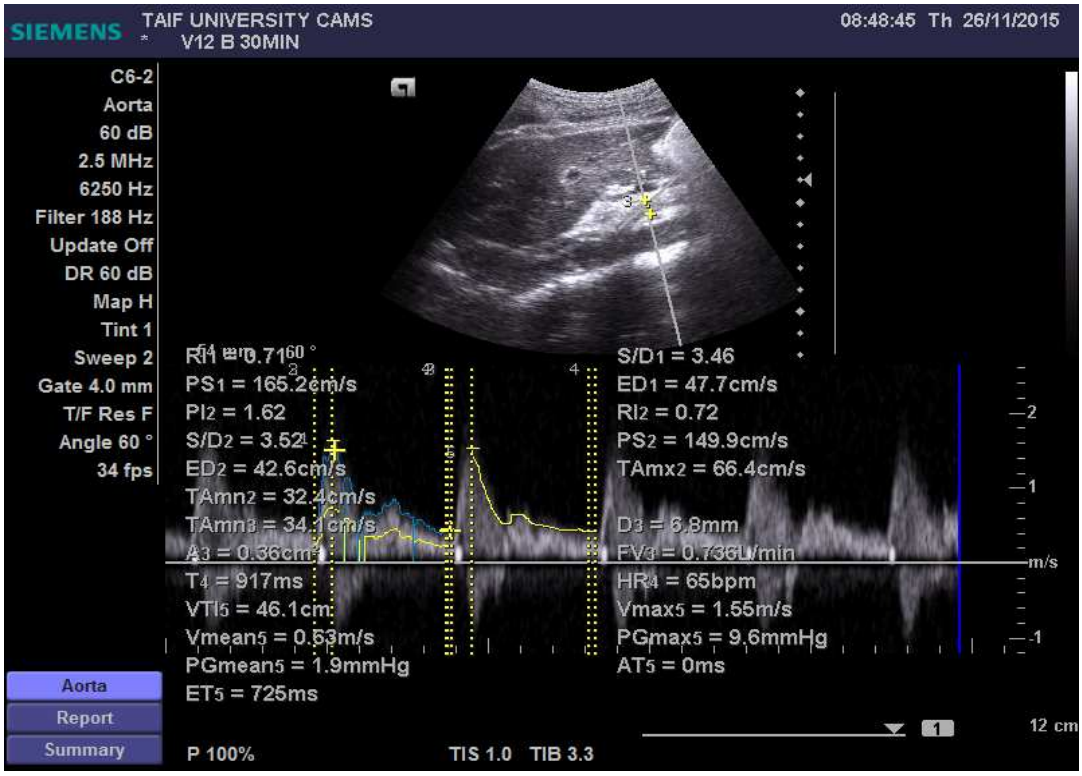

Image No (29) *Duplex sonogram of the SMA showing peak systolic velocity (166.2 cm/s) for carbohydrate meal after 30 min.*

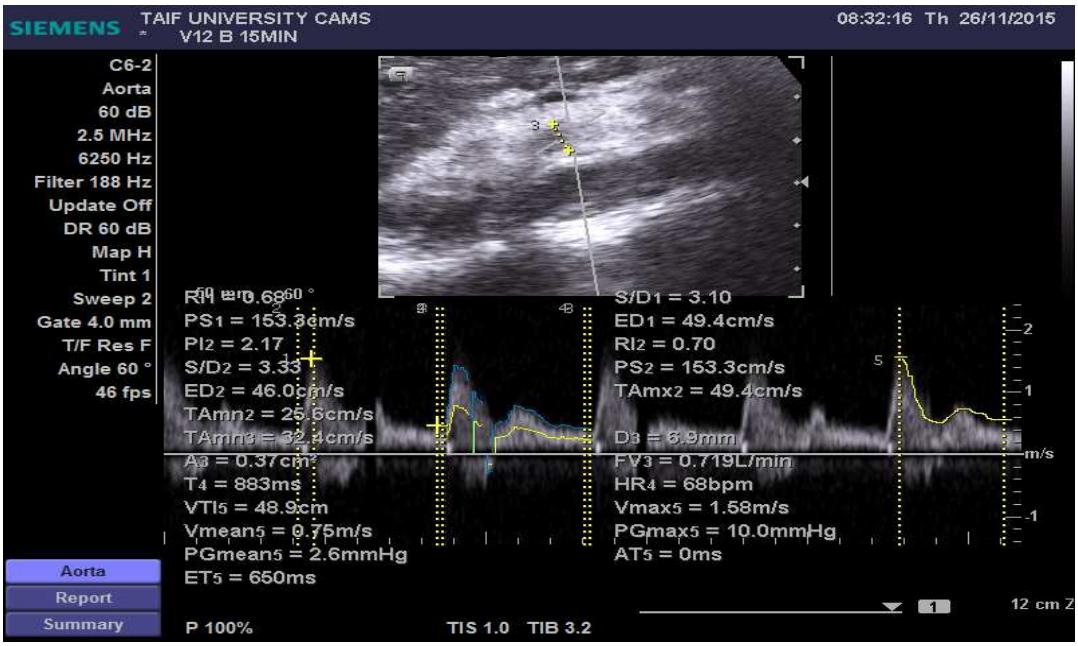

Image No (30) *Duplex sonogram of the SMA showing peak systolic velocity (153.3 cm/s) for carbohydrate meal after 30 min.*

