Healthy Heart

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Honorary Editor :
Dr. Vineet Sankhla

From the Desk of Hon. Editor:

The association between acute coronary events and acute allergic reactions has been recognized for several years. The first reported case occurred in 1950, during an allergic reaction to penicillin. In 1991, Kounis and Zavras described the syndrome of allergic angina and allergic myocardial infarction, currently known as Kounis syndrome. Two subtypes have been described: type I, which occurs in patients without predisposing factors for coronary artery disease and is caused by coronary artery spasm, and type II, which occurs in patients with angiographic evidence of coronary disease when the allergic events induce plaque erosion or rupture. This syndrome has been reported in association with a variety of medical conditions, environmental exposures, and medication exposures. Entities such as Takotsubo cardiomyopathy, drug-eluted stent thrombosis, and coronary allograft vasculopathy appear to be associated with this syndrome. In this issue, we discuss a case report, pathobiology, clinical features, associated entities, and management of Kounis syndrome.

- Dr. Vineet Sankhla

Allergic angina following wasp sting: Kounis syndrome

In 1991, Kounis and Zavras described the syndrome of allergic angina and allergic myocardial infarction, currently known as Kounis syndrome (KS). KS is the concurrence of acute coronary syndromes with conditions associated with mast cell activation, such as allergies or hypersensitivity and anaphylactic or anaphylactoid insults. The classical symptoms include acute onset of breathlessness, palpitations, diaphoresis and chest tightness following an acute allergic insult. We report a 50-year-male patient who presented with acute ST elevation myocardial infarction following a wasp sting.

Case Report

A 50-year-old man with no cardiovascular risk factors sustained a wasp sting. He was bitten by 10-15 wasps over his body. Fifteen minutes later, he experienced sudden shortness of breath, feeling of instability, palpitations, chest tightness and excessive sweating. On admission at our institute, patient was hypotensive (80/60 mmHg) with an arterial oxygen saturation of 85%. He was given 1 mg of subcutaneous adrenaline (1 : 1000), 100 mg of intravenous hydrocortisone, lamp of Inj Avil & lamp of Inj Ranitidine. The electrocardiogram revealed ST elevation in V1-V4 suggestive of acute AWMI. (Fig. 1). Echocardiogram showed anterior wall hypokinesia with an LVEF of 40%. Following administration of adrenaline and corticosteroids, the patient became relatively hemodynamically stable. Coronary angiogram (Fig. 2) revealed acute proximal LAD thrombotic occlusion. He underwent successful PCI to LAD with good result (Fig. 3). He was continued for hydrocortisone for 2-3 days apart from usual anti-ischemic and anti-platelet therapy. He was discharged in stable condition after 4 days.

Discussion

KS was first described in 1991 by Kounis and Zavras [1] as 'the coincidental occurrence of chest pain and allergic reactions accompanied by clinical and laboratory findings of classic angina

pectoris caused by inflammatory mediators released during the allergic insult'. They called the progression from chest pain to acute myocardial infarction 'allergic myocardial infarction' [2, 3]. This occurs during episodes of anaphylaxis, and frequently in patients with prior coronary disease, although it has also been observed in patients with healthy coronary vessels. The main mechanism implicated is vasospasm of the coronary arteries.

Three variants of syndrome have been described:

i Type I variant—This applies to patients with normal or nearly normal coronary arteries without predisposing factors for coronary artery disease. The acute mediator release can induce either coronary artery spasm or coronary artery spasm progressing to acute myocardial infarction.

ii Type II variant—This applies to patients with culprit, quiescent pre-existing atheromatous disease, and the mediator release can induce either coronary artery spasm or coronary artery spasm together with plaque erosion or rupture manifesting as acute myocardial infarction.

iii Type III variant is seen in patients with stent thrombosis in whom thrombus harvested and stained with hematoxylin–eosin and Giemsa shows eosinophils and mast cells, respectively. Type III variant is diagnosed in patients with stent implantation who died suddenly and histological examination of coronary intima, media or adventitia adjacent to the stent is infiltrated by eosinophils and/or mast cells.

