



## Biochemical Analysis on the Lipids of the Heart of the Camel and Cardiac Marker Enzymes

Marwa- Babiker, A. M. <sup>1</sup>, H. I. Ismail<sup>1</sup> and A. A. M. Taha<sup>2</sup>

1. Department of Anatomy, College of Veterinary Medicine, University of Bahri.

2. Department of Anatomy, Faculty of Veterinary Medicine, University of Khartoum  
[alitaha3@hotmail.com](mailto:alitaha3@hotmail.com)

Corresponding Author: [marwa.eltlib@gmail.com](mailto:marwa.eltlib@gmail.com)

Article history: Received: July 2015

Accepted: October 2015

### Abstract

The aim of this study was to investigate the lipids and cardiac marker enzymes on the heart of the camel with myocardial bridges. Blood samples were taken from seven adult camels showing myocardial bridges. The samples were collected from Al-Buga'a slaughterhouse, Sudan. The samples were analyzed biochemically for two cardiac enzymes (cardiac markers); Creatine kinase (CK) and Cardiac Troponin (T). Values for (CK) ranged from 25-48 U/L, whereas those for (T) enzymes were almost negative. Simultaneously fresh hearts of other 7 camels were used to study the total lipids, triglycerides and phospholipids, using biochemical techniques. The obtained values for total lipids, triglycerides and phospholipids were ranging from (18.62-22.55g) 84,0% (2.23-3.43g) 0.11% and (0.37-0.60g) 19.0% respectively. The obtained results for the enzyme markers suggest that myocardial bridges in camel heart may be normal feature although they are considered as anomalies elsewhere.

**Keywords:** camel, lipids, myocardial bridges, cardiac markers

© 2015 Sudan University of Science and Technology, All rights reserved

### Introduction

Myocardial bridges were defined as a congenital coronary abnormality in which a branch of a coronary artery runs intramurally through the myocardium (Kosinski and Grzybiak, 2001; Chen, *et al.* 2004; Singh, *et al.* 2005; Alegria, *et al.* 2005; Demirsoy, *et al.* 2006). Cardiac markers are substances released from the heart muscle when it is damaged as a result of myocardial infarction. Depending on the marker, it can take between 2-24 hours for its level to increase in the blood (Ross, *et al.* 2004). There are a number of cardiac markers which could be useful as diagnostic tools for the damaged myocardium. These markers include:

creatine kinase, troponin, lactic dehydrogenase, aspartate transaminase, myoglobin. Ross *et al.* (2004) stated that the most sensitive and specific test for myocardial damage is creatine kinase (CK). Its relative specificity is limited by the damage of the skeletal muscle. Its peak is reached in 10-24 hours and it has a short duration.

Nowadays it is believed that troponin is most sensitive and specific than creatine kinase for assessing myocardial damage. Its peak is reached in 12 hours only. It is released in 2-4 hours and persists for 7 days (Nissen, *et al.* 1965). On the other hand, (Mockel, *et al.* 1999) claimed that cardiac

troponin is neither diagnostic for acute coronary syndrome nor predictive of the outcome.

Cardiac markers like aspartate transeaminase, lactate dehydrogenase, and myoglobin have low specificity for myocardial infarction and are used less frequently than the previously mentioned cardiac markers (Ross, *et al.*, 2004).

Mirgani (1991) studied the total lipids, phospholipids, triglycerides and total cholesterol in the heart, kidney, liver, serum, hump and perinephric fat in the normal one humped camel. The total lipids extracted from the heart tissue of a fed camel were  $24.2 \pm 5.7$  mg/g wet tissue weight. The phospholipids are the main constituents of the heart lipids, constituting 70% of the total lipids. The triglycerides constituted 21% and the total cholesterol 9%. Cholesterol ester comprises 67% of the total cholesterol. Fatty acids composition of heart triglycerides: palmitate 44.4% is the main fatty acid present in heart triglyceride. Oleate constitutes 26%, stearate 21.8% and myristate 5.8% of the total fatty acids present in the heart triglycerides.

