

Allergic myocardial infarction (Kounis syndrome) in a child with scorpion sting

Sir,

A 4-year-old boy with an alleged history of scorpion bite on the left finger presented with complaints of restlessness, breathing difficulty, and cough. Examination revealed cold peripheries with low volume pulses. There are blood pressure of 80/58/74 mmHg, saturation of 95% on 5 L oxygen, heart rate of 170/min, respiratory rate of 56/min, bilateral basal crepts with normal heart sounds, and no murmur. Temperature of 99°F was noted. Electrocardiogram (ECG) showed ST elevation with tall T-waves in anteroinferior leads suggestive of myocardial ischemia [Figure 1]. Echocardiography revealed severe left ventricular (LV) dysfunction with ejection fraction of 24% [Figure 2]. Chest X-ray showed pulmonary venous congestion [Figure 3]. Prazosin, dobutamine (10 mcg/kg/min), and furosemide infusion were started. Nitroglycerine was given in view of widespread ST changes. By 5 hours, ECG changes normalized [Figure 4a] with improved LV ejection fraction to 80% [Figure 4b]. Such presentation of acute coronary spasm and autonomic crises is related to mast cells destabilization known as Kounis syndrome (KS) or allergic myocardial infarction, first described by Kounis

and Zavras in 1991.^[1] Many triggers are described ranging from drugs, foreign bodies, insect stings, food, medical conditions, or environmental exposure, most common being antibiotics (27.4%) and insects' bites (23.4%).^[2] Mainly described in adults but pediatric cases have been reported worldwide since its first description in 2009.^[3,4] It resulted from hypersensitivity causing release of vasoactive mediators from mast cells such as histamine, leukotrienes, and serotonin and proteases such as tryptase and chymase, either locally or into the systemic circulation.^[5] There are two types of KS, namely Type I: without coronary artery disease and predisposing factors with or without increase in cardiac enzymes, Type 2: with coronary artery disease, and rarely, Type 3 also described in patients with coronary artery stent thrombosis. Symptomatic KS is always associated with subclinical, clinical, acute, or chronic allergic reactions accompanied by cardiac symptomatology.^[6]

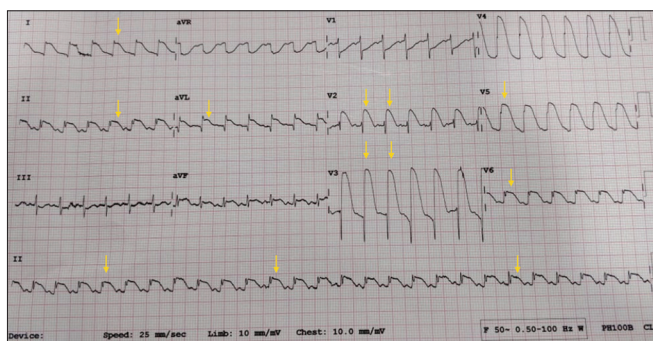


Figure 1: Baseline electrocardiogram on admission showing widespread ST and T changes suggestive of anteroinferior ischemia

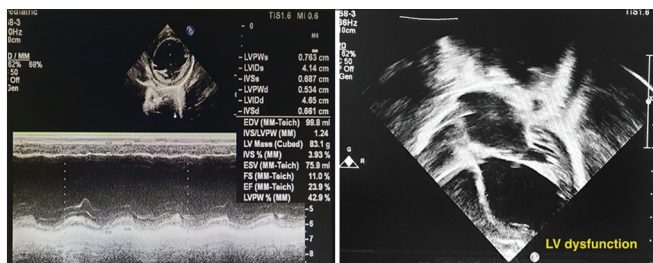


Figure 3: Two-dimensional echocardiogram with M mode showing dilated left ventricular with severe left ventricular dysfunction with ejection fraction of 24%

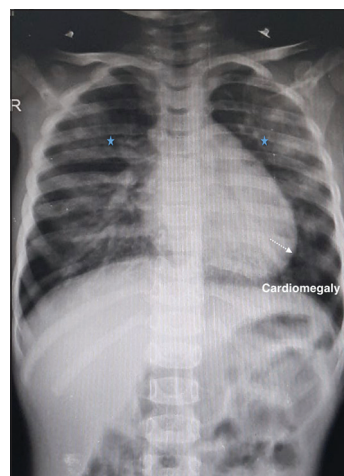


Figure 2: X-ray showed cardiomegaly and pulmonary venous congestion

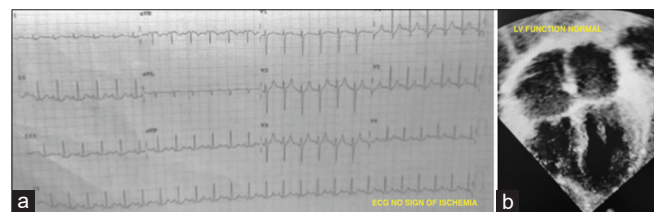


Figure 4: ECG (a) and ECHO (b) after 5 hours of admission showing normalisation of ischemic pattern and LV function

Systemic allergic reactions associated with clinical, electrocardiographic, and laboratory findings of acute myocardial ischemia should be suspected as having KS. Although the most frequent ECG finding is ST-segment elevation in the four anterior and inferior leads, the tracing may be normal or show only nonspecific findings.^[2,5] This entity is variant of stress cardiomyopathy but differentiating both is very challenging. Coronary angiography reveals normal coronaries in Type I KS and stress cardiomyopathy.

Treatment of the allergic event alone can abolish symptoms in Type I KS. The use of intravenous corticosteroids such as hydrocortisone (1–2 mg/kg/day) and antihistamines such as diphenhydramine (1–2 mg/kg) and ranitidine (1 mg/kg) is adequate. The administration of vasodilators such as calcium channel blockers and nitrates can abolish hypersensitivity-induced vasospasm. In patients with Type II variant, treatment should be initiated with an acute coronary event protocol together with corticosteroids and antihistamines. Other measures to stabilize the patient such as pain relief, volume expansion, and oxygen should be taken simultaneously to manage this condition.^[2,5,6]

CONCLUSIONS

Allergic myocardial infarction as a cause for LV dysfunction can be suspected with a history of hypersensitivity or allergic reactions resulting in autonomic storm. It can present in severe form like myocardial dysfunction with significant ECG changes. Most of the cases without underlying coronary artery disease can be managed with nitroglycerin or antihistaminic. Even ECG showing widespread ST changes suggestive of ischemia can be reversible with decrease in autonomic storm.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Sudeep Verma¹, Reena Mathew²,
Khalil Mohammed Khan³, Vejendla Gouthami¹

¹Department of Paediatric Cardiac Sciences, Krishna Institute of Medical Sciences, Secunderabad, Telangana, India, ²Department of Paediatrics, Ankura Hospital, Hyderabad, Telangana, India, ³Department of Paediatrics, Ankura Hospital, Telangana, India
E-mail: drsudeep.verma@gmail.com

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