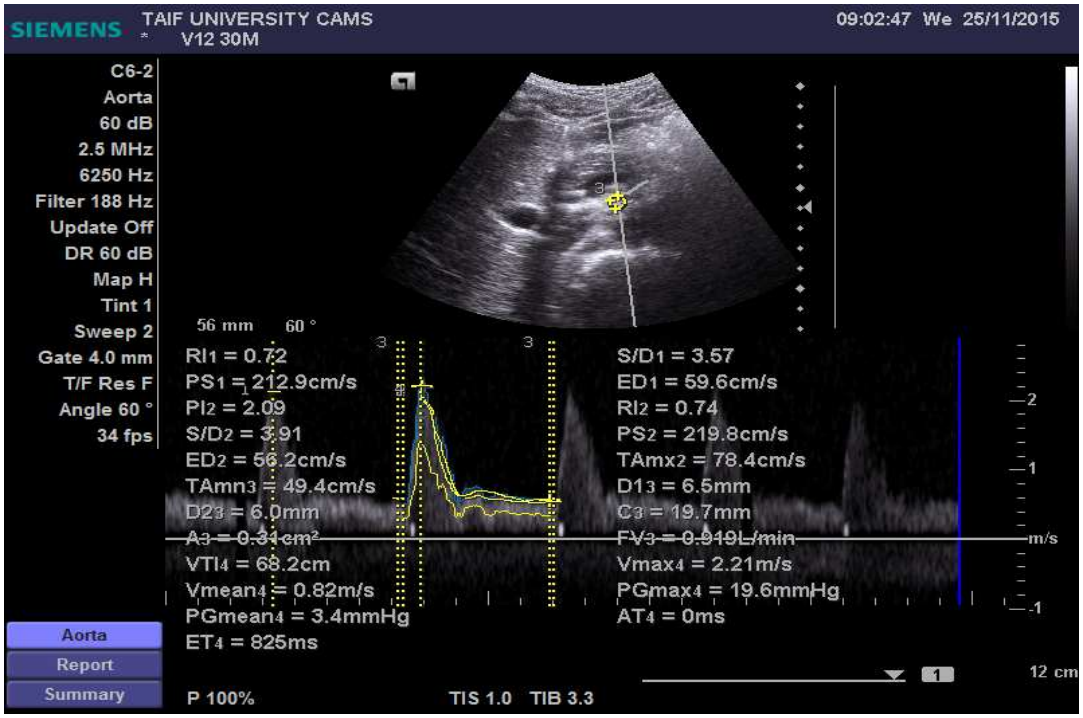

Image No (31) *Duplex sonogram of the SMA showing peak systolic velocity (212.9 cm/s) for carbohydrate meal after 30 min.*

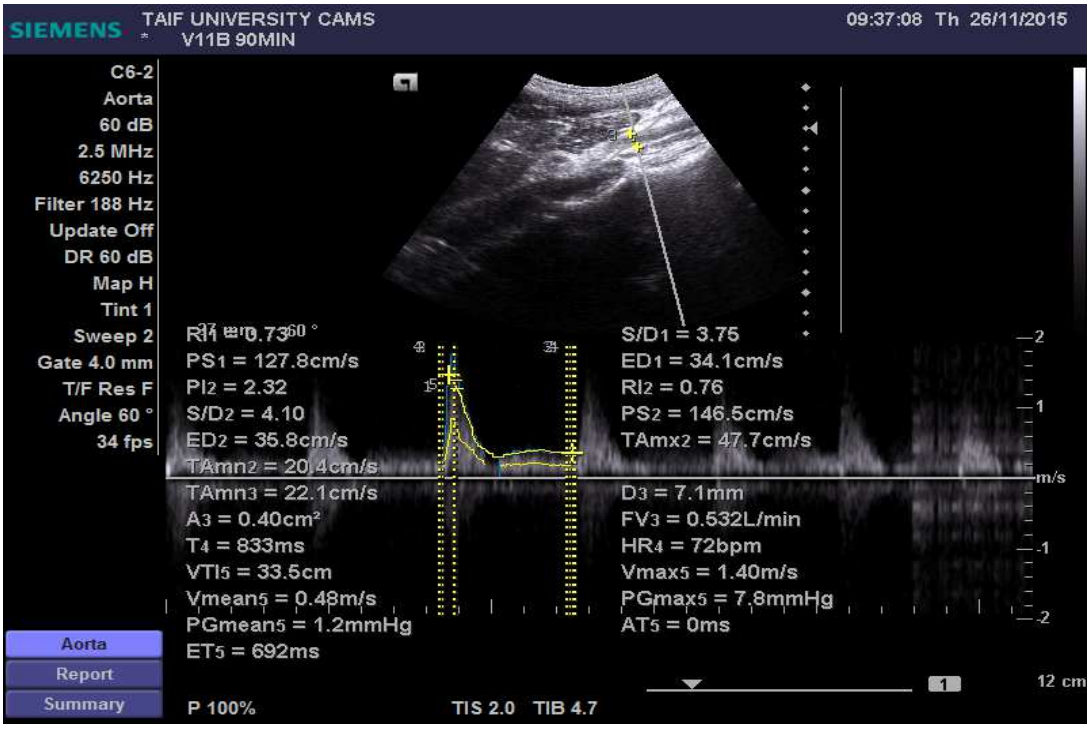

Image No (32) *Duplex sonogram of the SMA showing pulsitilty index(2.32) for carbohydrate meal after 90 min.*

