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CASE REPORT Open Access

Premature atherosclerosis and acute coronary syndrome in a child with end-stage renal disease

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Abstract

Background: Although acute coronary syndrome is rare in children, it is the most important cause of mortality in children with end-stage renal disease.

Case presentation: Here, a 16-year-old pediatric patient, who has been on dialysis since the age of 3, and who was diagnosed with acute coronary syndrome and placed an emergency percutaneous transcatheter stent in the left anterior descending branch of the left coronary artery is presented. It is important that the present patient does not have any electrocardiography findings in favor of cardiovascular disease and that he cannot fully explain the complaint of chest pain due to his mental retardation.

Conclusions: Early detection of acute coronary syndrome is life-saving, especially in children with chronic kidney disease.

Keywords: Acute coronary syndrome, End-stage renal disease, Myocardial infarction, Children

Background

Cardiac causes and myocardial ischemia are rarely detected in children admitted to the hospital with chest pain compared to adults. Although acute coronary syndrome (ACS) is rare in children, the most common causes of ACS in children are coronary artery anomalies (CAAs), congenital heart diseases (CHDs), vasculitis, congenital prothrombotic disorders, and iatrogenic coronary artery injuries. However, cardiovascular disease (CVD) is a significant cause of mortality and morbidity in children with chronic kidney disease (CKD). Therefore, chest pain is more important in children with chronic kidney disease than in the healthy population, and a careful differential diagnosis should be made [1]. Herein, we present a 16-year-old boy with mental retardation and CKD presented with ACS due to premature atherosclerosis. We also aimed to highlight the clinical, laboratory, and imaging findings of the present case.

Case presentation

A 16-year-old male patient followed up with the diagnosis of both end-stage renal disease (ESRD) developed due to vesicoureteral reflux and mental-motor retardation had been receiving dialysis treatment since the age of 3. After peritoneal dialysis treatment for the first 3 years, he was being followed in hemodialysis (HD) treatment for the last 10 years. Because of his motor-mental retardation, the patient could not make sentences, express himself using single words, and walk with a broad base and short distances. According to his mother's statement, in his last visit, he had started to hold his rib cage intermittently for the last three days and could not lie on its side. He did not have a history of fever, cough, or trauma, and he did not use any other medicines or herbal products other than those he used for chronic

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kidney disease. Until the arteriovenous fistula was opened 3 years ago, the patient had to undergo six HD catheter revisions.

He received multiple inpatient treatments due to lung and catheter infections. A chronic calcified thrombus in the superior vena cava (SVC) was detected in the unenhanced chest computed tomography (CT), which was performed 3 years ago when he received treatment for a lung infection (Fig. 1). In the control, chest CT obtained during the period when he was followed up for lung infection 1 year ago, the persistence of calcified thrombus in the SVC and newly emerging widespread atherosclerotic calcifications in the left anterior descending (LAD) coronary arteries were detected (Fig. 2). In addition to these complications, the patient had osteitis fibrosa cystica, also known as brown tumors (proven by bone biopsy), due to renal osteodystrophy. The patient received oral anti-phosphate and active vitamin D treatments since his first diagnosis of chronic kidney disease (CKD). Due to his mental retardation, he did not comply with a phosphorus-poor diet. Cinacalcet has been added to treatment for the last 2 years, but the patient started to be unable to walk during follow-up. For this reason, pamidronate treatment was also given. He started walking again after cinacalcet, and pamidronate treatments were given during follow-up. Parathyroidectomy was planned 1 year ago due to the obvious clinical findings of renal osteodystrophy and very high parathyroid hormone (PTH) values, but since the patient could resume walking and his PTH levels, regressed the need for parathyroidectomy in the follow-up period was ruled out.

