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CASE REPORT PEER REVIEWED | OPEN ACCESS

Myocardial steatosis: An autopsy-discovered case

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ABSTRACT

Introduction: Myocardial steatosis, a poorly documented pathology, is characterized by the accumulation of abnormal amounts of triglycerides in the cardiomyocytes. Myocardial steatosis is generally asymptomatic, but it can be the cause of heart failure. It is primarily of hypoxic or metabolic origin.

Case Report: We report a case of postmortem diagnosis in a 53-year-old male patient, without known his pathological history, who was a victim of sudden death from abdominal pain. The autopsy revealed diffuse atheromatous cardiac, aortic, and hepatic lesions.

Conclusion: It is very common for myocardial steatosis to be diagnosed postmortem due to its nonspecific symptomatology. The risk of mortality and morbidity could be reduced through a healthy lifestyle and regular biological examination to identify risk factors.

Keywords: Cardiomyocyte, Pathological anatomy, Steatosis

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INTRODUCTION

Steatosis or fatty degeneration is the abnormal accumulation of triglycerides in parenchymal cells. It is frequently observed in hepatocytes, which are significantly involved in lipid metabolism. Rarely, it is found in heart tissue, where it may be found in the epicardium, the endocardium, or the myocardium. Myocardial localization is the most severe type causing heart failure with a risk of sudden death [1]. The causes are usually hypoxic or metabolic. In this report, we describe a case discovered after an autopsy.

CASE REPORT

A 53-year-old male patient without a known pathological history with class II obesity (Body Mass Index of 38) died suddenly as a result of abdominal pain. External examination of the body did not find any lesions of violence. The abdominal wall of the subcutaneous adipose tissue was thick and measured 3.5 cm thick at autopsy (Figure 1). Examination of the viscera revealed a pancreas in a state of necrosis on two-thirds of the organ and an aorta which is the site of raised, ulcerated, or calcified yellowish plaques characteristic of advanced lesions of atherosclerosis (Figure 2A and B). The cardiac tissue presented a yellowish appearance due to fatty infiltration in certain places (Figure 3). The

other viscera, liver, kidneys, and other organs were macroscopically healthy. Microscopic examination showed that the pancreatic tissue was the site of massive cytosteatonecrosis. The cardiomyocytes displayed lipomatous infiltration, dissociating the myocardial fibers in some places (Figure 4). Hepatocytes showed macro- and microvacuolar steatosis lesions associated with mild portal lymphocyte inflammatory infiltrate (Figure 5). At the level of the aorta, atherosclerotic lesions were observed, consisting essentially of foam cells, lymphocytes, and cholesterol crystals within a fibrosis.

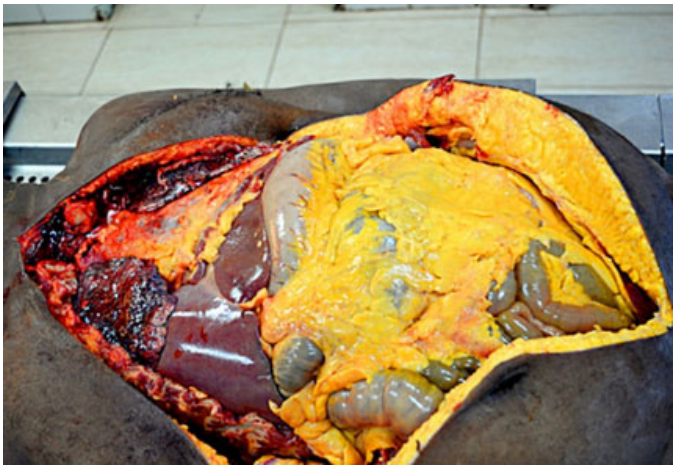

Figure 1: Thick subcutaneous adipose tissue.

Figure 2: Aortic arteriosclerosis: (A) ulcerated and calcified foci, (B) Aortic arteriosclerosis.

Figure 3: Slice of heart section showing fatty infiltration of the wall.