Mirgani (1981) stated that fasting caused a marked decrease in heart muscle total lipids, the mean value for the fed camel heart total lipids is 24.7 mg/g wet tissue. Fasting for 11 days reduced the total lipids to 11 mg/g. Whereas fasting led to a decrease in palmitate from 44.4% to 16%, it led to an increase in c18 fatty acids; stearate, has increased from 21.8% to 45.3% and oleate from 26% to 38.9.

## Materials and Methods

### Cardiac markers

A total of 7 blood samples were collected in the vacotainer tubes of 5ml each, from adult camel (*Camelus dromedarius*). The hearts of all these camels had myocardial bridges.

### Creatine kinase (CK):

The reagent formed according to (Wu and Bowers, 1982; and Young, 1995).

**Procedure:** one ml of the working reagent was mixed with 40 ml of serum in a cuvette. The mixture was agitated gently and the cuvette was then transferred to the photometer. It was incubated for 5 min and the reading of the absorption was recorded.

This procedure was repeated for another sample and the reading of the absorption was again recorded. The difference between two readings used to obtain the average change.

### Calculation:

Total (CK) activity U/L =  $\Delta A \times 8095$  at 37°C (Wu and Bowers, 1982; and Young, 1995).

### Cardiac Troponin (T):

Two drops of serum were transfer to the specimen well of the test device of buffer then add one drop then start the timer and then wait for the colored lines to appear, read results at 10 min.

- Positive: Two distinct colored lines will appear.
- Negative: one colored line appears in the control line region (Mehegan and Tobacman, 1994).

### Biochemical Analysis:

#### Determination of fat in heart tissue:

A total of 7 fresh normal hearts obtained from adult camels (*Camelus dromedarius*) were used to detect total lipids, triglycerides and phospholipids. The weight of the hearts was determined using normal balance. Each heart was homogenized and two samples were taken from each heart.

The total lipids, triglycerides and phospholipids were determined according to the methods of Fringes and Dunn, (1970), Biggs, *et al.* (1975) and Varely (1967) respectively.

### Results

#### Cardiac markers

#### Creatine Kinase (CK) and Cardiac Troponin (T)

The values obtained from the blood samples collected from the seven adult camels which exhibited myocardial bridges in their hearts, are summarized in table 1. The values ranged between 25-48 U/L.

Serum cardiac troponins are the earliest appearing biochemical markers during the

myocardial damage. The cardiac troponin was detected in only one blood sample of the seven camels with myocardial bridges. All other six blood samples from the remaining six camels with myocardial bridges in their hearts were negative.

Table 1: showing the analysis of blood serum in camels with myocardial bridges; Creatine Kinase (CK) and Cardiac Troponin (T).

Enzymes Camels	Creatine kinase U/L	Cardiac Troponin (T)
1	28	+
2	25	-
3	42	-
4	32	-
5	27	-
6	39	-
7	48	-

**Biochemical Analysis:**

**Determination of Fat in heart tissue (tables 2 and 3)**

The weight of the seven hearts used in this study ranged between 2050-3000 g with a mean value of 2485.71±331.87. The weight of total lipids ranged between 18.62-22.55 g, the

percentage of the mean value was 0.84%; the triglycerides ranged between 2.23-3.43 g, the percentage of the mean value was 0.11%, and finally that of the phospholipids ranged between 0.37-0.60 g with percentage of 0.019%. The mean and standard deviation were calculated (Fig. 1).

Table 2: showing the weight of the hearts, determination of the total lipid, triglyceride and phospholipids.

Heart No.	Heart Wt. in (g)	Total Lipids (g/kg)	Triglycerides (g/kg)	Phospholipids (g/kg)
1	2050	18.62	2.63	0.49
2	2500	20.16	2.86	0.55
3	2600	22.03	2.42	0.44
4	2650	19.64	2.23	0.60
5	2200	22.04	3.43	0.50
6	2400	21.01	3.18	0.39
7	3000	22.55	2.92	0.37
<b>Mean±SD</b>	<b>2485.71±331.87</b>	<b>20.87±1.45</b>	<b>2.81±1.42</b>	<b>0.48±0.08</b>

Table 3: showing the weight of the heart and the percentage of the total lipids, the triglycerides and the phospholipids in seven camels