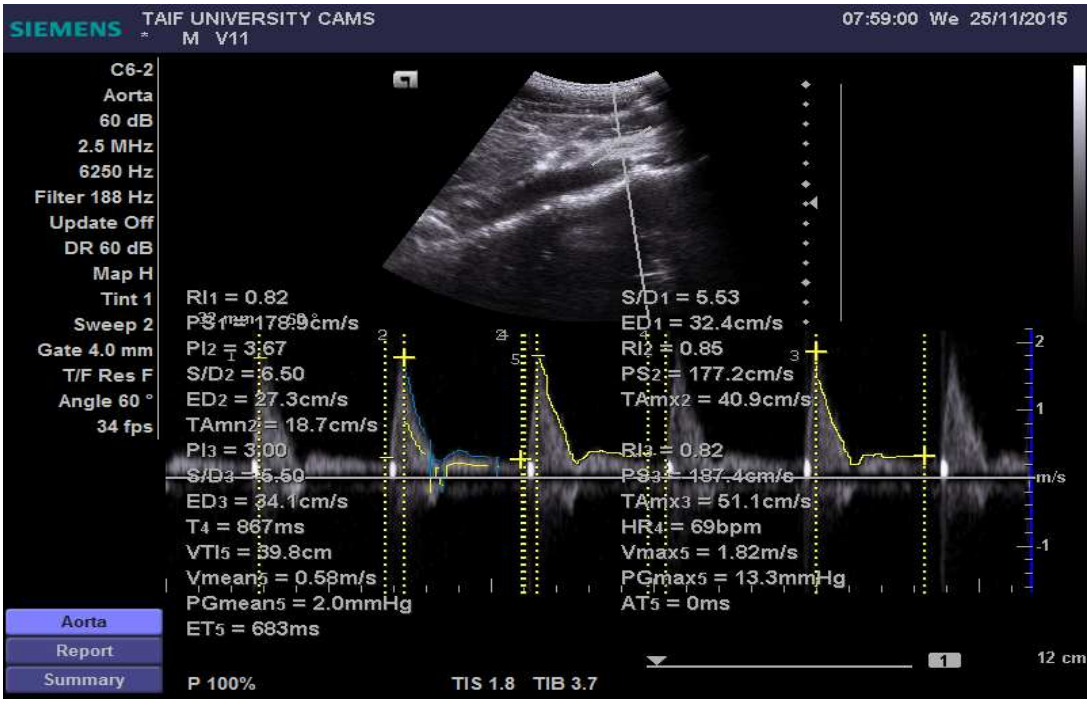

Image No (33) *Duplex sonogram of the SMA showing end diastolic velocity (32.4 cm/s) for carbohydrate meal after 5 min.*

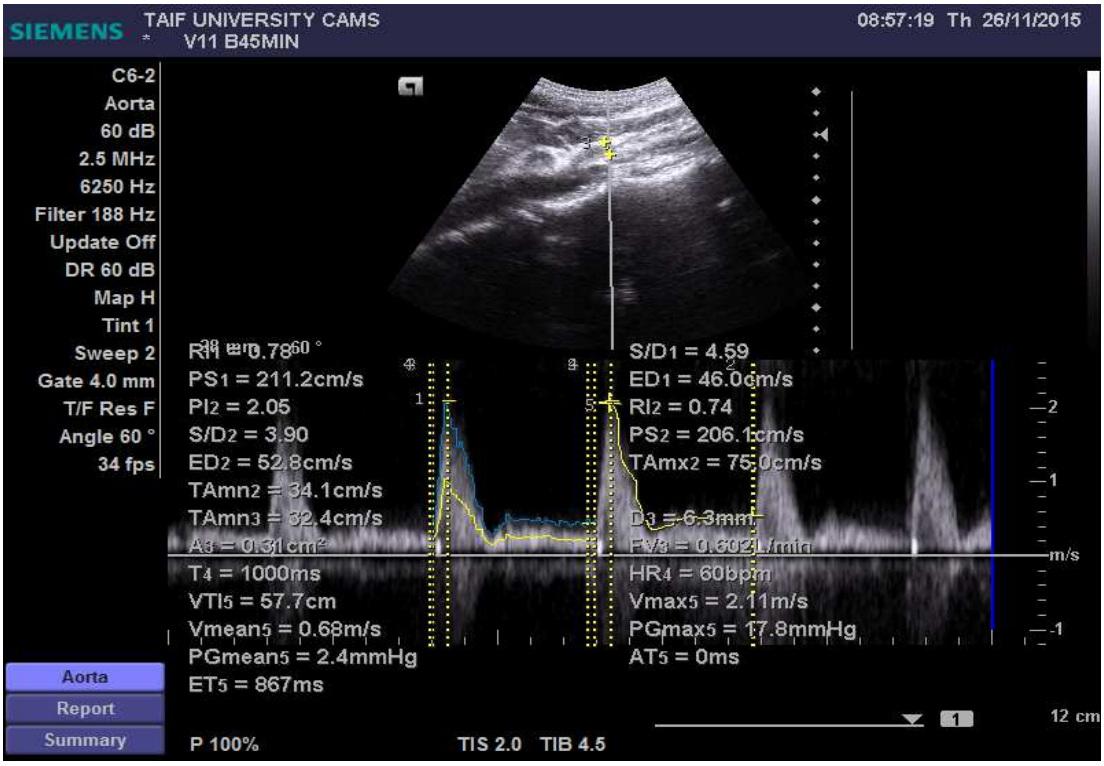

Image No (34) *Duplex sonogram of the SMA showing SMA diameter (7.8mm) for fatty meal after 45 min*