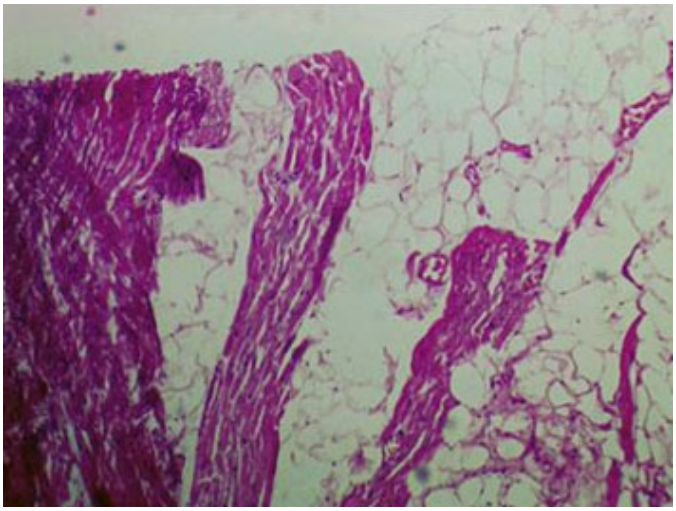

Figure 4: Myocardial steatosis. G400, H&E staining fatty infiltration of cardiomyocytes.

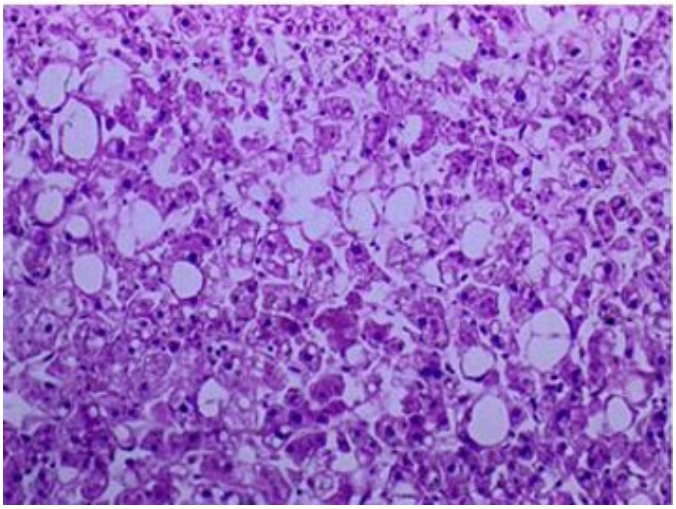

Figure 5: Hepatic steatosis. G400, H&E staining hepatocytes with intracytoplasmic lipid vacuoles.

DISCUSSION

Steatosis or fatty degeneration is the abnormal accumulation of triglycerides in parenchymal cells. It is frequently observed in hepatocytes, which are strongly involved in lipid metabolism. The causes are multiple: toxic (alcohol, drugs), nutritional, metabolic (diabetes, obesity), hypoxic, or infectious (viral hepatitis C) [2]. In developed countries, its most common cause is alcoholism [2]. In the normal state, fatty acids from adipose tissue or food are transported to hepatocytes, where they are esterified into triglycerides. They are then converted into cholesterol or phospholipids or oxidized into ketone bodies. Other fatty acids can be synthesized from acetate. The release of triglycerides by hepatocytes takes place in the form of lipoproteins after their conjugation with apoproteins. The accumulation of triglycerides can be linked, depending on the etiology, to an anomaly at the level of each metabolic stage, from the entry of fatty acids to their exit in the form of lipoproteins: alcohol is

toxic for the function of microsomal and mitochondrial hepatocytes, malnutrition decreases apoprotein synthesis, anoxia inhibits fatty acid oxidation, and fasting increases peripheral mobilization [2]. Myocardial localization is rare and associated with a significant risk of developing cardiovascular disease with the consequent possibility of sudden death [3]. Fatty infiltration of the myocardium could be the cause of heartbeat disorders with cardiocirculatory failure. The mechanisms of the occurrence of these arrhythmias have not yet been elucidated [4]. The incidence of myocardial steatosis is increased due to some other factors such as aging, female sex, obesity, insulin resistance, diabetes, and aortic atherosclerosis [5–7]. The multi-visceral localization of steatosis in our patient is consistent with a metabolic origin, caused by a lipid metabolism disorder. Prevention through the identification of risk factors remains the main challenge in the management of this disease [4].

CONCLUSION

Myocardial steatosis poses a challenge for antemortem diagnosis due to its non-specific symptomatology. A healthy lifestyle and regular biological examination of potential risk factors could contribute to the reduction of morbidity and mortality.

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Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.

ABOUT THE AUTHORS

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