No. of Hearts	Weight kg	Total Lipids %	Triglycerides %	Phospholipids %
1	2.05	0.91	0.13	0.024
2	2.50	0.81	0.11	0.022
3	2.60	0.85	0.09	0.017
4	2.65	0.74	0.08	0.023
5	2.20	1.00	0.16	0.022
6	2.40	0.88	0.13	0.016
7	3.00	0.75	0.10	0.012
<b>Mean±SD</b>	<b>2.49±0.332</b>	<b>0.84±0.085</b>	<b>0.11±0.026</b>	<b>0.019±0.0041</b>

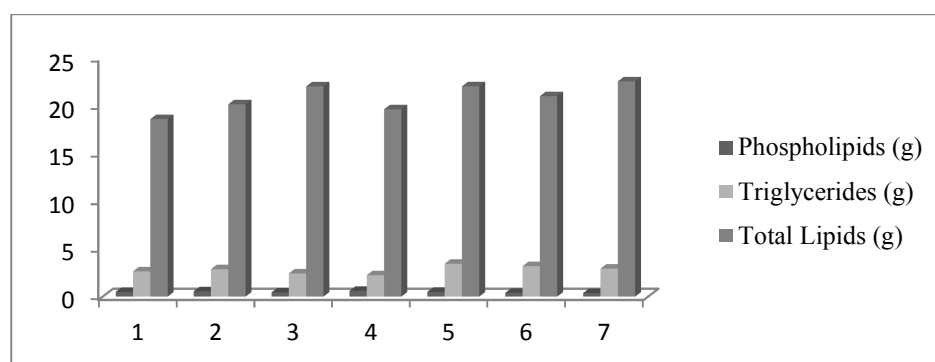


Fig. 1: The relationship between the total lipids triglycerides and phospholipids in 7 hearts of camels

### Discussion

Mirgani (1991) studied the total lipids, phospholipids, triglycerides and total cholesterol in the heart of the camel, and presented the following values: the total lipids extracted were  $24.2 \pm 5.7$  mg/g wet tissue weight. The phospholipids were the main constituents of the heart lipid constituting 70% of the total lipids. The triglycerides constituted 21% and the total cholesterol 9%. The findings contradict the result obtained in the present investigation, where total lipids value was 8.38 mg/g, triglycerides were 1.13 mg/g and phospholipids 0.19 mg/g. The description of the result was being attributed to the methods used.

Cardiac markers are substances released from the heart muscle damaged as a result of myocardial infarction. Additionally, determining the levels of cardiac markers in the laboratory takes substantial time. Eisenman (2006) stated that the cardiac markers or cardiac enzymes are proteins that leak out of injured myocardial cells through their damaged cell membranes into the bloodstream. Until the 1980, the enzymes glutamic oxaloacetic transaminase (SGOT) and lactate dehydrogenase (LDH) were used to assess cardiac injury. Now, the markers most widely used in detection of myocardial infarction are creatine kinase and both cardiac troponin T and cardiac troponin I. The cardiac troponins T and I which are

released within 4–6 hours of an attack of myocardial infarction and remain elevated for up to 2 weeks have nearly complete tissue specificity and are now the preferred markers for assessing myocardial damage.

Ross *et al.* (2004) stated that the most sensitive and specific test for myocardial damage is creatine kinase (CK). They added that other enzymes, including aspartate transaminase (AST), lactate dehydrogenase (LDH), and myoglobin (Mb), have low specificity for myocardial infarction and are less used than the other markers. Creatine kinase (CK) in 7 blood samples of the camels with myocardial bridges in this study ranged between 25-48 U/L. However, the normal value of Creatine kinase in 5 adult she-camels was  $408.6 \pm 127.6$  U/L (Osman and Al-Busadah, 2003).

Hekmatimoghaddam, *et al.* (2011) measured serum cardiac troponin I in 84 dromedary camel; Troponin I concentrations were  $0.467 \pm 0.09$  ng/mL (ranging from  $<0.2$ -3.8 ng/mL). There was no significant difference between sex and age in camel troponin. The samples which were used in this research gave 6 negative and only one positive sample of cardiac troponin T.