The physical examination findings of the patient with the improved general condition were as follows: body weighs 59 kg (10 p); height, 146 cm (< 3 p); BMI, 27.6 kg/m^2 (90 p); uremic skin color; normal respiratory sounds; and normal cardiological examination findings without edema. His biochemical test results were as follows: white blood cell count, 9.52 K/uL; hemoglobin, 13.2 g/dL; platelet count, 355 K/uL; urea, 164 mg/dL; creatinine, 8.9 mg/dL; albumin, 49.9 g/L; calcium, 9.5 mg/dL; phosphorus, 9.1 m/dL; PTH, 648 ng/L; triglyceride, 256 mg/dL; total cholesterol, 187 mg/dL; HDL cholesterol, 34 mg/dL; VLDL cholesterol, 51 mg/dL; LDL cholesterol, 102 mg/dL; erythrocyte sedimentation rate, 29/h; C-reactive protein, 20.3 mg/L (< 5); CK-MB, 8.9 μg/L (0–5); cardiac Troponin T (cTnT), 471 ng/L (0–14). All of his test results were evaluated in the blood samples obtained just before the application of hemodialysis. ECG was within normal limits for his age, and there was no dysrhythmia or ST-T variation. In the echocardiographic evaluation, no pathological finding was found, except for mild tricuspid valve insufficiency. Left ventricular systolic functions and dimensions were within normal limits. Left ventricular ejection fraction (LVEF) was calculated as 61%. There were atelectatic changes consistent with chronic lung disease on chest X-ray without any finding in favor of pneumonia and pleural effusion.

Since ECG findings of the acute coronary syndrome and arrhythmia and echocardiographic findings in favor of other possible cardiological pathologies were not detected, the patient was hospitalized and monitored. In serial cTnT measurements, the cTnT level increased up to 1469 ng/ml. Control echocardiographic evaluation showed that left ventricular systolic functions decreased. LVEF was calculated as 50%. Since calcification was detected in the

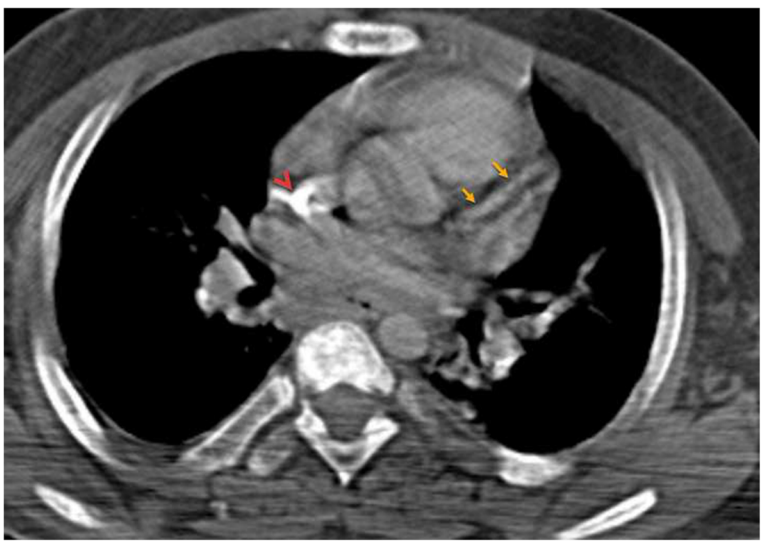

Fig. 1 The un-enhanced chest CT image obtained 3 years ago shows a chronic calcified thrombus (arrowhead) in the superior vena cava. Left anterior descending (LAD) coronary artery (arrows) is normal, and no calcific atherosclerotic plaque is seen

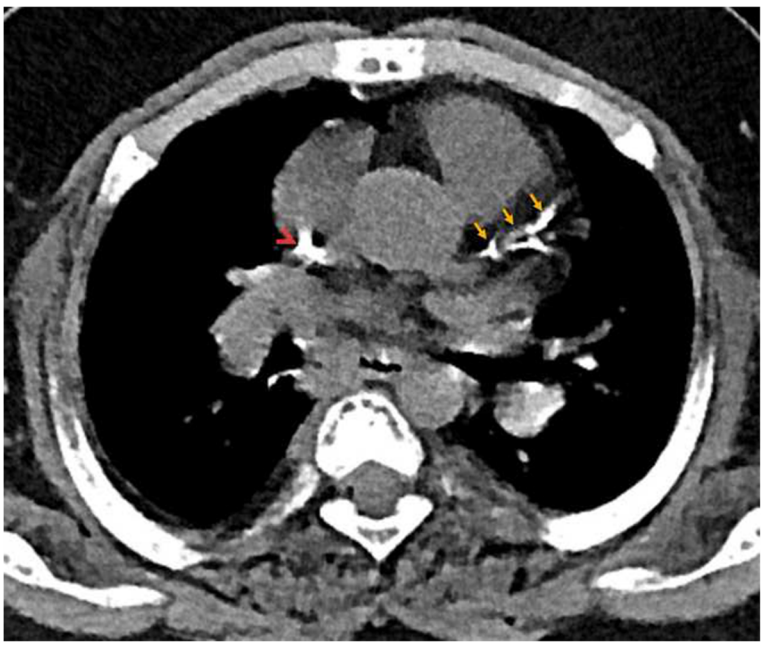

Fig. 2 In the contrast-enhanced chest CT image obtained 1 year ago, the persistence of the chronic calcified thrombus (arrowhead) in the superior vena cava and newly formed calcific atherosclerotic plaques are observed in the left anterior descending (LAD) coronary artery (arrows)

