Case Report

Pediatric Education and Research
Volume 8 Number 1, January – March 2020
DOI: https://dx.doi.org/10.21088/per.2321.1644.8120.6

Dengue Myocarditis, Bicuspid Aortic Valve, Acute Chest Pain in a 12-year Boy Treated with Streptokinase

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How to cite this article:

Jayendra R Gohil, Atul M Sheladiya, Chintu C Chaudhary, et al. Dengue Myocarditis, Bicuspid Aortic Valve, Acute Chest Pain in a 12-year Boy Treated with Streptokinase. Pediatr Edu Res. 2020;8(1):35–38.

Abstract

Myocardial ischemia and myocardial infarction is rare in children, with Kawasaki disease in children and substance abuse in adolescents, being the main cause. We present a case of a 12-year child suffering from Dengue, with bicuspid aortic valve, complicated by aortic valve insufficiency, who presented with chest pain and ECG changes of myocarditis and acute coronary syndrome; a rare occurrence. He was referred to us with complains of fever, cough, black colored stool, burning abdominal pain, and vomiting after 4 days of hospitalization at a district hospital as dengue illness. On day 9 of illness, he developed large hematemesis, with severe precordial chest pain, and was diagnosed initially as a case of acute myocardial ischemia due to myocarditis following dengue fever. ECG showed ST depression and troponin-I was markedly raised. Oxygen, ventilator, inotropes, streptokinase in divided doses (after bleeding had stopped), and steroids, zinc and vitamins C, E were administered. ECG changes reversed in a few hours and *q* waves started to appear. Later, 2D ECHO showed bicuspid aortic valve with aortic valve insufficiency. Myocardial enzymes, troponin-I and ECG returned to normal after 14 days. He was discharged on cardio supportive medications and advised valve replacement. Bicuspid aortic valve may remain undiagnosed till adolescence and can present rarely as acute myocardial ischemia following Dengue that was treated with streptokinase.

Keywords: Bicuspid aortic valve; Adolescent; Electrocardiogram; Acute myocardial infarction; Streptokinase; Dengue.

Introduction

Though rare, Myocardial infarction and ischemia (MI) in adolescents is associated with substance and alcohol abuse. We report a young patient (12-year) with MI with bicuspid aortic valve, an association that has been reported only once.[1,2]

Case History

A 12-year-old boy was referred from District hospital (120 km) as clinical dengue fever, after 4 days of hospitalization with: fever since 4 days, cough since 3 days, black stool since 2 days, burning abdominal pain since one day, and vomiting two

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Received on 10.01.2020, **Accepted on** 19.02.2020

episodes. His vitals were Temp- 37.3 C, Pulse: 82/min, Respiratory rate: 24/min, BP: 96/62 mm Hg. IV fluid was started (4 ml/kg/h). No Antibiotics were given. Patient was improving except for dry cough. On D3 hemoglobin level reduced from 11 to 9 g/dl and platelet from 60,000 to 35,000/mL. On D8, hemoglobin further reduced to 5 g/dl, while there was no bleeding from any site. On D9, patient developed two episodes of hematemesis and severe burning retro-sternal pain. Red cell concentrate was given and stomach wash with cold saline was done, along with iv pantoprazole, ethamsylate, haemo-coagulase, and tranexamic acid. There was no improvement and retro-sternal pain did not respond to analgesics. 400 ml of blood was collected in seven hours via Ryle's tube.

Vitals: temp-N, pulse-102/min, BP-86/66 mm Hg, resp. rate-30/min. Auscultation revealed a murmur thought to be haemic.

Investigation: On ninth day, as per Table 1, and cardiac markers were: Creatinine kinase (CKMB)-35 U/L, Lactate dehydrogenase (LDH)- 500 U/L.

Table 1: Investigations on ninth day

Investigations	Values
Hb/Hematocrit	4.2 g/dL / 12%
TLC K/uL	6
DLC P/L/E/M %	50/45/3/2
Platlets K/uL	120
ALT/AST U/L	74/110
S creatinine	0.06 mg/dL
Urea	15.3 mg/dL
Sodium	131 mmol/L
Potassium	4.3 mmol/L
Prothrombin time	14 sec/INR 1

TLC— Total leucocyte count, DLC— Differential LC %, ALT— Alanine amino transferase, AST— Aspartate amino transferase, K— Thousand

On *tenth* day were ALT - 55 U/L, AST - 143 U/L, Cardiac Marker: CKMB 124 U/ L (18-51), LDH-754 U/L (125–220), S. Amylaze-71 U/L, S. Troponin-I - 4.5 µg/L (0.0–0.01). ECG: ST depression in all leads with sinus tachycardia 150/min (Fig. 1).

