CASE REPORT

Kounis syndrome following solenopsis (fire ant) bite

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SUMMARY

Anaphylaxis is rarely associated with the vasospastic acute coronary syndrome with or without the presence of underlying coronary artery disease. We report here a case of Kounis syndrome in a man with no known cardiovascular risk developed acute ST-elevation myocardial infarction complicated with complete heart block following Solenopsis (fire ant) bite.

CASE REPORT

A 52-year-old man with no known cardiovascular risk factors sustained a fire ant (possibly *Solenopsis* Invicta) bite on his left hand and was admitted to the General Hospital of Melaka, Malaysia on the 24 November 2017. He initially presented to the emergency and trauma department (ETD) with the complaints of foreign body sensation in his throat occurring 6 hours after being bitten by the fire ant. The assessment in the ETD revealed blood pressure (BP) of 146/84mmHg, regular heart rate (HR) at 70bpm, oxygen saturation of 98% on ambient air with clear lung fields on auscultation. He was subsequently discharged from the ETD.

He presented again to the ETD three hours later with diaphoresis and syncope. This time physical examination revealed a Glasgow Coma Scale of 14/15, borderline BP of 108/62mmHg, regular HR of 74bpm. Respiratory examination revealed expiratory rhonchi and an injected throat. An electrocardiogram (ECG) showed sinus rhythm and right bundle branch block (RBBB). He was admitted to the medical ward. However, sixteen hours after the ant bite, he suddenly developed severe hypotension (BP of 60/42mmHg) with a HR of 22bpm. The ECG revealed complete heart block with ST-segment elevation of 1mm in leads III, aVF, and reciprocal changes in the anterolateral leads. An immediate serum Troponin I was elevated at 1.83ng/mL. Echocardiogram showed inferior wall hypokinesia with the LVEF of 50%. In light of this clinical presentation and ECG changes, Kounis syndrome was suspected.

The patient required temporary inotropic support. Intravenous hydrocortisone and diphenhydramine were administered, followed by the standard thrombolytic dose of intravenous streptokinase and dual-antiplatelet of Aspirin and Clopidogrel. He was successfully thrombolysed with the ECG returning to normal sinus rhythm 60 minutes post thrombolysis.

Subsequently, he was referred to the central cardiac centre for a coronary angiogram. A coronary angiogram revealed 80-90% occlusion of the distal right coronary artery. His left main stem artery was smooth, the left anterior descending artery with proximal 40-50% occlusion and the left circumflex artery showed distal subtotal occlusion. Balloon inflation at the RCA and stenting with a drug eluting stent allowed restoration of blood flow (TIMI flow 3). The final angiography result was favourable, and he was put on one year of dual-antiplatelet therapy.

DISCUSSION

Kounis syndrome (KS) manifests as the concurrence of an acute coronary syndrome during an allergic activation process which includes the spectrum of allergic, hypersensitivity, anaphylactic and anaphylactoid reactions. This allergic angina is named after a Greek physician, Dr. Nicholas Kounis, who in 1991 was the first to report a case of coronary spasm progressing to allergic acute myocardial infarction.[1] There is little known about KS, and most of the available data comes from the clinical case report. KS commonly affects the people in the age group of 40-70 years old.[2] The commonest triggers of the KS are antibiotics followed by venomous insect bites.[2]

Furthermore, KS also has been linked with allergy diseases (bronchial asthma, urticaria) and drugs that are commonly used on routine clinical practice such as antibiotics, aspirin, morphine, non-steroidal inflammatory drugs, contrast media and intravenous anaesthetics.[3] Drug-induced KS are mainly associated with the production of inflammatory mediators including histamine, tryptase, chymase and arachidonic acid that cause coronary artery spasm.

The pathophysiology of KS involves an interaction of mast cells with macrophages and T-lymphocytes that induces mast cell degranulation with subsequent release of the vasoactive mediators (histamine, leukotrienes, serotonin) and proteases (tryptase, chymase).[4] Histamine and leukotrienes are potent coronary vasoconstrictors, while tryptase and chymase may induce collagen degradation and erosion of the atheromatous plaque. Furthermore, histamine can also induce hypotension, molecular adhesion, platelet aggregation and thrombin formation.

Three variants KS have been described. Type 1 KS includes response in those patients with normal or near normal coronary arteries in whom acute allergic reaction induces coronary artery spasm, while the cardiac enzymes may either normal or reflect progression towards acute MI. Type 2 KS