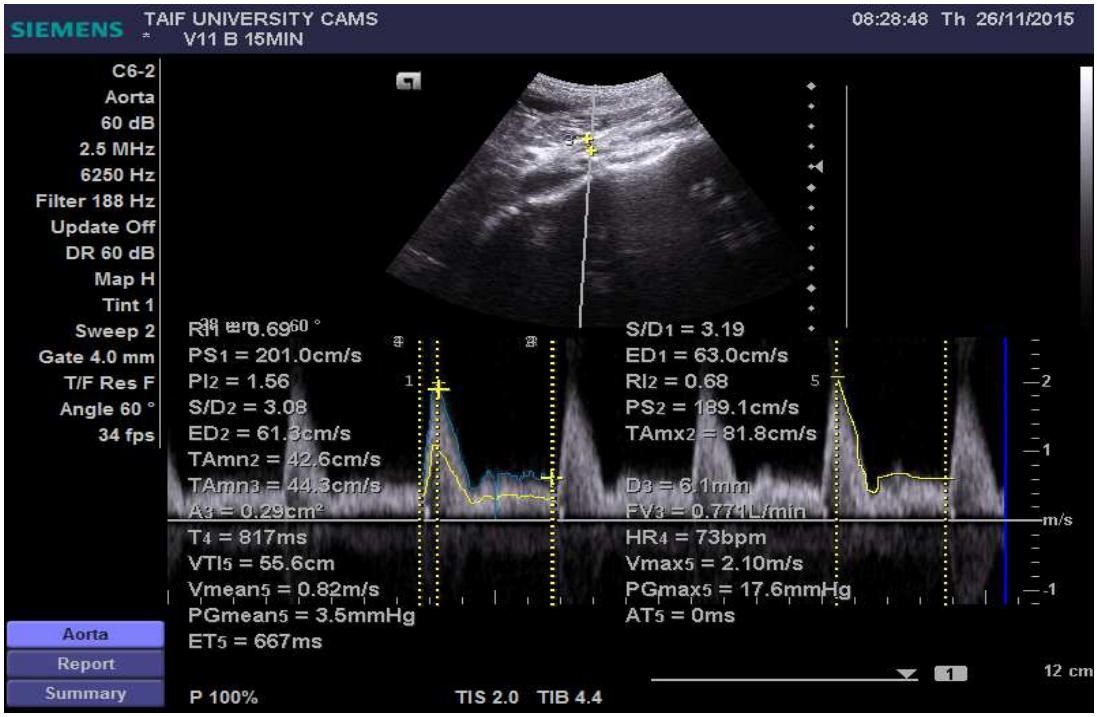

Image No (35) *Duplex sonogram of the SMA showing blood flow volume(0.774L/min) for carbohydrate meal after 30 min*

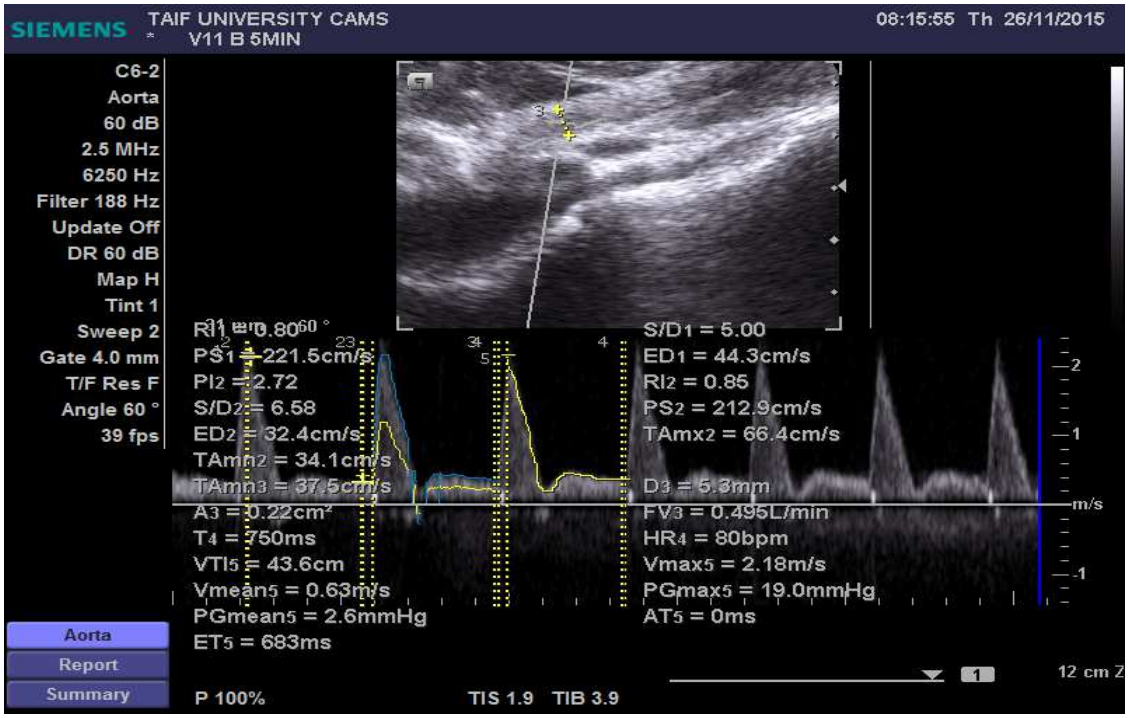

Image No (36) *Duplex sonogram of the SMA showing mean velocity (0.63m/s) for carbohydrate meal after 15min*

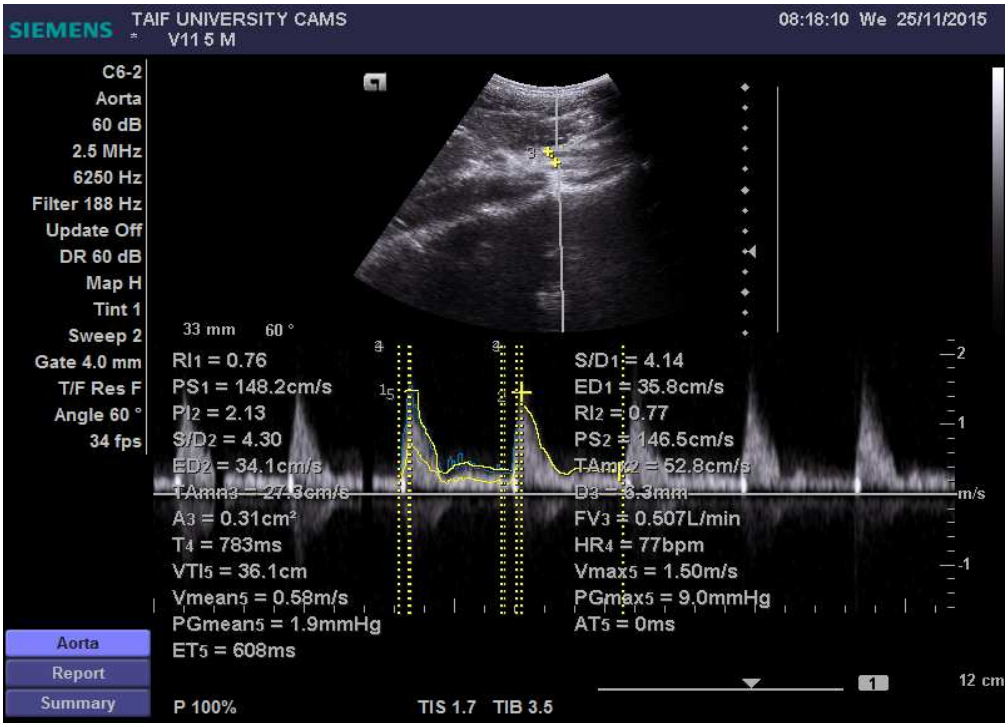

Image No (37) *Duplex sonogram of the SMA showing peak systolic velocity (148.2 cm/s) for carbohydrate meal after 5 min.*

