Vomiting as the initial clinical presentation of myocardial infarction in children with anomalous left coronary artery from the pulmonary trunk

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Anomalous origin of the left coronary artery from the pulmonary trunk is a rare condition. The clinical presentation is usually nonspecific and varies from completely asymptomatic form to sudden cardiac death. We report a two-month-old infant with vomiting as a presenting symptom of anomalous origin of the left coronary artery from the pulmonary trunk.

Key words: coronary, infant, infarction.

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Case Report

A two-month-old male infant was admitted to the Clinic of Pediatric Surgery - Clinical Center Nis, on 4 October 2005. The baby was born from the first term pregnancy via normal delivery, with an Apgar score of 10 and a body weight of 3750 g. He was free of symptoms during the first two months of life. On the day of admission the baby became pale, irritable, refused to eat and started vomiting.

With the aforementioned symptoms, he was referred to the Department of Pediatric Surgery. After the physical examination, routine laboratory analysis and plain abdominal roentgenogram, surgical disease was excluded and the baby was referred to our institution.

At the time of admission to the Clinic of Pediatrics, the infant was afebrile, irritable, pale, with cold extremities, sweating, tachypnea (61/min), and tachycardic heart rate (HR) of 180/min. The physical examination revealed weak cardiac sounds, and pulmonary auscultation indicated normal breathing sounds.

After initial assessment, an intravenous line was established and blood was taken for laboratory evaluations. The laboratory results showed elevated cardio-specific enzymes: isoenzyme fraction of creatine phosphokinase activity (CPK-MB) (CPK: 325 U/L and CPK-MB: 209.7 U/L), and high values of α-hydroxybutyrate dehydrogenase (HBDH) activity (370.3 U/L) and cardiac troponin I (14.8 ng/ml). Blood analyses showed mild hypochromic anemia (Er: 3.8%, Hb: 9.8 mg/dl, Hct: 31%, MCV: 78 fl, and RDW: 15.5%), with normal leukocyte differential as well thrombocyte count.

Having considered the results of these analyses, we also performed additional non-invasive diagnostic procedures: chest roentgenogram, echocardiography (ECG) and heart ultrasound. Chest roentgenograms showed a significant cardiomegaly and ECG showed signs of anterolateral myocardial infarction (Fig. 1). ECG examination revealed minimal mitral regurgitation, increased left ventricular internal dimensions in diastole (LVIDd: 45 mm), decreased global contractility, as well as segmental contractility disturbance (fractional shortening [FS]: 9-11%) (Fig. 2). The color and pulsed Doppler imaging clearly showed the diastolic retrograde flow from the left coronary artery to the main pulmonary artery (Fig. 3).

Finally, the patient was referred to a tertiary pediatric cardiology institution, where the diagnosis of anomalous left main coronary

