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Case Report

Sudden Death by Myocardial Infarction in Children

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Abstract

We report the case of fatal myocardial infarction in a 14-year-old child. In these antecedents, he had an 11-year history of nephrotic syndrome due to mesangial proliferation disease. He presented a generalized edema, acute vomiting and chest pain. Faced with the worsening of his health condition and the appearance of consciousness disorders, he consulted in the emergency room, arrived in cardiorespiratory arrest, not recovered despite adequate resuscitation. At autopsy, we noted the presence of acute pulmonary edema associated multiple lipid plaques in the coronary arteries. Pathological examination confirmed the presence of complete stenosis of the coronary arteries associated with myocardial infarction. Despite the exceptional nature of these observations and because of their serious and fatal prognosis, it seems important to us that cardiological follow-up is essential for any child with nephrotic syndrome.

Keywords: Autopsy; Acute myocardial infarction; Death; Nephrotic syndrome

Introduction

Sudden death in children is a rare phenomenon at present, especially for children over one year of age. Several etiologies may be responsible for this death [1]. Moreover, the cardiovascular origin of this sudden death remains the most incriminated in this family drama [2]. Children with nephrotic syndrome require special monitoring due to the high comorbidity and mortality linked to this pathology [3]. In the following report, we describe an uncommon case of suspect death of a teenager whose autopsy revealed a natural death of myocardial infraction whose diagnosis was never suspected before his death.

Case Reports

A 14-years-old boy with no family history holds a personal history of nephrotic syndrome since the age of 3 years old. A nephrologist followed him, and put him under a corticotherapy and a diuretic treatment. It was a notion of poor compliance with his treatment and his diet. In the previous days before his death, he had started to show a generalized edema. He did not show any sign of renal failure or of primary disease, and presented normal blood pressure. The day of his death, the young boy presented an acute vomiting and chest pain. Faced with the worsening of his health condition and the appearance of consciousness disorders, he consulted in the emergency room, arrived in cardiorespiratory arrest not recovered despite adequate resuscitation. We received the cadaver the day of his death. On external examination of the corpse, we noted the absence of traumatic lesions. We noted also the presence of generalized edema. At autopsy, there was an acute pulmonary edema associated multiple lipid plaques in the coronary arteries.

Pathological examination confirmed the presence of complete stenosis of the coronary arteries associated with myocardial infarction and lipid deposition on the wall of the aorta (Figures 1-3). The histological examination of the various samples taken shows a polyvisceral congestion: an acute edema of the lungs without embolism. At the level of the heart, the coronary arteries are completely obliterated by a fibro-inflammatory atherosclerotic plaque rich in cholestérols and surmounted by a fibrino-cruoric thrombus obliterating. The myocardium contains at the level of the left ventricle a focus of recent infarction strewn with polynuclear with the presence of some vascular neo-capillaries. The kidney is the seat of tubulo-interstitial fibro-inflammatory lesions, glomeruli-tubular calcifications, some glomeruli are in bread to be sealed, and others are in the process of sclerosis. The remains of viscera do not show any anomalies.

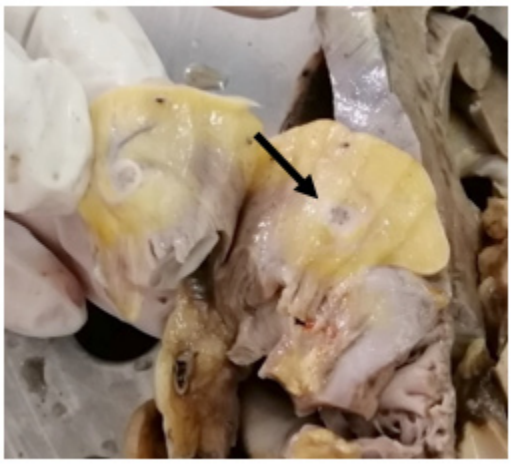

Figure 1: Coronary trunks with an increase in diameter (6 * 5 mm) and severe luminal narrowing.