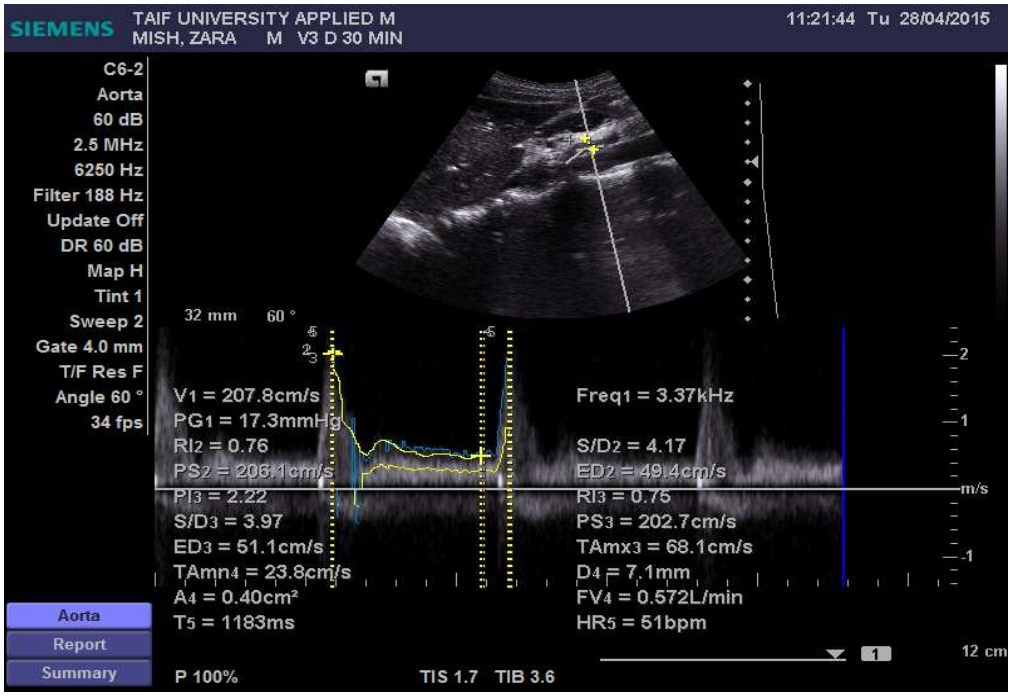

Image No (38) *Duplex sonogram of the SMA showing SMA diameter (7.1 mm) for fatty meal after 30 min .*

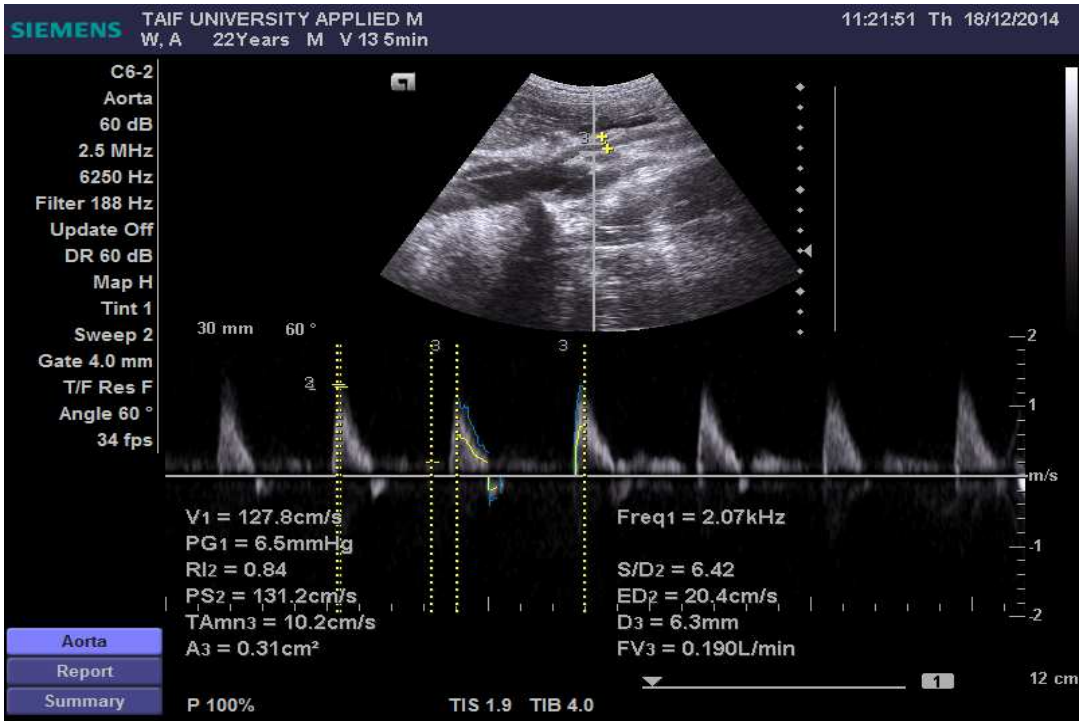

Image No (39) *Duplex sonogram of the SMA showing peak systolic velocity (131.2 cm/s) for carbohydrate meal after 90 min*

