

# Concurrent subarachnoid haemorrhage and ST elevation myocardial infarction

Joon H Tan, Akhlaque Uddin and Juan P Fernandez

Lincolnshire Heart Centre, Lincoln County Hospital, Lincoln LN2 5QY, UK

Corresponding author: Joon H Tan. Email: joon.tan@nhs.net

## Summary

Rare case of concurrent subarachnoid haemorrhage and ST elevation myocardial infarction, highlighting the importance of detailed history in an emergency.

## Keywords

ischaemic heart disease, resuscitation, neurosurgery, radiology and imaging

## Case history

A previously fit 66-year-old Caucasian man was admitted to hospital following an out of hospital cardiac arrest. He complained of chest pain followed by dizziness and lost consciousness thereafter. He received bystander cardiopulmonary resuscitation for 10 min. Spontaneous circulation returned following a single defibrillation and was found to have ST elevation in leads V1, V2 and V3 with reciprocal changes in leads II and III on the 12 lead electrocardiogram. Primary percutaneous coronary intervention pathway was activated, and he was transferred to the angioplasty centre. Left heart catheter (Figure 1(a)) revealed a thrombotic subtotal occlusion of the mid-left anterior descending artery which was treated with a single 3.0 mm × 32 mm synergy drug eluting stent. This was optimised and post-dilated with a 3.5 mm non-compliant balloon, with excellent angiographic result. Thrombolysis in myocardial infarction grade 3 normal flow was restored. The rest of the coronary arteries were unobstructed.

Aspirin (75 mg a day) and ticagrelor (90 mg twice daily) were administered post-primary percutaneous coronary intervention along with 18 hours of Eptifibatide (Glycoprotein IIb/IIIa receptor blocker). Transthoracic echocardiogram post-primary percutaneous coronary intervention showed moderate impairment of left ventricular systolic function (ejection fraction 35%) and mild-to-moderate aortic regurgitation.

The next day, the patient described severe generalised frontal headache with nausea and photophobia. On further questioning, it transpired that the