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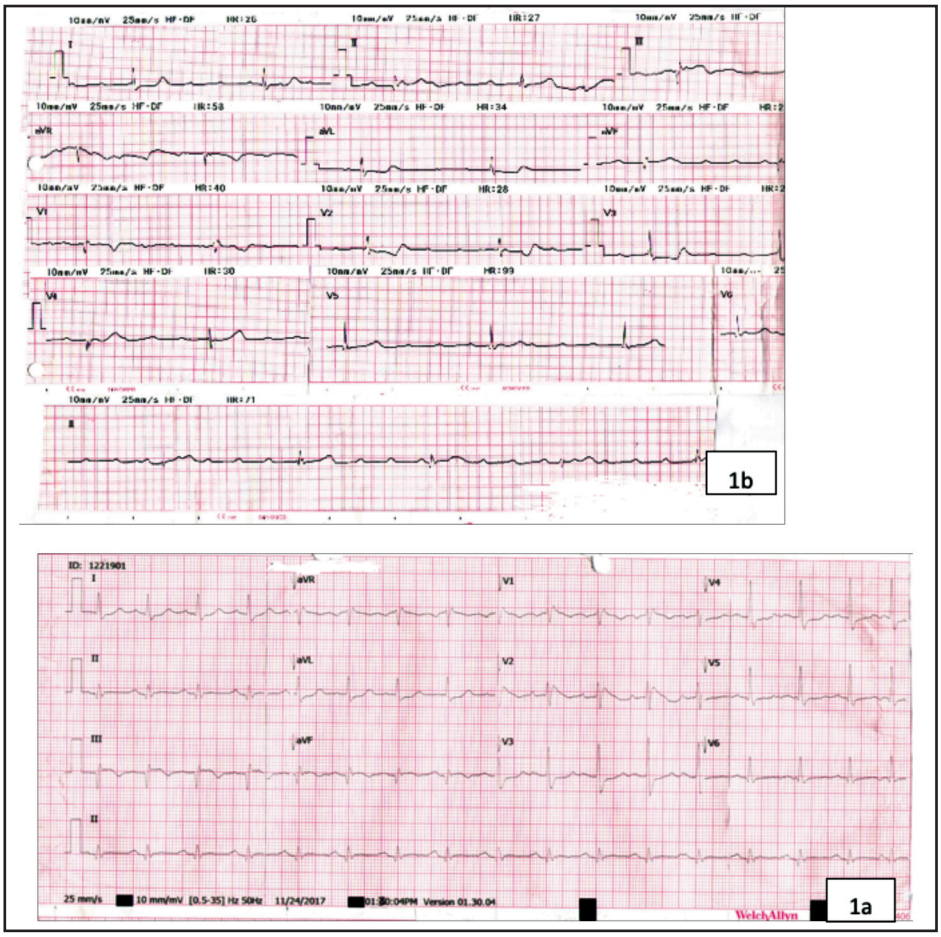

Fig. 1: a) ECG showing complete heart block with ST-segment elevation in inferior leads III, aVF and reciprocal changes in anterolateral leads. b) ECG 1 hour post thrombolysis showing restoration of sinus rhythm with Q wave and T wave inversion in inferior leads III and aVF.

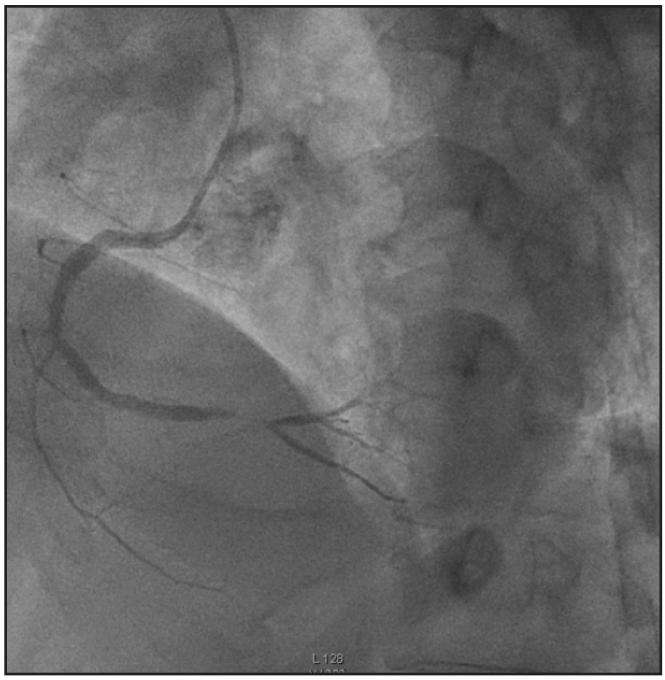

Fig. 2: Coronary angiogram revealed 80-90% occlusion of the distal right coronary artery.

include response in those patients with quiescent pre-existing atheroma plaque where an acute allergic reaction induces coronary artery spasm and atheroma plaque erosion or rupture. Type 3 KS include mast cells and eosinophilic infiltration into the pre-existing coronary stent leading to in-stent thrombosis. As for our patient, he was classified as type 2 variant of KS.

This case is undoubtedly a diagnostic challenge as there is no pre-existing confirmatory test to diagnose KS. The diagnosis requires high index of suspicion which entails all clinical symptoms and signs of allergic reaction, electrocardiographic, echocardiographic, angiographic and laboratory evidence in a holistic approach. The measuring of serum tryptase, histamine, and cardiac enzymes are helpful in KS. The newer method such as thallium-201 single proton emission computed tomography (SPECT), 125I-15-(p-iodophenyl)-3-(R,S) methylpentadecanoic acid (BMIPP) SPECT are reliable tools for assessing cardiac involvement in KS.[5]

At present, there is no specific consensus or guideline pertaining to KS. The therapeutic management of KS is based on the variants of KS. The treatment principles include corticosteroids, antihistamines, a vasodilator such as calcium

channel blockers, acute coronary event protocol, and cardiac revascularization.

KS is not uncommon, but it is infrequently reported in the literature and unrecognized or undiagnosed in clinical practice. In conclusion, more study or research needs to be done to ascertain whether KS represents a distinct clinical entity or an extreme expression of conventional coronary vasospasm.

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