Following an allergic insult, arachidonic acid cascade is activated; histamine is released leading onto a series of functional and metabolic changes in the heart, termed as cardiac anaphylaxis. Authors have also described the role of renin and adrenaline released during anaphylaxis in causing cardiac dysfunction. Elevated serum tryptase will aid in differentiating KS from routine forms of acute coronary syndrome, provided that the blood sample is drawn between the first and second hour after onset of symptoms.

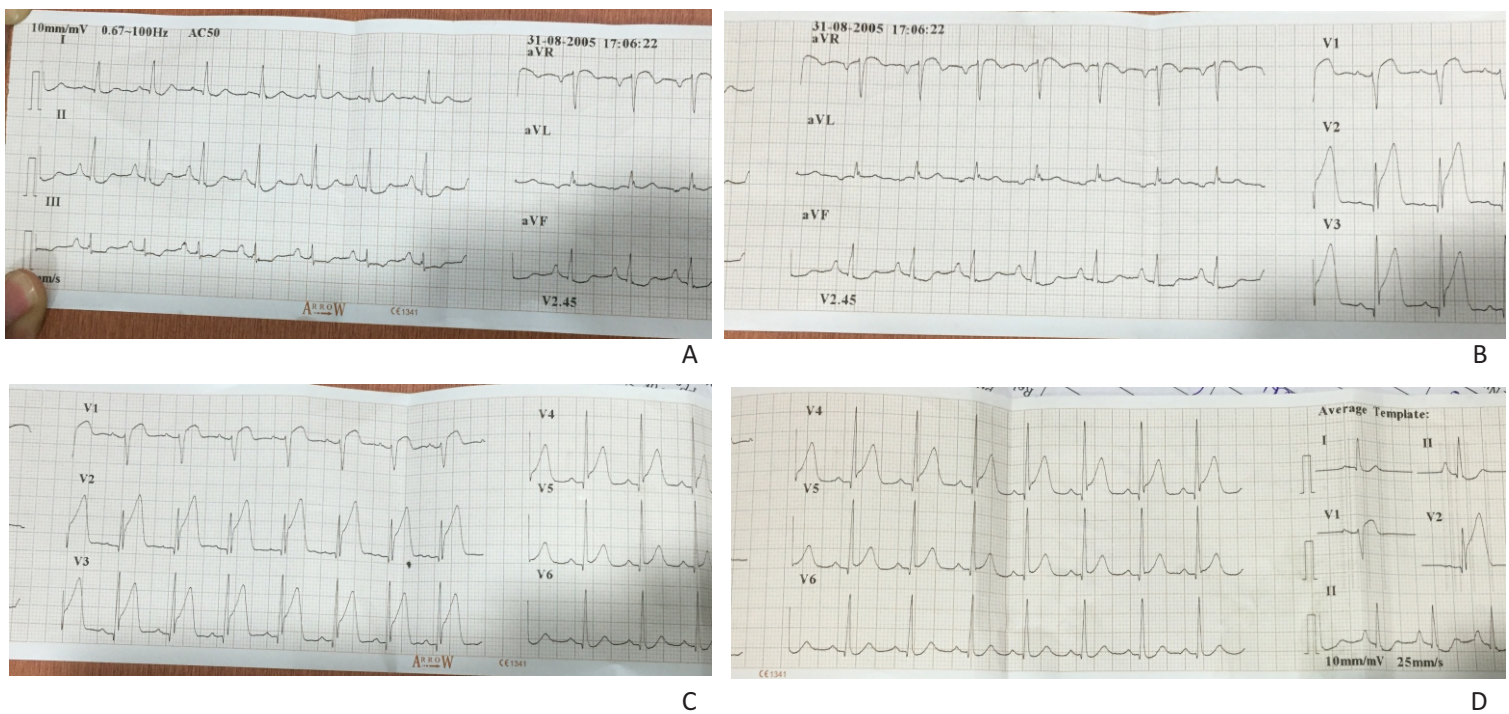

Fig. 1 : The 12 Lead ECG showing Acute AWMI on presentation.

Executive Summary

Pathophysiology

The human heart seems to be the primary target of anaphylaxis because minutes after the insult, left ventricular end diastolic pressure increases significantly indicating pump failure, cardiac output decreases significantly and blood pressure decreases after an initial increase. Concurrently, electrocardiographic signs of acute myocardial ischemia become evident.