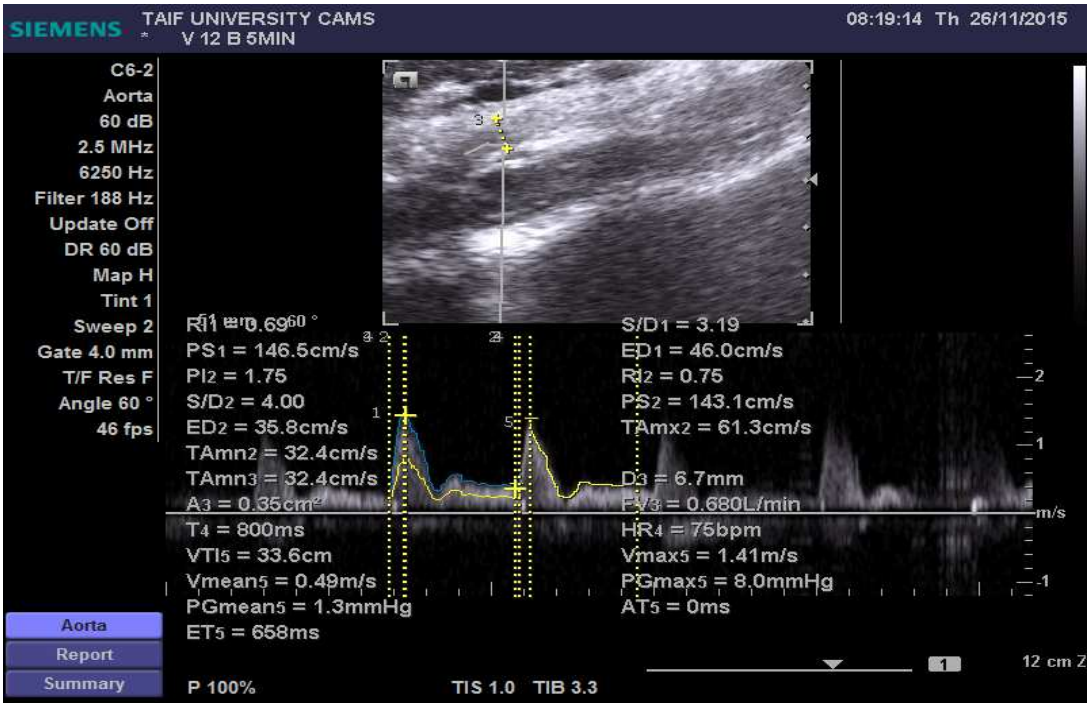

Image No (40) *Duplex sonogram of the SMA showing SMA diameter (6.7 mm) for carbohydrate meal after 15 min.*

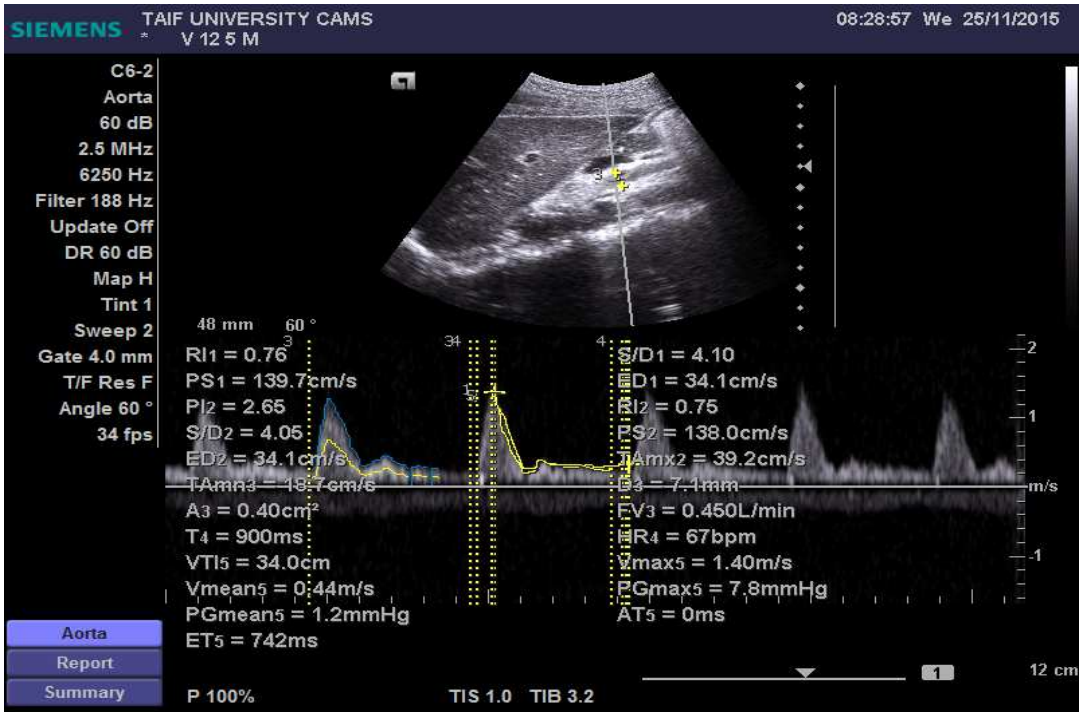

Image No (41) *Duplex sonogram of the SMA showing resistive index (0.76) for fatty meal after 30 min*