He was provisionally diagnosed as Myocardial Ischemia due to myocarditis caused by dengue

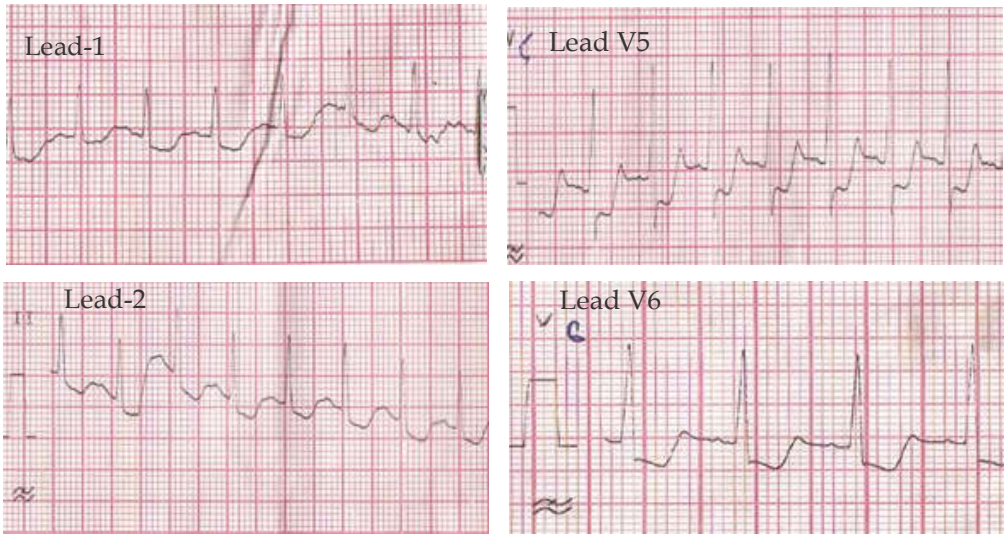

Fig. 1: ECG showing ST depression in all leads with sinus tachycardia.

fever. Chest pain was not relieved by analgesics so inj tramadol was given at 00:30 am. Inj dexamethasone was given as 3 mg/kg loading, followed by 1 mg/kg 8 hourly for myocarditis. Following tramadol chest pain was relieved but he developed respiratory depression, so he was intubated, ventilated and inotropes were started. He was unconscious, on ventilator and with low BP. Within 12 h *Troponin-I* level increased from 4.5 to *17.7* µg/L, however, bleeding had stopped. Further repeat ECG was not obtained. As the triad of severe chest pain, raised biomarker and ECG changes suggested *Acute Coronary syndrome* (ACS); inj. streptokinase was administered as bleeding had stopped and, in ¼ dose of 2000 IU/kg slowly over 15 min.[3] Remaining three doses were given at an interval of 1 hour each. Divided doses were given as bleeding is its side effect. At 9 am, Patient

started improving, with *q* wave in V5 starting to appear, and ST-T changes disappearing (Fig. 2). He recovered from shock and was weaned from ventilator.

Next morning patient was referred to Cardiology center, Ahmedabad (180 km). There he received mechanical ventilator care for 3 days and inj. Methyl Prednisolone (40 mg/kg) for 3 days. 2D ECHO

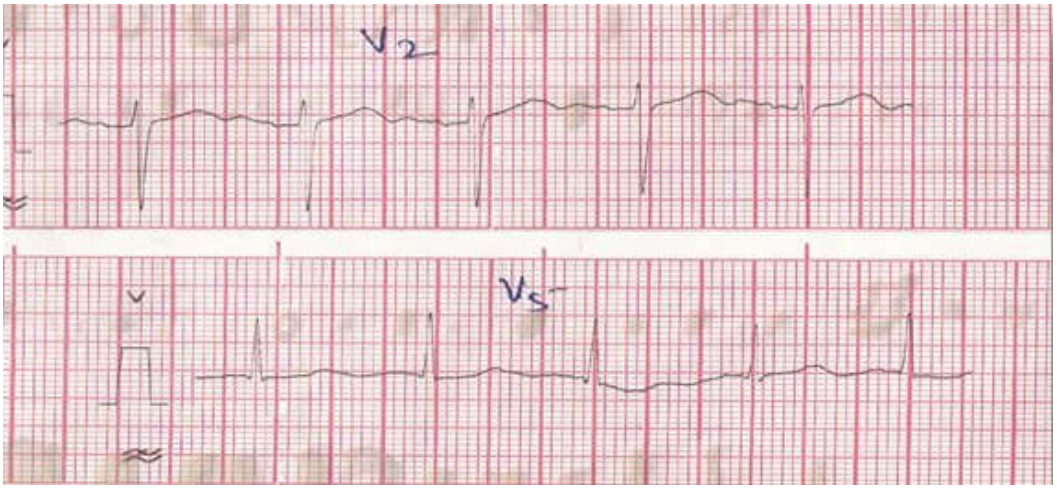

Fig. 2: ECG following 4 divided doses of streptokinase showing disappearance of ST depression.

done for the first time showed *bicuspid aortic valve with valvular insufficiency*, hitherto undiagnosed. He was discharged after five days with advice for valve replacement on follow-up.