Causality

Myocardial ischemia and myocardial infarction occurring during an allergic episode manifesting as Kounis syndrome, does not seem to be the result of coronary hypoperfusion due to systemic vasodilation, reduced venous return, leakage of plasma and volume loss from increased vascular permeability and depression of cardiac output.

Clinical Manifestation

In any case of acute coronary syndrome, careful patient interrogation for atopy, allergies and hypersensitivities may reveal a mechanism involving mast cell activation preceding the event. Routine cardiac evaluation for myocardial injury biomarkers such as cardiac enzymes and troponins, together with tryptase and if possible histamine levels should be

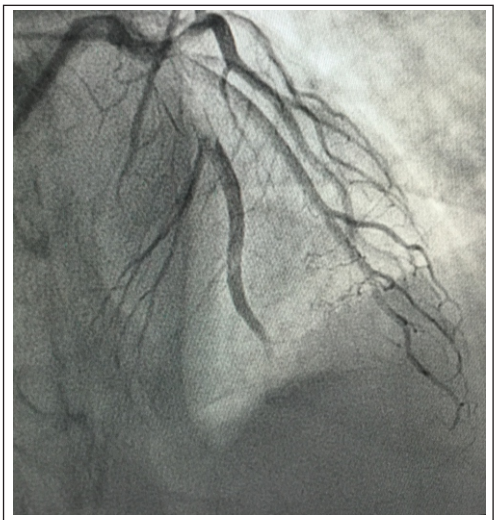

Fig. 2: CAG showing thrombus in Proximal LAD

undertaken in any patient with any grading of allergic reaction.

Anaesthesia & Kounis Syndrome

In anaesthesia, many agents are involved including neuromuscular blocking drugs, antibiotics, latex, contrast media, hypnotic agents, opioids, colloids, apronitin, protamine, chlorhexidine, dyes, local anaesthetics and blood transfusions which can cause direct or immunoglobin mediated mast cell degranulation. It appears likely that the more drugs and agents an anaesthetized patient is exposed to, the easier and quicker the mast cell degranulation occurs.

Kounis Syndrome & Hypersensitivity Myocarditis

In hypersensitivity myocarditis there is presence of eosinophils, atypical lymphocytes, and giant cells in myocardial biopsy but the coronary angiogram is normal. In Kounis syndrome, myocardial biopsy is typically normal but coronary angiogram, especially in type II variant, shows coronary artery disease with coronary stenosis. In eosinophilic arteritis, vasospastic angina, myocardial infarction, coronary artery dissection and sudden death appears to share the same pathophysiology with Kounis syndrome.