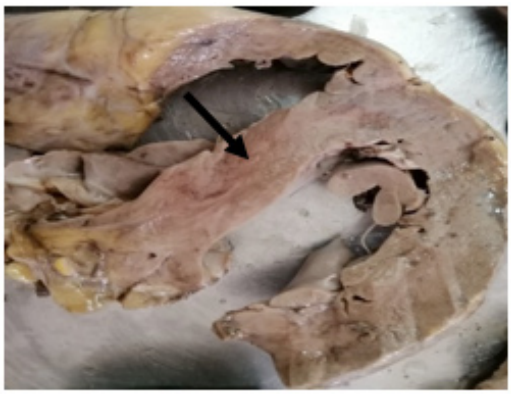

Figure 2: Septal myocardial infraction.

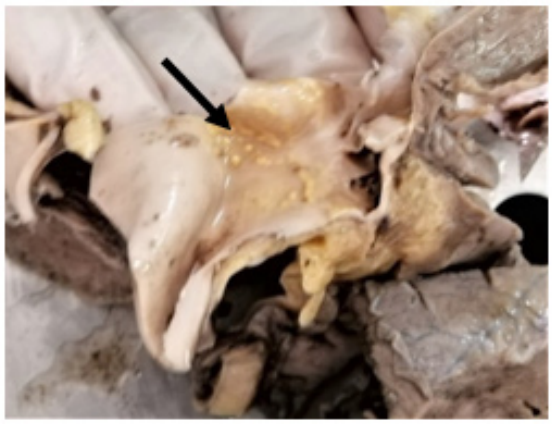

Figure 3: Lipid deposition on the wall of the aorta.

Discussion

Patients with nephrotic syndrome have several risk factors for developing ischemic heart disease, including steroid therapy, increased clotting tendency, hyperlipidemia and platelet hyperfunction [4,5]. Nevertheless, the incidence of ischemic heart disease among nephrotic patients is hotly debated. Acute myocardial infarction is distinctly unusual in the young pediatric age group. When it occurs, it is usually associated with anatomical heart or coronary artery disease, or with rare metabolic conditions [6]. In an autopsy study of 40 children with nephrotic syndrome due to mixed renal disease, a significantly increased incidence of mild to severe atherosclerotic changes was found when compared with 29 matched controls who died of non-renal causes [7].

The existing controversies regarding the incidence of atherosclerosis among nephrotic patients may be explained by several factors [4]. Nephrotic syndrome is often intermittent and early atherosclerotic changes may be reversible [8]. Therefore, even if initial lesions are formed during relapses of nephrotic syndrome, these lesions may resolve during the remission phase of the disease [3]. Most studies examining the relationship between nephrotic syndrome and atherosclerosis have included middle-aged and elderly patients, who may have other risk factors or a coexisting pathological condition leading to atherosclerotic changes [9]. This could obscure a mild to moderate propensity for atherosclerosis among nephrotic patients who do not have such risk factors or condition [10]. Long-term follow-up of a large cohort of nephrotic patients is lacking.

The evolution of clinical symptoms can take up to several decades from the development of the earliest atherosclerotic lesions, and our ability to diagnose asymptomatic cases is limited. As a result, an excess of atherosclerotic morbidity in nephrotic patients may be missed by studies with follow-up shorter than a few decades. In summary, the presence of an acute myocardial infarction in this nephrotic child suggests a possible role for nephrotic syndrome in the evolution of symptomatic atherosclerosis. In this process, hyperlipidemia could be the primary insult but other factors commonly associated with nephrotic syndrome may also play a role: hypercoagulability, platelet hyperfunction and drug treatment. We cannot exclude the possibility that, in order for acute myocardial infarction to develop in a nephrotic child, familial predisposition for lipid abnormalities must preexist.

Conclusion

Our case suggests that children with long-lasting nephrotic syndrome may be at increased risk for ischemic cardiovascular events, due to hyperlipidemia as well as a hypercoagulability state. Thereby, the management of children with nephrotic syndrome

should be multidisciplinary, and regular monitoring in a cardiologist is essential to detect the associated ischemic heart disease to avoid the dramatic development of this type of complication.

Conflicts of Interest

None.

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