Discussion

Myocardial infarction is defined as: Elevated blood levels of cardiac enzymes (CKMB or Troponin T) in typical pattern and one of the following criteria are met: ischemic symptoms, pathological Q waves, ST elevation or depression or coronary intervention (stent).[4]

ACS or acute myocardial ischemia/infarction (MI) is extremely rare in adolescents. Patient characteristics associated with ACS include substance abuse, tobacco use, and male sex. The hospital survival for ACS in adolescents is excellent, and the need for catheter or surgical coronary artery intervention is uncommon.[1]

Bicuspid aortic valve is a congenital cardiac anomaly, having an incidence 0.9 to 2.0% and a frequency of 54% in valvular aortic stenosis after 15 years.[5] Association of bicuspid aortic valve is with aortic stenosis, regurgitation, dissection; and infective endocarditis. Left coronary artery is dominant (29–57%) and in 90% it is <5 mm in length, leading to insufficiency.[6,7]

Towbin reported incidence of MI in children from retrospective data from 1954 to 1986. Over 1,00,000 ECGs on 54,605 patients were obtained. Pediatric admissions were 1,013,210 and 31,305 were referred to cardiology. 72 had ECG consistent with MI. These included: anomalous left coronary artery (20%), Kawasaki disease (14%), myocarditis (13%), neonatal critical aortic stenosis (11%), ventricular tumor (5.5%), dilated cardiomyopathy (4%), pulmonary atresia-intact ventricular septum (4%), birth asphyxia (4%), aortic thrombosis (4%), muscular dystrophy (2.5%), coarctation of aorta (2.5%), rheumatic carditis (2.5%), total anomalous pulmonary venous return TAPVR (2.5%), and chest trauma (2.5%).

The positive ECGs represented 0.13% of ECGs, 0.23% of cardiology referrals and 0.007% of pediatric admissions. There was no case of bicuspid aortic valve in this series.[8]

The initial ECG indicated ST segment depression in all the leads and Q wave appeared after the disappearance of the ST segment curve after treatment with streptokinase. The major initial triaging decision in acute coronary syndrome (ACS) is whether or not percutaneous coronary intervention (PCI) is the primary treatment; current guidelines recommend primary PCI in ST-elevation ACS (STEACS)[9] and initial antithrombotic therapy in non-ST-elevation ACS (NSTEACS).[10] However, electrocardiographic STE and NSTE patterns are not uniquely related to distinctly

different pathophysiological mechanisms.[10] Cardiac troponin-I (appears after 4–6 hours and increases and remains high up to 7–10 days) is a sensitive and specific marker of ACS or MI. In this boy, the initial cardiac troponin-I and CK-MB increased by 400 times and 1700 times, respectively. The congenitally bicuspid valve may function normally and go undetected throughout life, may develop calcification and stenosis or may develop regurgitation or infection. Calcification is the most common cause of isolated aortic stenosis.[11] Aortic regurgitation was reported to be the cause in 1.5 to 3%.[12] It may occur in isolation, usually as a result of prolapse of the larger of the cusps, but also in association with aortic root dilatation, coarctation of the aorta, or infective endocarditis.[13] Along with bicuspid aortic valve, this patient had aortic valve insufficiency. Kabra reported that 17% (7/54) children had decreased Ejection Fraction.[14] Bicuspid aortic valve may be a rare (only one case reported so far) cause of ACS in children especially when it is complicated by aortic valve insufficiency. Sometimes ECG changes can be non-supportive, in such cases typical precordial chest pain with raised myocardial enzymes can help in diagnosis of ACS or MI. As guidelines for ACS are for adult patients, we had difficulty in adopting them for a child with congenital heart disease (CHD).

Limitation: Dengue tests were not done and, portable Echo facility was not available at our building.

Conclusion

- Undiagnosed *bicuspid aortic valve* can present as Acute myocardial ischemia (or infarction) or *Acute coronary syndrome* in an adolescent with Dengue fever.
- Administration of inj. *streptokinase* in divided doses over a period of 4 hours in patient having ACS with history of gastrointestinal bleeding resulted in improvement.
- Portable or POC (point of care) Echocardiography is a necessity in intensive care.

Acknowledgments: We thank the UN Mehta Institute of Cardiology, BJMC, Ahmedabad.

Participation involved informed consent received from the family. All authors declare no conflict of interest and no financial relationship or assistance or interest.

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