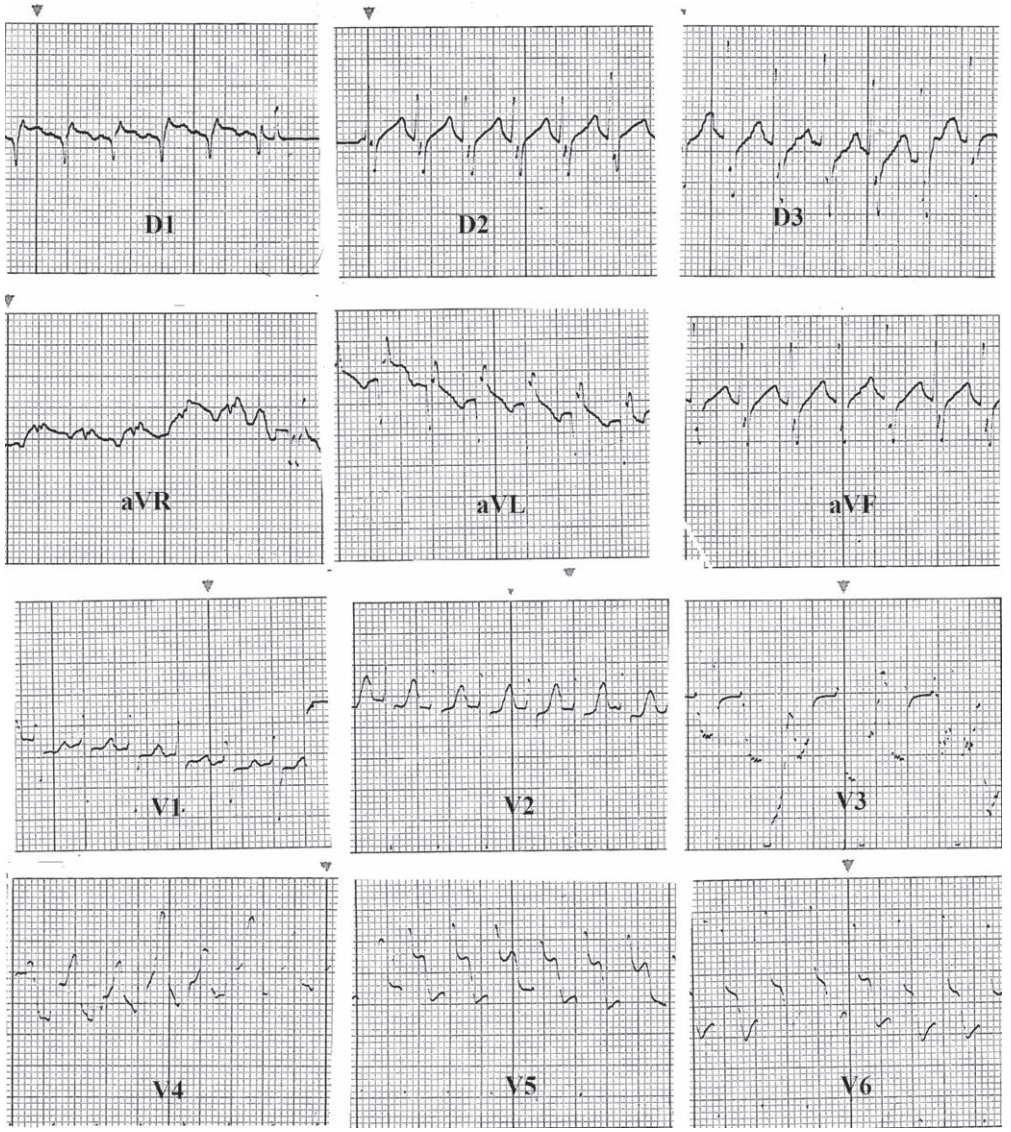

Fig. 1. Deep Q waves with ST elevation in leads I and aVL as well as left precordial leads.

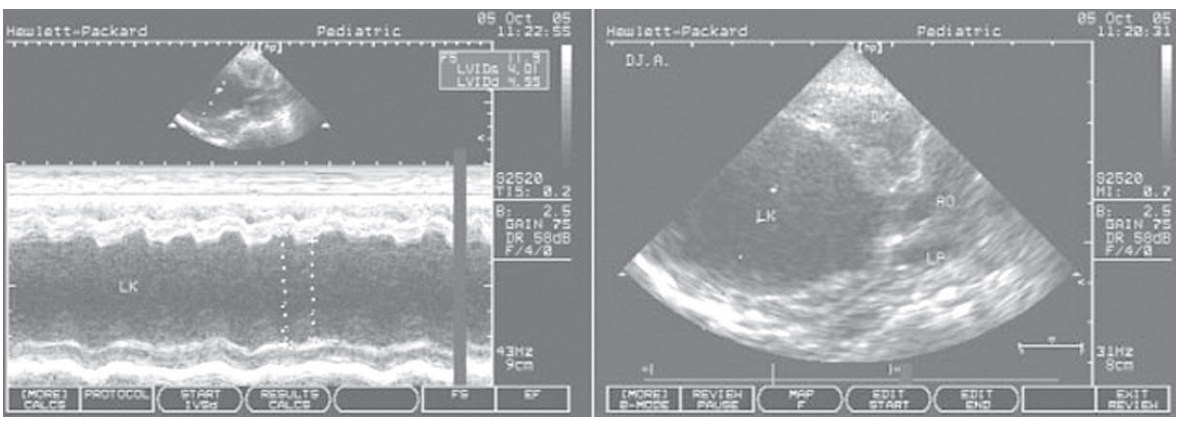

Fig. 2. Decreased left ventricle contractility and dilatation.

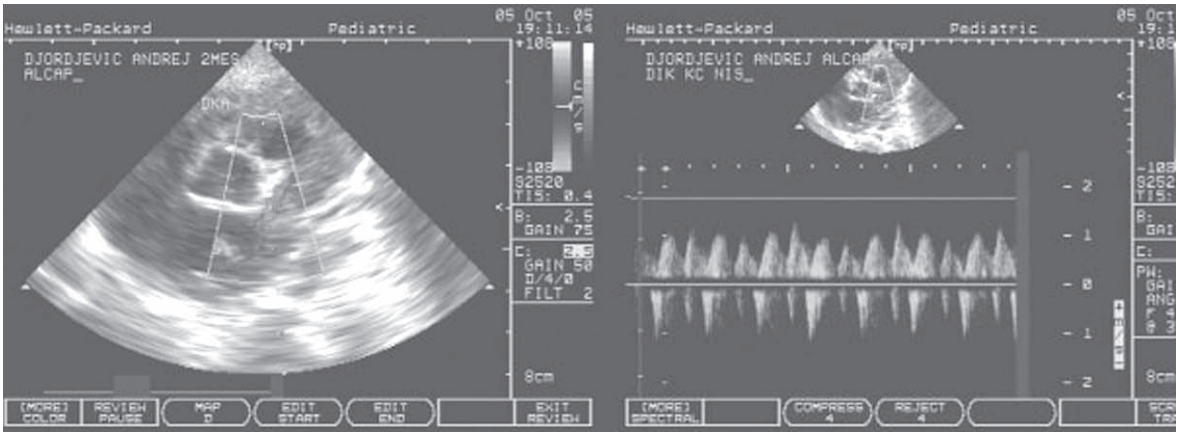

Fig. 3. Diastolic retrograde flow from the left coronary artery to the main pulmonary artery.

artery originating from the pulmonary artery (ALCAPA) was definitely established on the third hospital day, after preoperative aortography (Fig. 4).

