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.

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HISTORY SAVED IT ALL, CASE REPORT

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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

thrombolysed with IV Tenecteplase 50mg and subsequently admitted to ICU. Patient showed overall marked improvement. CT-PA done later showed right pulmonary artery embolism.

DISCUSSION AND CONCLUSION

The above case was a classic presentation of massive pulmonary embolism which is marked with persistent hypotension. CT-PA is the gold standard for diagnosing pulmonary embolism but in unstable massive pulmonary embolism it is not possible to be done. In the latest guidelines by European Heart Journal and American College of Physician recommended the use of prediction probability test to proceed with the treatment. In cases of uncertainty and with many differential diagnosis, it is important to combine history taking, clinical findings and other modalities such as prediction probability test and bedside echocardiography to determine diagnosis and management.

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ARISE FROM THE DEAD, CASE REPORT

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INTRODUCTION

Lazarus phenomenon is a rare clinical condition, first reported by Linko et al. in 1982. The pathophysiology is not well understood. Hyperinflation, myocardial stunning, hyperkalaemia, delayed action of drugs, countershock asystole, and unobserved minimal vital signs amongst others have been considered to be the most common mechanisms.

CASE REPORT

40 year old Indian male, presented to emergency department with sudden onset of typical chest pain. Patient developed ventricular fibrillation en route to PCI center. CPR commenced immediately and resuscitation per ACLS guidelines was done. In view of refactory VF, resuscitation continued with IV Vasopressin 40, IV methylprednisolone 40mg and IV esmolol 30mg, despite the additional medications and CPR for 45 minutes, there was no ROSC and cardiac monitor deteriorated to asystole and resuscitative effort discontinued. Patient was extubated and explained to family members regarding poor prognosis. Death was not pronounced to family members in view of the presence of agonal breathing. After 30 minutes cessation of CPR noted patient had good spontaneous respiratory effort and started moving his upper limb and localizing pain, cardiac monitor showed sinus rhythm. Airway was than secured. Patient was than thrombolysed and admitted to ICU.

DISCUSSION AND CONCLUSION

The decision to stop CPR is a challenging clinical task. In general, CPR should continue as long as shockable rhythm or the other reversible cause for cardiac arrest persists. It is widely accepted that asystole for more than 20 minutes without reversible factors is a reasonable cause. The decision to stop is based on resuscitation team judgement, time before initiation of CPR, primary rhythm, comorbidity, and duration of resuscitation.