coronary arteries on chest CT obtained 1 year ago, coronary CT angiography was performed, and a total occlusion was detected in the left anterior descending (LAD) coronary artery (Fig. 3). With the acute coronary syndrome diagnosis, conventional emergency angiography was performed, and occluded LAD was observed. A percutaneous transluminal angioplasty and stenting was performed in the LAD coronary artery (Fig. 4). During follow-up, the patient's cTnT levels gradually decreased, and LVEF was calculated as 60% in control echocardiography.

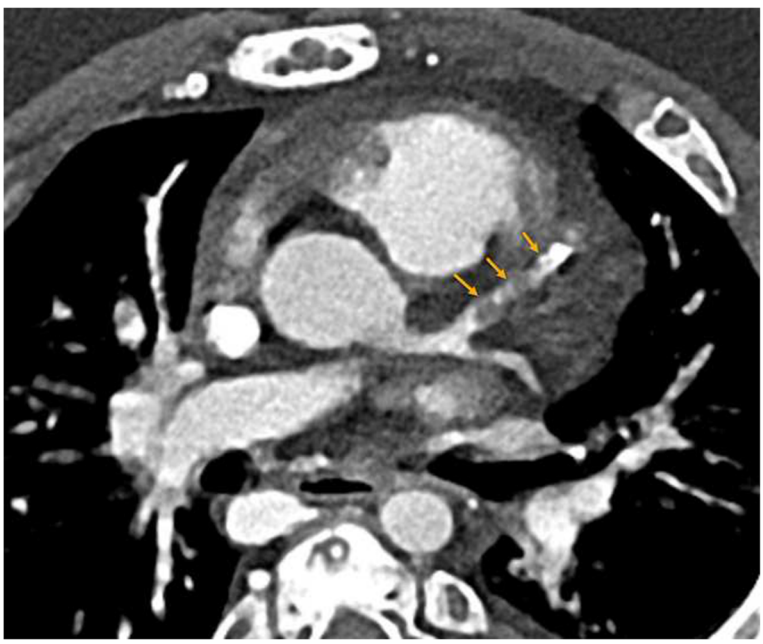

Fig. 3 The coronary CT angiography image obtained on the patient's last admission shows calcific and non-calcific atherosclerotic plaques (arrows) causing subtotal occlusion of the left anterior descending (LAD) coronary artery