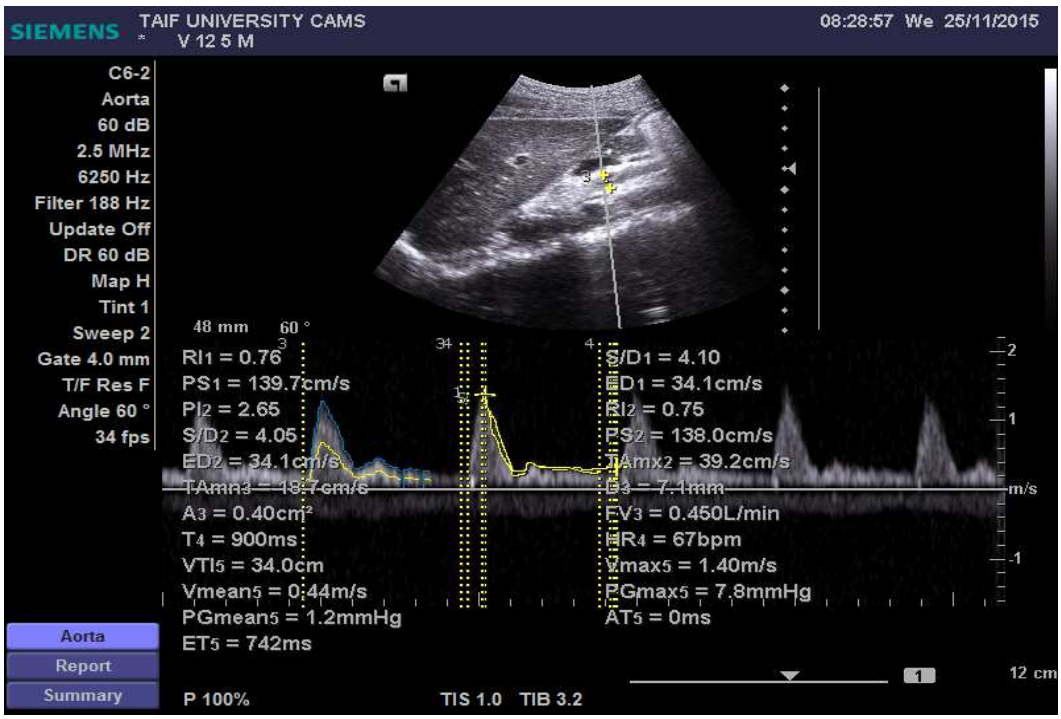

Image No (42) *Duplex sonogram of the SMA showing end diastolic velocity (43.1 cm/s) for fatty meal after 30 min*

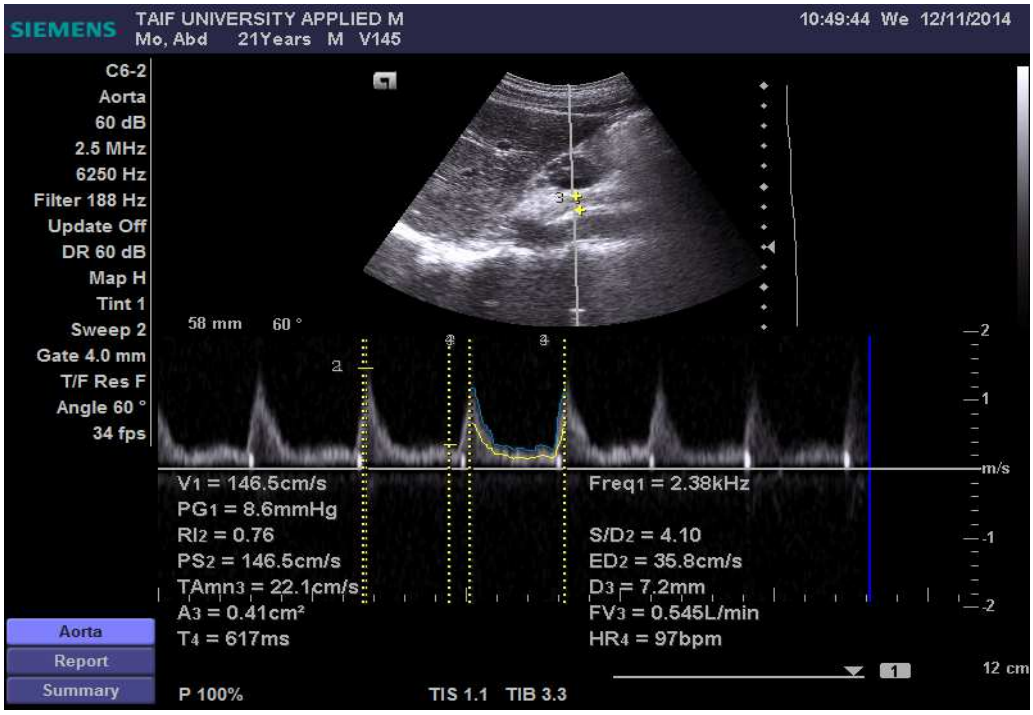

Image No (43) *Duplex sonogram of the SMA showing peak systolic velocity (146.5 cm/s) for fatty meal after 45 min*

