# A SURVEY ON SPONTANEOUS ATHEROSCLEROSIS OF CAMELS (CAMELUS DROMEDARIUS) IN IRAN\*

A. EZZI<sup>1</sup> and B. ZAKARIAN<sup>2</sup>

## SUMMARY

In a survey carried out on 200 camels (Camelus dromedarius) from different parts of Iran, three cases were diagnosed positive for atherosclerosis on gross and microscopic examination. A sex and age predilection is suspected. The complications of atherosclerosis were not present. To the best of the authors' knowledge this is the first report of atherosclerosis in camels.

### INTRODUCTION

Atherosclerosis, a disease of large and medium-sized is characterised by focal thickening of the tunica intimalis and usually is associated with fatty deposits. It is reported in domestic and wild herbivores as cattle (McMartin, Geer, Koestner and Corwell, 1970; Wiggers, Jacobson and Getty, 1971), sheep (Roser and Magarey, 1964), goats (Kenealy, 1974), pigs (Hill, 1972), deer (Jennings, Jennings and Burton, 1969), buffaloes (McKinney, 1968), elephants (McCullagh, 1972) and rhinocerus (McKinney, 1962). However, so far as the authors can ascertain there has been no report in camels. This report describes the results of a survey carried out on spontaneous atherosclerosis of camels in Iran.

#### MATERIALS AND METHODS

Thoracic and abdominal-aortas of 200 camels (*Camelus dromedarius*) slaughtered in Teheran abbatoir were examined for the presence of atherosclerosis. Of these, 112 were males and 88 females aged 2-12 years. Camels were brought to the abbatoir from different provinces of Iran.

The aortas were examined *in situ* with the naked eye and then cut open with a pair of artery scissors 20-30 min after slaughter. The different tunicae

<sup>\*</sup> Reprinted from: Trop. Anim Hith prod. (1979) 11,102-105

<sup>&</sup>lt;sup>1</sup> Present address: Razi Institute, PO Box 656, Teheran, Iran.

<sup>&</sup>lt;sup>2</sup> Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Teheran, PO Box 3262, Teheran, Iran.

were examined for variations in size, thickness, colour and resistance. Specimens for routine and histochemical microscopic examinations were taken and fixed in 10% buffered fromol saline; 6 nm paraffin sections were stained with haematoxylin and eosin (H &E), Van Gieson (VG), von Kossa's (VK) and Verhoeff (VH) for routine, connective tissue, calcium and elastica, respectively; 20 nm frozen sections were prepared and stained Oil-Red-O for lipids.

#### RESULTS

Atherosclerosis was diagnosed in 3 out of 200 camels. All were male and came from Shiraz (Fars Province, south-central part of Iran). One was 10 and the other 2 were 6 years old.

Macroscopically the lesions were whitish light of dark yellow. In 2 camels, viz. the 10-year-old and a 6-year-old one, the lesions were elevated protruding into the lumen of the aorta, measuring 4 mm in diameter and  $1 \times 3 \times 0.5$  cm, respectively, The first one was located in the arch of aorta and the latter 10 cm distal to the diaphragm. Both were rather hard and deeply attached to the inner layer of the aorta. In the third camel the lesion was not elevated and lay under the tunica intimalis, near the bifurcation of the anterior mesenteric artery. It was oval in shape with irregular edges and measured  $1 \times 3$  cm.

In addition, in a 12-year-old female camel from Zahedan (South-East Province) multiple small yellow plaques were observed in the thoracic aorta, covering an area of  $1 \times 3$  cm. Microscopically this was proved to be a parasitic infestation.

Microscopic examination of H & E stained sections in the 10-year-old camel revealed a mature fibrous plaque which was attached to the tunica intimalis. This consisted of fibroblasts with fusiform dark-staining nuclei, which were abundant at the base of the plaque at the site of its attachment to the tunica medialis. Here the cells were arranged perpendicularly to the long axis of the aorta. In the centre of the lesion and near to the lumen of the blood vessel their arrangement was parallel to the long axis of the aorta. The inner elastic membrane was dissociated and fragmented at the base and site of the attachment. This was also shown on VH stained sections. VG stained sections revealed that the plaque consisted of fibrous connective tissue. At the base of the lesion the smooth muscle cells were intermingled with the connective tissue components.

In the other 2 cases the intima was intact. The lesion was found to be located in the tunica medialis near to the tunica intimalis. It consisted of focal calcification surrounded by fibrous tissue and hyalinised smooth muscle cells. The nuclei of these cells demonstrated different stages of cellular necrosis. In addition to the fibroblasts, the calcified foci were occasionally surrounded by macrophages and lymphocytes. Section stained VG and VK were positive for connective tissue and calcium deposits. The VH stained sections showed that the elastica of the wall was fragmented in the centre of the lesion and changed its course at the boundaries.

