
Myocardial Infarction in a Rhesus Monkey

Case Report

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Received 12 Mar 2005; accepted 28 Oct 2005

Introduction

Myocardial necrosis can be result from a number of causes including nutritional deficiencies, chemical and plant toxins, ischemia and metabolic disorder. The outcome of myocardial necrosis varies depending on the extent of the damage (Donald 2001, Jubb 1993, Radostits 1994, Vanvleet 1986). Myocardial infarction without demonstrable of atherosclerosis were reported in a rhesus macaque (Gonder 1982) and in a Kenya Baboon (Groover 1963).

Case history

A male rhesus monkey weighting 6 Kg without any primary clinical signs fell down on the surface of cage that presumed to be cardiac attack. Clinical examinations during shock sever tachycardia, dyspnoea and unconscious were noted and after few minute the animal died. There was not any problem during one year living in the cage. The animal had eaten all its food before its death. The animal necropsied. The heart configuration and size were almost normal, but large pale area was visible on the anterior wall of myocardium (Figure 1). Right ventricle was slightly dilated. A thrombus formed around chordae tendinae of the left atrioventricular valve and was loosely adherent to the endocardium. Lung and liver were congested. Liver was

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slightly enlarged. Whole heart and parts of internal organs such as liver, stomach, intestine, kidney, spleen lung and skeletal muscle were fixed in 10% formalin.

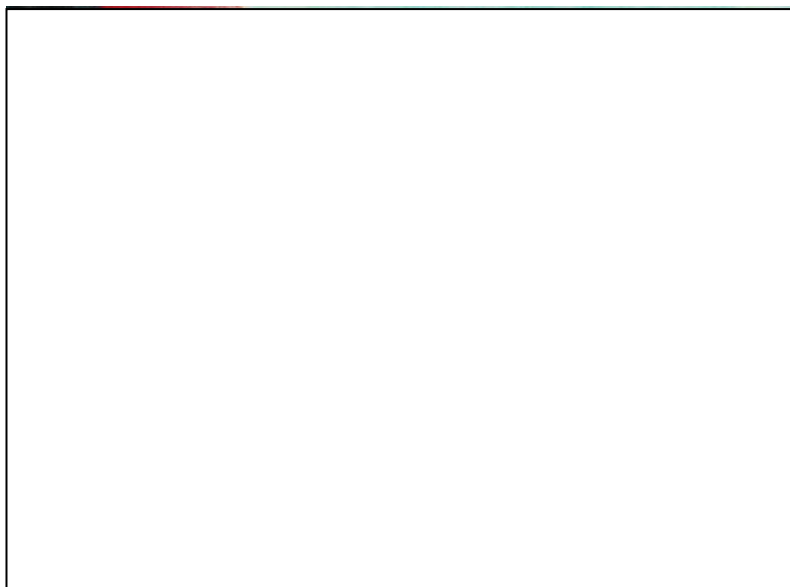


Figure 1. **Myocardial infarction**, pale area of myocardium indicated by arrow.

Tissues were processed by standard method and 5 micron paraffin block sections were prepared and stained with Haematoxylin and Eosin (H&E). Bacterial culture from bone marrow, liver and blood were negative. Serial sectioning from different parts of heart, myocardial necrosis were evident in some areas. The muscle fibers showed coagulative necrosis characterized by cellular swelling, nuclear hyperchromasia and loss of striation. Necrotic muscle fibers showed granular appearance, myocytolysis and sarcoplasmic vacuolation. In some areas hemorrhages between myofibers were noted (Figures 2 and 3). In some sections fibrin thrombi were found within the vessels of the base of heart. In lung thickening of alveolar walls and hyperemia were seen.



Figure 2. **Myocardial necrosis**, zone of normal and necrotic myofibers. The necrotic region indicated by arrow. H&E ,X100



Figure 3. **Myocardial necrosis with myocytolysis**, vacuolation and hemorrhages. H & E, X 200

Liver sections showed centro lobular congestion with accompanied by dilation of central veins and hepatic cord atrophy (Figure 4).