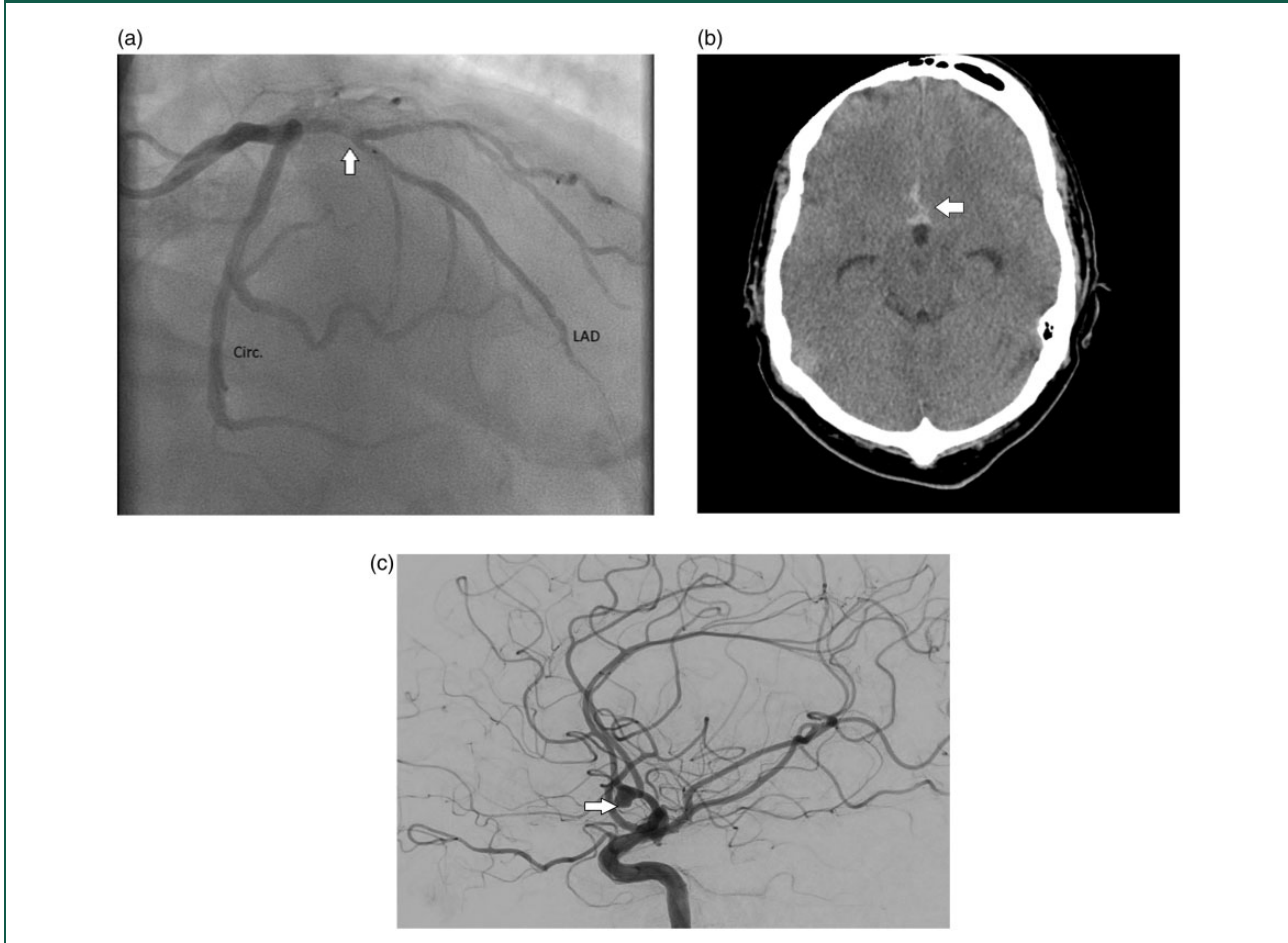
headaches actually preceded the chest pain and cardiac arrest. The full blood count, coagulation, renal and liver function were normal (haemoglobin 132 g/L, white blood cells  $9.3 \times 10^9/L$ , platelets  $229 \times 10^9/L$ , neutrophils  $7 \times 10^9/L$ , international normalised ratio 1.0, sodium 136 mmol/L, potassium 3.6 mmol/L, urea 6.5 mmol/L, creatinine 72  $\mu\text{mol/L}$ , estimated glomerular filtration rate (eGFR) >90, bilirubin 17  $\mu\text{mol/L}$ , alanine transaminase 26 U/L, alkaline phosphatase 72 U/L, albumin 30 g/L). Urgent computer tomography head and computer tomography cerebral angiogram were performed (Figure 1(b) and (c)). Computer tomography brain confirmed the presence of a subarachnoid haemorrhage mainly in the interhemispheric fissure, while computer tomography angiogram revealed a 6 mm aneurysm of the anterior communicating artery. No cerebrospinal fluid sample was obtained for xanthochromia as radiological imaging was significantly diagnostic and any lumbar puncture attempt would pose a significant bleeding risk due to the dual anti-platelet therapy and eptifibatide. Dual antiplatelet therapy was not stopped due to the high risk of stent thrombosis. Coil embolisation of the aneurysm was performed with platelet cover and excellent occlusion of aneurysm was achieved.

Patient made a good gradual recovery and four weeks after initial cardiac arrest was discharged from hospital.