#### DISCUSSION

Pathogenesis of atherosclerosis in man and primates has been studied in detail by Scott, Florentin, Daoud, Morrison, Jones and Hutt (1966), Scott, Jones, Daoud, Zumbo, Coulston and Thomas (1967) and Rose and Glomset (1973), who suggested 3 classes of changes, viz. pre-atheromatous changes or formation of fatty streaks, atheromatous lesions or necrosis of intimal cells and fibrous plaques. Parallel studies in animals other than primates, such as rats (Testo, Canestrini and Oldni, 1975), pigs (Hill, Lundberg and Titus, 1971) and chicken (Pick, Katz, Century and Johnson, 1962) have also been carried out. However, the sequence of events leading to the production of atherosclerosis in herbivorous ruminants is still obscure.

In one camel the lesion consisted of a fibrous which macroscopically and microscopically corresponded with the third stage of the above-mentioned changes, but the authors failed to demonstrate any fatty streak in paraffin and frozen sections of solid necrotic lesions in the intima of the aorta. The absence of fat droplets could possibly be attributed to the chronic nature of the process. In the other 2 cases the intima was intact and the outstanding lesion was the calcification of the tunica medialis. Calcification, either in the presence or absence of intimal changes, is recorded by Simpson and Harms (1966) and Smith, Jones and Hunt (1972). Nevertheless, it is worth mentioning that in many respects the medial calcification observed in this survey is similar to that of Mönkberg disease of man if the class of artery is ignored.

The examination of data for the influence of age, sex, and environmental factors showed that, in spite of the small number of positive cases, they could be significant, since all affected camels were in the age range of 6 - 10 years old, coming from the suburbs of Shiraz, in the south-east of Iran. Age influence is also reported by other investigators and it has been suggested that the levels of sphyngomyelin and phospholipids increase with age in the blood vessels (McMartin et al, 1970; Rouser and Solomon, 1969; Böttcher, 1964). Phospholipids are suggested to be atherogenic. In the present report, since it was a pilot study, biochemical and histochemical analyses were not done. Thus, it is difficult to attribute the atherosclerosis of camels to the substances mentioned. Simpson and Harms (1966) in a series of experiments carried out on atherosclerosis of turkeys showed a higher frequency in males. Epstein (1971) reported race difference and environmental influences in atherosclerosis of man. Our findings regarding the origin of the camels (i.e. Shiraz), suggests an influence of either the environment, the breed of camel or the effect of nutrition. As yet this is not clear but may be important from the epidemiological point of view.

#### ACKNOWLEDGEMENTS

This survey was supported by the Faculty of Veterinary Medicine, University of Teheran, Teheran, Iran.

#### REFERENCES

BOTTCHER, C. J. F. (1964). Proceedings of the Royal Society of Medicine, 57, 792-796.

EPSTEIN, F. H. (1971). Atherosclerosis, 14, 1-11.

HILL, E, G. (1972). Lancet, 26, 478 - 479.

HILL, E.G., LUNDBERG, W.O. & TITUS, J. L. (1971) Mayo Clinic Proceedings, 46, 620.

JENNINGS, I. W. A. R., JENNINGS A. R. & BURTON, M.A.F. (1969). Journal of Comparative Pathology, **79**, 65.

KENEALY, M. D. (1974). Abstracts International, 35, 2002.

MC MARTIN, D. N., GEER, J.C., KOESTNER, A. & CORWELL, D. G. (1970). Archives of Pathology, 89, 164.

MC KINNEY, B. (1968). Journal of Pathology and Bacteriology, 95, 301.

MC KINNEY, B. (1962). Lancet, 2, 281.

MC CULLAGH, K. G. (1972). Atherosclerosis, 16, 307.

PICK, R., KATZ, L. N., CENTURY, D. & JOHNSON, P. J. (1962). Circulation Research, 11, 811-819.

ROSER, B. J. & MAGAREY, F. R. (1964). Journal of Pathology and Bacteriology, 88, 73. ROSE, R. & GLOMSET, J. A. (1973). Science, 180, 1332.

ROUSER, G. & SOLOMON, R. D. (1969), *Lipids*, **4**, 232.

SCOTT, R. F., JONES, R., DAOUD, A. S., ZUMBO, O., COULSTON, F. & THOMAS, W. A. (1967). Experimental and Molecular Pathology, 7, 34.

SCOTT, R. F., FLORENTIN, R. A., DAOUD, A. S., MORRISON, E. S., JONES, R. M. & HUTT, M. S. R. (1966). Experimental and Molecular Pathology, 5, 12.

SIMPSON, C. F. & HARMS, R. H. (1966) Experimental and Molecular Pathology, 5, 183.

SMITH, H. A., JONES, T. C. & HUNT, R. D. (1972). Veterinary Pathology, 4th edn, Lea & Febiger, Philadelphia, 1151.

TESTO, R., CANESTRINI, C. & OLDANI, C. (1975). Journal of Pharmacy and Pharmacology, 27, 699.

WIGGERS, K. D., JACOBSON, N. L. & GETTY, R. (1971). Atherosclerosis, 14, 379.