PP 49
A BEE-ZY COMPARTMENT SYNDROME

Mohd Rizwan M[1], Nur A K[2]
[1]Hospital Sungai Buloh, Sungai Buloh, Selangor, Malaysia
[2]Faculty of Medicine, UiTM Sungai Buloh, Selangor, Malaysia

INTRODUCTION

Bee stings by the genus Apidae give rise to a variety of life threatening conditions due to the systemic effect of amines, peptides and toxins released by the stings. Rarely, local reactions result in compartment syndrome, a dreadful limb threatening complication. We describe a case of left middle finger compartment syndrome after a bee sting; requiring emergency fasciotomy.

CASE REPORT

A 21-year-old man was stung by a honeybee over the palmar surface of his left middle finger 19 hours prior to presentation. He complained of worsening pain and swelling over the bite site. He had a short history of fever following the sting but subsided after antipyretics. Upon examination, his vital signs as well as systemic examinations were unremarkable. Local examination of his left middle finger revealed a grossly swollen, sausage-like, erythematous and tender finger. There was no punctum and the bite site was obscured. Flexion and extension over the interphalangeal joints were severely restricted and the passive stretch test was positive. Peripheral sensation was also affected. X-rays of the finger were normal. Considering the positive clinical findings and time since insult, this patient underwent a fasciotomy to release the compartment pressure. Secondary suturing was done 2 weeks later and he achieved full function in 1 month.

DISCUSSION

Most clinicians' worry about the systemic effects of a bee sting but the wide variety of toxins present in a bee sting may just as well cause dangerous local reactions. The inflammatory reactions triggered by these amines and peptides in an enclosed area such as the finger raise inter-compartmental pressures and jeopardize neurovascular integrity and should not be confused with secondary bacterial infection.

CONCLUSION

Compartment Syndrome is a real and present complication of a bee sting that should be considered especially when bitten over the limbs.

PP 50
DELAYED ONSET HEART FAILURE AFTER DELIBERATE DIHYDROPYRIDINE OVERDOSE

Nur A K[1], Shah Jahan M Y[2]
[1]Faculty of Medicine, UiTM Sungai Buloh, Sungai Buloh, Selangor, Malaysia
[2]Hospital Kuala Lumpur, Kuala Lumpur, Malaysia

INTRODUCTION

Calcium-channel blocker toxicities are rare but account for about 40% of deaths in cardiovascular drug overdoses. Patients commonly present early and urgently with features of haemodynamic instability. We describe a case of a dihydropiridine toxicity that presented late and was in profound cardiac failure; assumed to be due to an acute coronary event.

CASE REPORT

A 29-year-old man presented to the ED with headache, vomiting and breathlessness 3 days after ingesting 300mg of Amlodipine besylate, in a para-suicidal attempt. He had gone to a

GP the day before and received intravenous fluids presumably due to a low blood pressure.

The history of deliberate ingestion was not known in the early stages. On arrival, he was tachycardic and mildly tachypnoeic but not hypotensive. Subtle clinical findings of congestive cardiac failure were compounded by a B-profile on lung ultrasound and a raised pro-BNP level. ECG showed sinus tachycardia with evidence of lateral wall ischaemia; the glucometer was 7.1mmol/L and remained normal throughout. His condition worsened with time and he eventually required non-invasive ventilation. Following supportive therapy he was discharged well 10 days later.

DISCUSSION

Amlodipine preferentially block the L-type calcium channels of the vasculature and have minimal effects on cardiac contractility with consequent hypotension and bradycardia. As the degree of toxicity was moderate (300mg), this selectivity was not lost and this patient presented with congestive cardiac failure likely due to being left untreated. The presence of ischaemic ECG changes was a red herring in the diagnosis.

CONCLUSION

Overdosage of any type of cardiac drugs require further investigation and monitoring to ensure delayed and subtle complications like this are not missed.

PP 51
HISTORY SAVED IT ALL, CASE REPORT

A H Mohd Mustamam[1], N Muhammad Farid Wong[1], Y J Lee, N Mohd Ali[1]
[1]Jabatan Kecemasan dan Trauma, Hospital Tengku Ampuan Rahimah, Klang, Selangor, Malaysia

INTRODUCTION

Massive pulmonary embolism is defined as acute pulmonary embolism with sustained hypotension, pulselessnes or bradycardia. The combination of history taking, clinical findings, prediction probability test and bedside echocardiography is of value in diagnosing and treating unstable pulmonary embolism patient.

CASE REPORT

A 48 year old Chinese male with underlying lung carcinoma ongoing currently on chemotherapy, presented to Emergency Department with sudden onset shortness of breath started the morning of arrival. Patient had has been having cough with minimal sputum, denies with no fever, no hemoptysis, no chest pain and no heart failure symptoms. On examination patient was hemodynamically unstable with moderate respiratory distress, other systemic physical examination is was unremarkable. Bedside echocardiography showed right ventricle hypertrophied and dilated with paradoxical septal movement, low end diastolic left ventricle volume and plethoric inferior vena cava. Well's criteria calculated with a score of 3 which is was high possibility of pulmonary embolism. In view that the patient was unstable for CT-PA and with combination of history given and other clinical findings suggested patient was having massive pulmonary embolism. The patient was