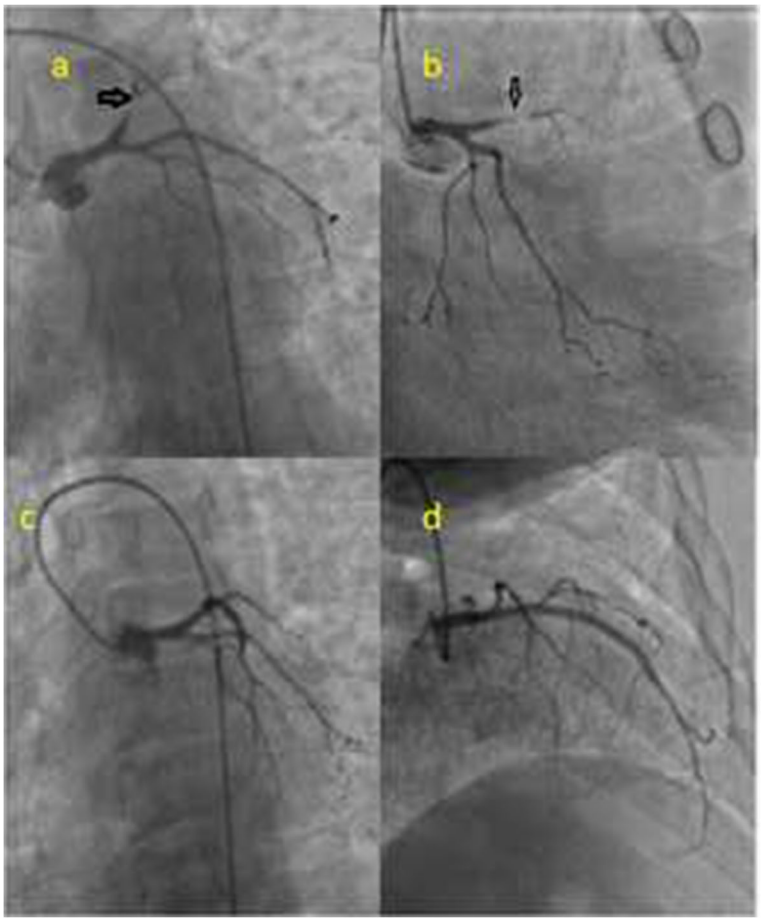

Fig. 4 **a** Complete occlusion of the left anterior descending (LAD) artery is seen in the left caudal oblique view before transcatheter intervention (arrow). **b** Complete occlusion of the LAD seen in the right caudal oblique image before transcatheter intervention (arrow). **c** Stents placed in the LAD seen in the left cranial oblique image after the transcatheter intervention. **d** Right cranial oblique view after transcatheter intervention stents placed in LAD are patent

Discussion

The first diagnosis to be considered in a patient with suspicious chest pain with troponin elevation is an acute coronary syndrome. However, since this patient is a child and had not an ECG finding in favor of acute myocardial infarction, other reasons that may cause cTnT elevation should be excluded. CTnT also increases in conditions such as myocarditis, pericarditis, heart failure, arrhythmia, stroke, sepsis, seizure, trauma, and intoxication [2]. In patients with chronic kidney disease, the cTnT level may increase without clinical signs of myocardial damage and ECG changes [3, 4]. These patients should be monitored on an inpatient basis. In the follow-up, troponin level is monitored by serial measurements. Along with close monitoring of vital signs, intermittent ECG and echocardiographic checks are performed. If the cTnT level continues to increase during the follow-up, coronary angiography (computed tomography and/or conventional) imaging should be performed [1, 3, 4].

The reference range for cTnT level between the ages of 1 and 18 years is similar to that of adults [5]. It is known that troponin level is permanently high in patients with chronic kidney disease. This situation makes the differential diagnosis of acute coronary syndrome difficult, especially in adult patients. Although the cause of persistent cTnT elevation in patients with chronic kidney disease is not known precisely, left ventricular hypertrophy, microinfarcts, oxidative damage, and decreased troponin clearance in addition to these pathologies are reported as possible etiological reasons [1, 3, 4].

CVD is the most common cause of death in patients with chronic kidney disease (CKD) in both adults and children. One-fourth of deaths in children with ESRD are due to CVD [1]. It has been reported that the risk of calcification and atherosclerosis in the coronary arteries is increased in patients with ESRD [6]. In addition to known risk factors such as hypertension, dyslipidemia, obesity, and abnormal glucose metabolism, bone mineral

disease, chronic inflammation, volume burden, and anemia are also associated with high cardiovascular risk in CKD. Although there are general risk factors for CVD disease, such as hypertension and dyslipidemia, the present case had a severe bone mineral disease. He had been on dialysis for 13 years, suggesting that these are the main causative factors for premature atherosclerosis and acute myocardial infarction he experienced in childhood. Myocarditis is one of the most likely diagnoses in such a case with left ventricular systolic dysfunction together with cTnT elevation. In this case, recovery of cTnT and ventricular functions after coronary intervention ruled out the diagnosis of myocarditis.

Conclusions

Although acute coronary syndrome is rare in the pediatric age group, it should be kept in mind in children with chronic kidney disease.

Abbreviations

ACS: Acute coronary syndrome; CAAs: Coronary artery anomalies; CHDs: Congenital heart diseases; CKD: Chronic kidney disease; CT: Computed tomography; ESRD: End-stage renal disease; HD: Hemodialysis; LAD: Left anterior descending; SVC: Superior vena cava

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Authors' contributions

TB and MY contributed to the conception and design of the work. Analysis and interpretation of data were performed by DG and SY. İG, NY, FU, and GN, were involved in the drafting of the work, and it was revised by TB and SY. All authors read and approved the final manuscript.

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Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent from the child's parent for publication was obtained.

Competing interests

All authors declare that they have no competing interests.

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