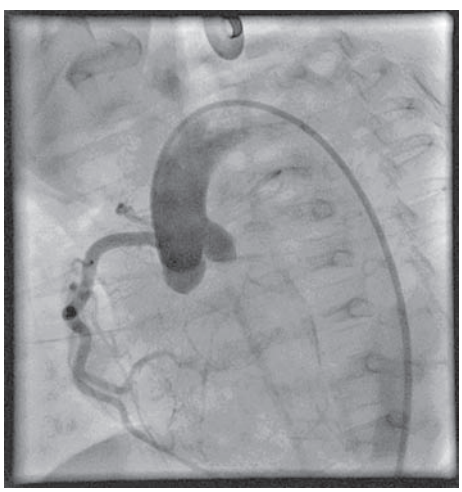

Fig. 4. Right coronary angiography showing dilated right coronary artery (RCA) filling of left coronary system through collaterals.

After cardiac catheterization, despite continuous maximal inotropic support, the baby died with signs of terminal heart failure.

Discussion

Anomalous left main coronary artery originating from the pulmonary artery (ALCAPA) is a rare cardiovascular congenital anomaly, with a reported incidence of one in 300,000 live births[5].

The pathophysiologic consequences of this condition are directly related to the adequacy of perfusion of the left ventricular myocardium after birth. During the first weeks of life, symptoms are rare, reflecting the elevated pulmonary vascular resistance that persists during the neonatal period. As soon as pulmonary vasculature resistance (PVR) falls, flow from the pulmonary artery (PA) to left coronary artery (LCA) reverses, enabling coronary steal effect, with the majority of infants (except those with excellent right coronary artery [RCA] to LCA collaterals) becoming symptomatic[7].

The clinical presentation is usually nonspecific and varies from completely asymptomatic form to sudden cardiac death[1-3].

A much more specific clinical picture suggestive for this entity often has late clinical presentation, including signs of congestive heart failure. According to Johnsrude et al.[8], even 18% of infants <2 years of age, presenting with dilatated cardiomyopathy and congestive heart failure, had ALCAPA, which underscores the importance of its diagnosis[4,8].

Symptoms like pallor, irritability, diaphoresis, crying, restlessness, feeding difficulties, sudden paroxysmal abdominal pain and vomiting are often misinterpreted as a food intolerance or infantile colic[9].

In this regard, early recognition of this rare entity remains a diagnostic challenge to the pediatricians and pediatric cardiologists, mainly due to nonspecific symptoms attributed to feeding difficulties. Vomiting is frequent and quite a nonspecific symptom in the youngest age group, rarely attributed to cardiovascular disease.

We would like to call attention to our patient, a two-month-old infant with ALCAPA, who presented with abrupt onset of abdominal colic and vomiting, suggestive of acute surgical disease.

Laboratory values of elevated CPK-MB fraction (CPK-MB fraction >50%) and high values of HBDH as well as cardiac troponin I, coupled with the clinical picture (physical findings, ECG pattern and echocardiographic examination) and lethal disease outcome, pointed to the cardiogenic origin of vomiting in our patient with ALCAPA.

We presumed that rapid fall in PVR provoked profound coronary steal effect with massive myocardial infarction or subsequent cardiogenic shock in our patient.

Isolated vomiting as well as colic in infants are quite nonspecific symptoms, indicating gastrointestinal pathology in most cases. There is still misapprehension that nausea and vomiting following myocardial infarction represent only inferior infarct localization. Herlihy et al.[6] found that cardiogenic nausea and vomiting are associated with larger myocardial infarctions but not a particular location. Additionally, Japanese experience with post-Kawasaki children younger than 4 years showed vomiting to be the most common presenting symptom of myocardial infarction[11].

Since a differentiation of ALCAPA, myocarditis and other forms of dilatated cardiomyopathy in infants has great clinical implication, it would be of great importance to reconsider some "nonspecific" clinical signs in similar clinical cases.

Sudden, isolated colic and vomiting as the consequence of myocardial infarction or subsequent cardiogenic shock in infants with ALCAPA could be one of them.

This clinical signs is never a presenting and/or isolated finding in children with myocarditis or other forms of dilated cardiomyopathy, as in our case.

Until ECG screening becomes a widely accepted tool and integral part of routine neonatal evaluation, vomiting as the cardinal presenting symptom of ALCAPA in infants should be considered in some cases[10].

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