## Discussion

Subarachnoid haemorrhage is commonly associated with electrocardiograph and echocardiographic changes. A patient with SAH may have ST segment changes, T-wave inversion, or prolongation of the corrected QT interval on their electrocardiograph, an elevated troponin level and transient global or segmental hypokinesia of the left ventricle on echocardiogram despite having unobstructed coronaries. The precise mechanism of these electrocardiograph changes is not clear; however, a possible explanation

**Figure 1.** (a) Thrombotic subtotal occlusion of the mid left anterior descending (LAD) artery on coronary arteriogram (right anterior oblique caudal view), (b) Computer tomography head post-percutaneous coronary intervention confirming the presence of a subarachnoid haemorrhage mainly in the interhemispheric fissure (white arrow) and (c) digital subtraction angiography of the brain revealing a 6 mm aneurysm of the anterior communicating artery (white arrow).  
Circ: circumflex artery.



is that catecholamine excess during an subarachnoid haemorrhage causes a neurogenic stunned myocardium, mimicking myocardial infarction.<sup>1</sup>

We present a case which highlights the rare correlation between true ST elevation myocardial infarction and subarachnoid haemorrhage. Only two other cases of subarachnoid haemorrhage with obstructive coronary arteries have been reported and in both cases, the predominant symptom was headaches with meningism.<sup>2</sup> This gentleman had no neurological signs and the predominant symptom was chest pain.

We postulate that the subarachnoid haemorrhage was the initial event, causing secondary plaque rupture in a patient with pre-existing coronary artery disease. Our case supports cardio-cerebral interaction beyond the hypothalamic-pituitary-adrenocortical and sympatho-adrenomedullary axes. Noradrenaline surge from hypothalamus ischaemia and vasospasm

of epicardial vessels are probable hypotheses. Noradrenaline stress response causing real cardiac injury is supported by cases where takotsubo cardiomyopathy is seen following a subarachnoid haemorrhage.<sup>3-5</sup> However, the pathophysiology behind true ST elevation myocardial infarction following subarachnoid haemorrhage has not been established.

The learning point from this case is to continue to obtain a detailed history even in the setting of a primary percutaneous coronary intervention pathway where time critical interventions need to be made. There are other causes of ST elevation such as acute pericarditis, left ventricular aneurysm and takotsubo that a physician needs to consider in the context of cardiac arrest.<sup>6</sup> This case also highlights that bystander cardiopulmonary resuscitation helps to reduce cognitive impairment and increases the chances of survival.

**Declarations**

**Competing interests:** None declared.

**Funding:** None declared.

**Ethics approval:** Written informed consent for publication was obtained from the patient.

**Guarantor:** JPF.

**Contributorship:** All authors contributed equally to the planning of the manuscript, the review of literature and the writing and editing of the original and final manuscript.

**Provenance:** Not commissioned; peer-reviewed by Osama Amin.

**References**

1. Davies KR, Gelb AW, Manninen PH, Boughner DR and Bisnaire D. Cardiac function in aneurysmal subarachnoid haemorrhage: a study of electrocardiographic and echocardiographic abnormalities. *Br J Anaesth* 1991; 67: 58–63.
2. Van der Velden LB, Otterspoor LC, Schultze Kool LJ, Biessels GJ and Verheugt FWA. Acute myocardial infarction complicating subarachnoid haemorrhage. *Neth Heart J* 2009; 17: 284–287.
3. Lyon AR, Rees PS, Prasad S, et al. Stress (Takotsubo) cardiomyopathy – a novel pathophysiological hypothesis to explain catecholamine-induced acute myocardial stunning. *Nat Clin Pract Cardiovasc Med* 2008; 5: 22–29.
4. Kono T, Morita H, Kuroiwa T, et al. Left ventricular wall motion abnormalities in patients with subarachnoid haemorrhage: neurogenic stunned myocardium. *J Am Coll Cardiol* 1994; 24: 636–640.
5. Banki NM, Kopelnik A, Dae MW, et al. Acute neurocardiogenic injury after subarachnoid haemorrhage. *Circulation* 2005; 112: 3314–3319.
6. Deshpande A and Birnbaum Y. ST-segment elevation: distinguishing ST elevation myocardial infarction from ST elevation secondary to non ischemic etiologies. *World J Cardiol* 2014; 6: 1067–1079.