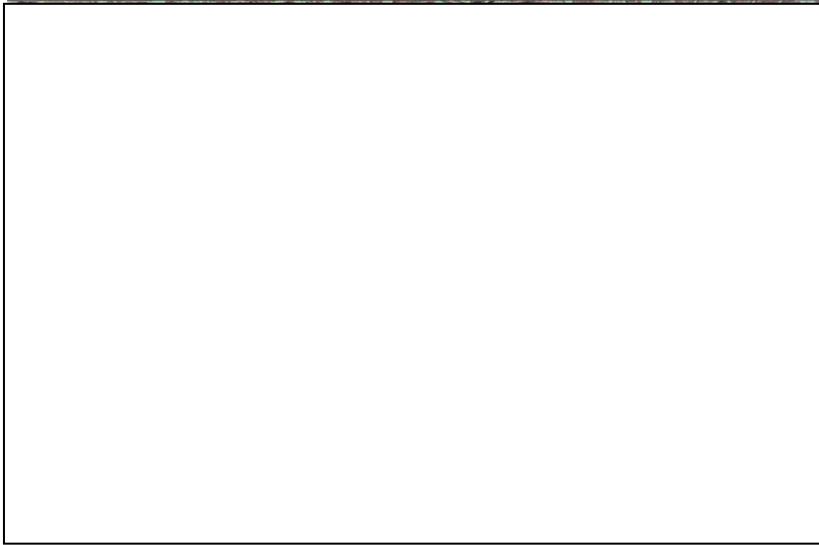


Figure 4. **Liver**, lobular center and sinusoids dilation and congestion .H&E, X 200

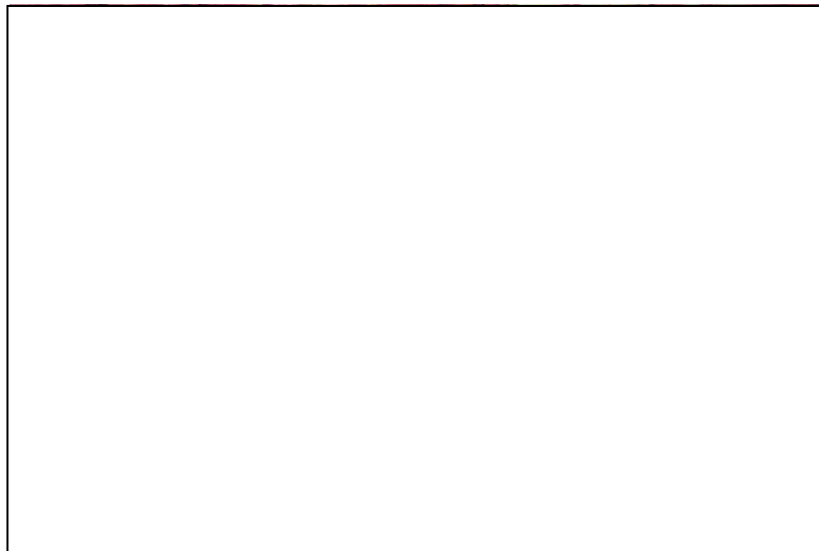


Figure 5. **Kidney**, different stages of tubular necrosis. H&E, X 200

Ischemic tubular necrosis and congestion of the kidney were also noticed (Figure 5). There were not seen any lesions in other organs.

Discussion

Histological and bacteriological examination there was not evidence of infectious disease. The etiology of heart lesions could not be determined with diagnostic tests. The hepatic and kidney lesions most probably are the consequences of secondary to the cardiac lesion. Syndromes are involved excessive exertion or stress where could be participated myocardial necrosis (Hollamby 2004, Jubb 1993). Stress –induced cardiac necrosis without accompanying skeletal muscle lesion. The cardiac lesions are presumed to result from sympathetic over activity and local catecholamine release in myocardium (Jubb 1993). Focal myocardial necrosis is also frequently observed as an individual finding on microscopic examination in many diseases, and its immediate pathogenesis can seldom be ascertained. The lesions are thought to have ischemic basis at the capillary level. Coronary embolism is a relatively common cause of focal myocardial necrosis (Jubb1993, Robbins 1984, Radostits 1994, Vanvaleet & Ferrans 1986). In human medicine the prevalence of myocardial infarction with normal coronary arteries are from 1% to 3%. Some thrombosis appear to have in arteries that have no morphologic evidence of prior atherosclerosis, other possible mechanisms are coronary emboli. Most cases however probably are attributable to vaso spasm. Spasm is not a phenomenon that can be recognized at autopsy and is a presumptive diagnosis when there is regional infarct (Damjanov & Linder 1996). The gross and microscopic appearance of myocardial necrosis is depending on the interval between the initial insult and death. Proliferative or thrombotic lesions completely occluding vessels in these areas are rarely observed (Jubb 1993, Robins 1984). Arteriosclerosis is a common cause of ischemic myocardial necrosis in human being, septic emboli and thrombi

the latter associated with disseminated intravascular coagulation may cause cardiac infarct (Hollamby 2004).

This case was happened out of 64 monkeys that housed each of them in one cage, the animals had good body condition and during one year all of them had same ration. Bacterial examination was negative and there was no any histological evidence of infectious in the tissues examined. Presence of loose thrombus in left ventricle and aorta and histological evidence of myocardial infarction may have been the cause of sudden death. Stress and psychological problem may predispose factors of infarction.

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