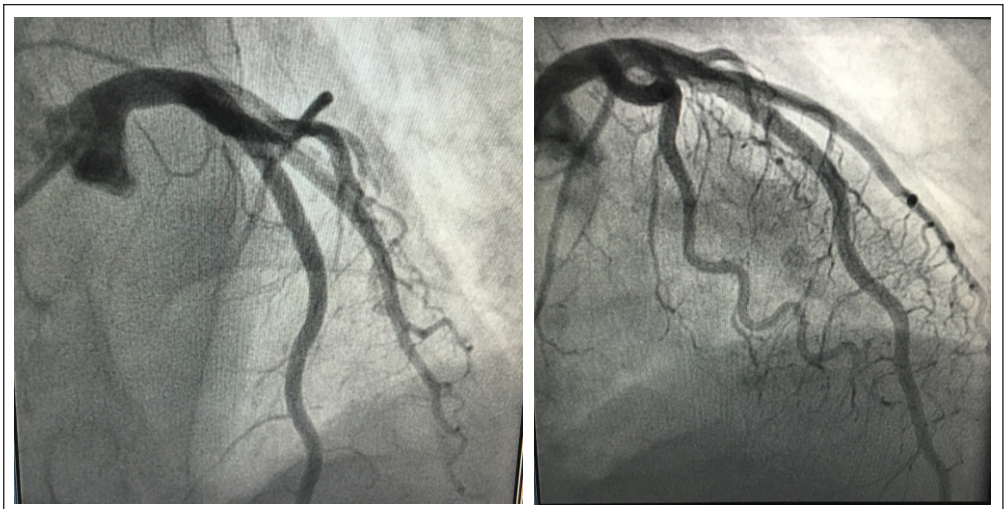

Fig. 3 : Successful PCI to LAD with DES

The Brain, the Heart & Kounis Syndrome

The existing pathway between the brain and heart culminating in mast cell activation and the development of Kounis syndrome includes: depressogenic stress impulses arising from high cortical centers, hypothalamus, paraventricular nucleus to release norepinephrine, serotonin, acetylcholine and corticotropin-releasing hormone. The activation of the sympathetic system centrally is also transmitted peripherally to the adrenal medulla and increases the amounts of epinephrine. Adrenocorticotropic hormone stimulates the adrenal cortex to produce corticosteroids. The sympathetic innervation of kidney contributes in involvement of the rennin–angiotensin system in this cascade of actions, stimulations, secretions and interactions, which can lead to the development of Kounis syndrome.

Takotsubo & Kounis Syndrome

Anaphylactic reactions can also induce Takotsubo syndrome, therefore the measurement of inflammatory mediators such as histamine, neutral proteases and arachidonic acid products, or the use of corticosterpoids or mast cell stabilizers for prevention and treatment may shed light on the etiology and pathophysiology of Takotsubo syndrome.

Stent Thrombosis & Kounis Syndrome

Stent components include the metal strut made from stainless steel containing nickel, chromium, manganese, titanium and molybdenum, the polymer coating and the eluted drugs. The stented patients also take aspirin and clopidogrel and are exposed to the environment. These six elements act as antigens and can induce intracoronary mast cell degranulation culminating in Kounis syndrome and stent thrombosis. Information sheets inside the commercial stent packages state this and warn about the possibility of Kounis syndrome.

Therapeutic Points

In Kounis syndrome, nitroglycerin can worsen hypotension, β-blockade can exaggerate coronary spasm, epinephrine can aggravate ischemia, worsen coronary vasospasm, promote platelet aggregation, increase thromboxane B2 production and due to its sulfite content, can induce anaphylaxis. Therefore, sulfite free epinephrine is preferable. Opiates can induce massive mast cell degranulation and aggravate allergic reaction, whereas fentanyl and its derivatives show slight mast cell activation and should be the drugs of choice when narcotic analgesia is necessary.

CONCLUSION

Kounis syndrome is not a rare disease but an infrequently diagnosed condition.It should be borne in mind when diagnosing patients with no cardiovascular risk factors who experience acute coronary syndrome accompanied by symptoms of anaphylaxis

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5. Kounis Syndrome. Pablo R. Lopez, MD; Alan N. Peiris, South Med J. 2010;103(11):1148-1155.

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#dildilkikahani

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Age: *27 years*
Location: *Jodhpur, India*

** name changed*

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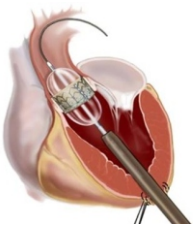
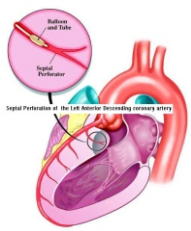
Transcatheter Aortic Valve Replacement TAVR (TAVI)

A minimally invasive surgical procedure to repair the valve without removing the old, damaged valve



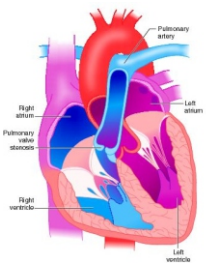
Percutaneous Transluminal Septal Myocardial Ablation (PTSMA)

A minimally invasive procedure (injecting absolute alcohol into the septal branch)to reduce the overgrowth of heart muscle



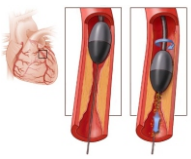
Structural Interventions & Adult Congenital Intervention

Balloon mitral / aortic / pulmonary valvuloplasty. A procedure to dilate abnormal valve by balloon. Non-surgical closure of para-valve leaks



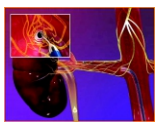
Rotablation

Rotablation uses a tiny drill, powered by compressed air, to remove calcified deposits in the arteries. Since 1990 CIMS Cardiology has one of the highest experiences in using Rotablator in hundreds of case



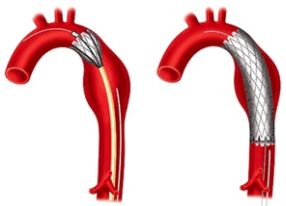
Renal Denervation

Renal denervation system uses a technique called Renal Denervation (RDN) to selectively calm hyperactive renal nerves. This causes a reduction in the kidneys' production of hormones that raises blood pressure and protects the heart, kidneys and blood vessels from further damage.