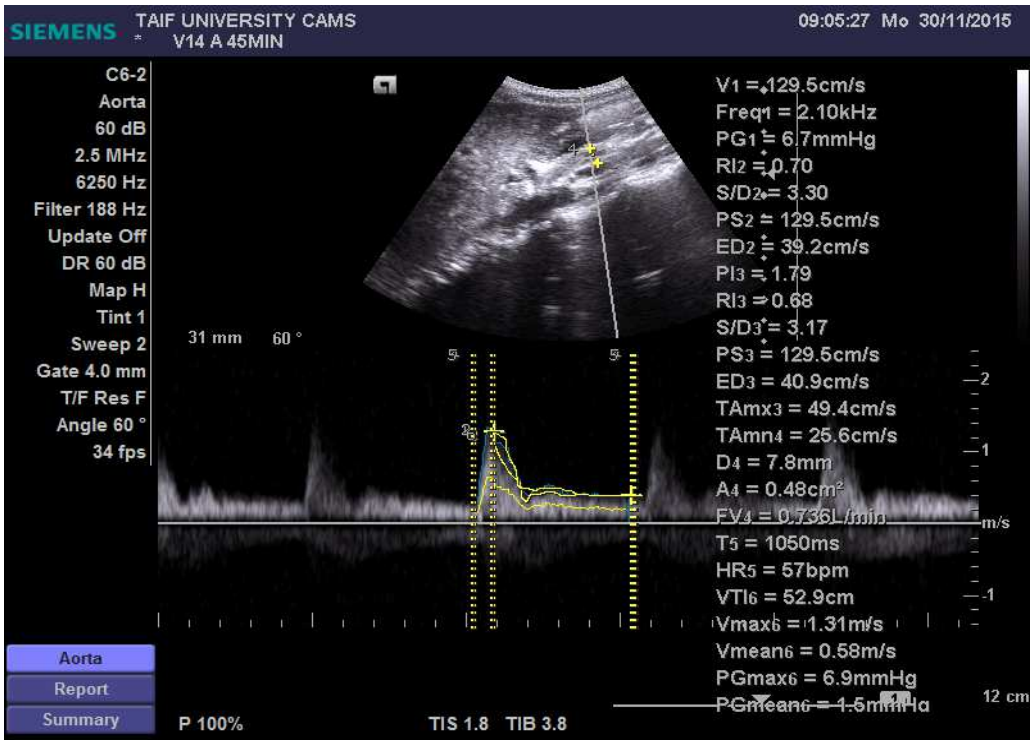

Image No (44) *Duplex sonogram of the SMA showing SMA diameter (7.8 mm) for fatty meal after 90 min*

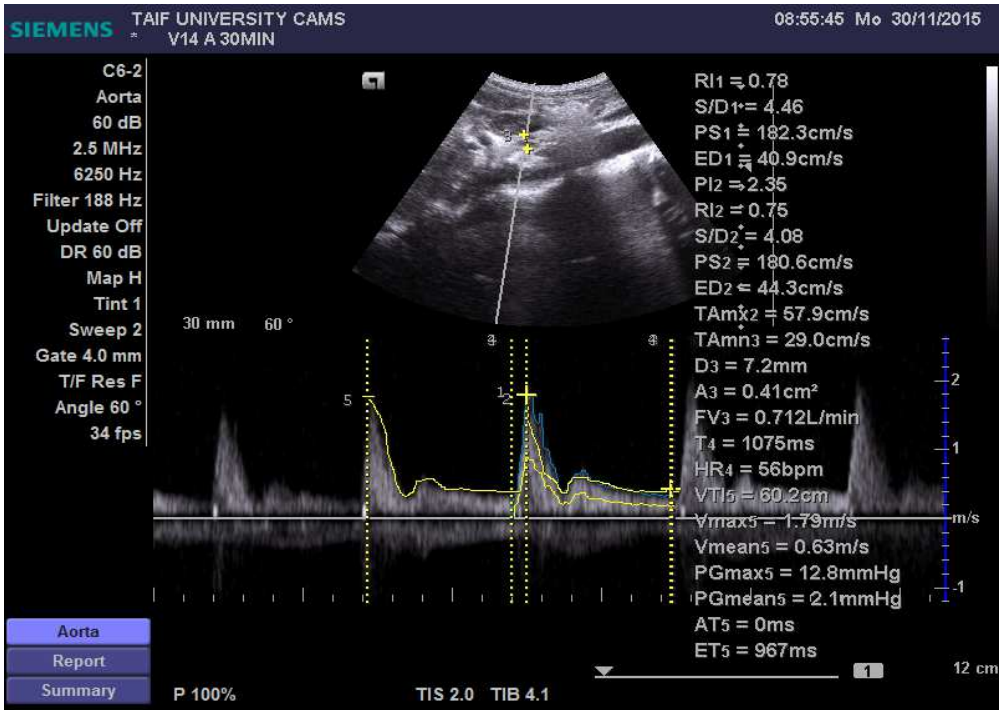

Image No (45) *Duplex sonogram of the SMA showing Blood flow Volume (.712 L/min) for fatty meal after 30 min*

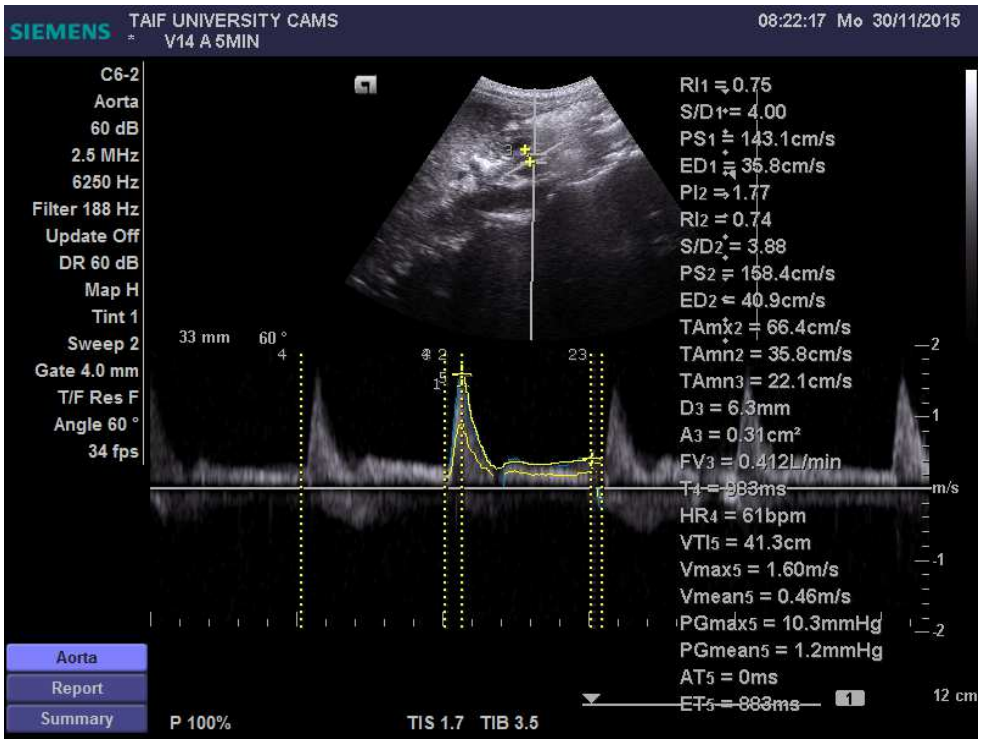

Image No (46) *Duplex sonogram of the SMA showing SMA diameter (6.3 mm) for fatty meal after 5 min.*