According to Brandt, *et al.* (2001), serum cardiac troponin I concentrations increase in several types of myocardial injury because of leakage from the damaged myocardial cells. In humans, release of cardiac troponin I from the heart into the systemic circulation in acute pericarditis representative of inflammatory myocardial cell damage. O'Brien, *et al.* (2006) studied the measurement of cardiac troponin I in various animal species, they found that it is a useful biomarker of myocardial injury. Tunca, *et al.* (2008) studied the changes of the cardiac troponin I expression in blood and tissue during the myocardial degeneration in calves with foot-and-mouth disease(FMD); the rapid cardiac troponin I assay kit revealed

that all cases were cardiac troponin I positive as indicated by the 2 colored lines appearing in the test window; controls were negative. Cardiac troponin I levels were measured by a commercially available enzyme-linked immunosorbent assay kit; mean cardiac troponin I ( $14.8 \pm 1.9$  ng/ml) concentration. Mockelet *et al.* (1999) stated that moderate elevation of cardiac troponins is common in clinically stable patients with renal disease and are neither diagnostic for an acute coronary syndrome nor predictive of the outcome. They concluded that increased troponins in asymptomatic renal patient are of questionable value for risk stratification, most probably due to unspecific elevations. It was concluded that the myocardial bridges in camel heart may be normal feature although they are considered as anomalies elsewhere.

#### Acknowledgements

The authors wish to thank the International Centre for Faith Researches- Khartoum for Financial support.

#### References

- Alegria, J. R., Herrmann, J., Holmes, R. D., Lerman, A. and Rihal, S. C. (2005). Myocardial bridging. *European Heart Journal*. **26**: 1159-1168.
- Biggs, H.G., Erkinson. J.M. and Moorehead, M.R. (1975). A manual colorimetric assay of triglycerides in serum. *Clinical Chemistry*. **21**: 437-441.
- Brandt, R.R., Filzmaier, K., Hanrath, P. (2001). Circulating cardiac troponin I in acute pericarditis. *American Journal of Cardiology*. **87**: 1326-1328.
- Chen, M. L., Chen, C. H., Chao, I. M. and Lo, H. S. (2004). Dipyridamole Thallium-201 Myocardial Single Photon Emission Computed Tomography in Myocardial Bridging a Case Report. *Acta cardiologica sinica*. **20**: 37-41.
- Demirsoy, E., Arbath, H., Ünal, M., Yağan, N., Yilmaz, O., Tükenmez, F., Şener,

- D. and Sönmez, B.(2006). Coronary rupture to the right ventricle during PTCA for myocardial bridge. *Anadolu Kardiyol Derg.* **6**: 377-379.
- Eisenman, A., (2006). Troponin assays for the diagnosis of myocardial infarction and acute coronary syndrome. *Expert Review of Cardiovascular Therapy.* **4** (4): 509-14.
- Fringes, C.S. and Dunn,R.T. (1970). Colorimetric method for determination of serum total lipids based on the sulfophosphovanillin reaction. *American Journal of clinical pathology.* **53**: 89-91.
- Hekmatimoghaddam, S. H. Sazmand, A. Tajik, J. Shirazi, M.R. (2011). Serum cardiac troponin I concentrations in the camel and buffalo. *Online Journal of Veterinary Research.* **15**(6): 514-519.
- Kosinski, A. and Grzybiak, M. (2001). Myocardial bridging in the human heart: myocardial aspects. *Folia Morphologica.* **60** (1): 65-68.
- Marwa- Babiker, A. M. and Taha, A. A. M. (2013). Myocardial Bridges of the Heart of the Dromedary Camel (*Camelus dromedarius*). *University of Khartoum Journal of Veterinary Medicine and Animal Production.* **4**: 1, (90-105).
- Marwa-Babiker, A. M. and Taha A. A. M. (2015). Histology of the myocardial bridges and the related arteries of the adult dromedary camel (*Camelus dromedarius*). *Sudan Journal of Science and Technology.* **16**(2): 1-8.
- Mehegan, J.P. and Tobacman, L.S. (1994). Cooperative interaction between Troponin molecules bound to the cardiac thin filament. *The Journal of Biological Chemistry.* **266**: 966.
- Mirgani, T. (1981). Effect of fasting on camel tissue lipid. *Journal Arid Environment.* **4**: 359-361.
- Mirgani, T. (1991). Lipid composition of the heart and kidney of the camel (*Camelusdromedarius*). *Journal Arid Environment.* **20** (1): Pp.119-122.
- Mockel, M., Schindler, R. Knorr, L., Muller, C., Heller, G., Stork, T. and Frei,U. (1999). Prognostic value of cardiac troponin T and I elevations in renal disease patients without acute coronary syndrome: 9- month outcome analysis. *Nephrology Dialysis Transplantation.* **14** ( Issue 6): 1489-1495.
- Nissen, N.I, Ranlov, P., Weisfogh, J. (1965). Evaluation of four different serum enzymes in the diagnosis of acute myocardial infarction. *British Heart Journal.* **27**: 520–6.
- O'Brien, P.J., Smith D.E., Knechtel T.J. (2006). Cardiac troponin I is a sensitive, specificbiomarkerofcardiac injury in laboratory animals. *Lab Anim* **40**: 153-171.
- Osman, T.E.A. and Al-Busadah, K.A. (2003). Normal concentration of twenty serum biochemical parameters of she=camels, caws, and ewes in Saudi Arabia. *Pakistan journal of biological science.* **6** (14):1253-1256.
- Ross, G.E., Bever, F.N., Uddin, Z., Devireddy, L. and Gardin, J.M. (2004). Common scenarios to clarify the interpretation of cardiac markers. *Journal of the American Osteopathic Association.* **104** (4): 165-76.
- Singh, H., Singh, C., Kumar, A., Aggarwal, N. and Banerji, A. (2005). Acute Myocardial Infarction Secondary to Myocardial Bridge Treated with Drug-Eluting Stent. *Indian Heart Journal.* **57**: 734-737.
- Tunca, R., Sozmen, M., Erdogan, H., Cital, H., Uzlu, E., Ozen, H., and Gokce,E. (2008). Determination of cardiac troponin I in the blood and heart of calves with foot-and-mouth disease.