TEVAR Thoracic Endovascular Aneurysm Repair

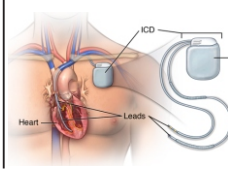
A non surgical treatment for the abnormal bulge of the arterial wall / aortic dissection



Electrophysiology

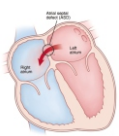
Carto-3 System
An advanced 3-dimentional mapping & imaging technology for accurate visualization of multiple catheters in a patient's heart and pinpointing the exact location of arrhythmogenic focus

Devices
- AICD
- CRT
- Pacemaker



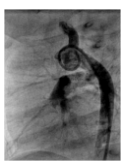
Atrial Septal Defect (ASD), Ventricular Septal Defect (VSD) & Patent Ductus Arteriosus (PDA)

A procedure wherein an umbrella device is inserted in to the hole in the septum, or muscular walls of the upper chambers (ASD) & lower chambers (VSD) of the heart to close it permanently. A procedure to control the extra flow of blood from the main artery of the body to the artery of the lungs



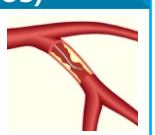
Complex Congenital Pediatric Intervention

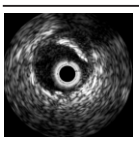
This includes, hybrid procedure for VSD closure, duct stenting or RVOT stenting in cyanotic neonates. They are effective options to surgery in small neonates / infants.



Fractional Flow Reserve (FFR) & Intravascular Ultrasound (IVUS)

This is a guide wire-based procedure to accurately measure blood pressure and flow through a specific part of the coronary artery to assess need for angioplasty / stent





IVUS allows us to see a coronary artery from the inside-out generating real time images, such as coronary angiography, performed in the cath lab. CIMS has been deploying the use of IVUS since many years including virtual histology.

MRI - Signa Explorer & CT Scan - Revolution EVO 128 slice coming to CIMS shortly (next month).

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Pediatric Master Classes - Basic EEG Course for Practicing Pediatricians - Certification Course "Making Pediatric EEG - Simpler"

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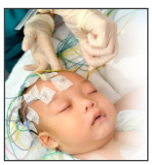
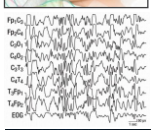
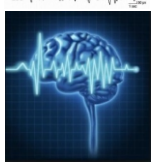
Course Coordinator : Dr. Amit Chitaliya / Dr. Snehal Patel
Date : April 24, 2016 (Sunday)
Venue : CIMS Auditorium

Program Overview: The goal is to bring awareness about this thought to be a difficult tool and to clear diagnostic dilemma for practicing pediatricians who are sitting in periphery and could be of a great help in treating children with epilepsy.







Program Highlights:

- The course will cover the basic elements of the practice of EEG in its application to the diagnostic work up and the management of children with suspected or already established epilepsy.
- It will be practice oriented and aimed at the general paediatrician dealing with - but not exclusively involved in - epilepsy care.
- Subject material will emphasize the basic role of the (standard) EEG in the diagnosis and management of epilepsy, common misconceptions (positive and negative) and potential pitfalls.
- Attention will be given to practical aspects, including electrodes, montages, provocation methods and the optimisation of EEG requests and reports, specifically in the context of epilepsy.
- Clinical aspects will include definitions of spike, spike and wave, sharp wave, etc., examples of various types of epileptiform activity, sensitivity, specificity and predictive value of specific EA patterns for diagnosis of epilepsy or specific epilepsy syndromes.

Online registration & payment on www.cims.me/clc

Registration Fees	: ₹ 1,000/- (Up to one month before course date)
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