Journal of veterinary diagnostic investigation. 20. (Issue 5), 598-605.  
Varley, H. (1967). Practical Clinical Biochemistry. 4<sup>th</sup> edition. Pp.319-320. Inter science books. Ino, New York.  
Wu, A.H.B. and Bowers, G.N.J. (1982). Creatine kinase. American

Association for Clinical Chemistry 28: 2017.  
Young, D.S. (1995). Effect of drugs on clinical laboratory test. 4<sup>th</sup> edition. AACC press.

### تحاليل كيميائية حيوية فى دهون القلب فى الجمل وإنزيمات العلامات القلبية

مروه عبدالقادر مصطفى بابكر<sup>1</sup>، حيدر إبراهيم إسماعيل<sup>1</sup> وعلى عبدالله محمد طه<sup>2</sup>

1- قسم التشريح، كلية الطب البيطرى - جامعة بحرى

2- قسم التشريح، كلية الطب البيطرى - جامعة الخرطوم

### المستخلص

تهدف هذه الدراسة لتقصى الدهون وإنزيمات العلامات القلبية فى قلب الجمل فى وجود الجسور العضلية. أخذت عينات الدم من سبعة جمال بالغة شوهدت بها جسور عضلية. جمعت العينات من مسلخ البقعة فى السودان. حللت العينات كيميويويا لإثنين من الإنزيمات القلبية (العلامات القلبية): كرياتين كائينز وتروبونين القلب. تراوحت قيم الكرياتين كائينز بين 25 و 48 وحدة ، بينما كانت تقريباً سالبة للتروبونين القلبي. سبعة من قلوب الجمال الطازجة استخدمت لدراسة الدهون الكلية، الدهون الثلاثية والدهون الفسفورية باستخدام التقنيات الكيميوحيوية. تراوحت القيم التى تم الحصول عليها للدهون الكلية، الدهون الثلاثية والدهون الفسفورية بين (18.62 و 22.55 جم) 0.84%، (2.23 و 3.43 جم) 0.11% و (0.37 و 0.60 جم) 0.019% على التوالي. النتيجة التى تم الحصول عليها لإنزيم العلامات توحى أن الجسور العضلية فى قلب الجمل قد تكون ظاهرة طبيعية على الرغم من أنها شذوذ خلقى فى الآخرين.