MASSIVE PULMONARY EMBOLUS PRESENTING WITH ABDOMINAL PAIN

Editor,

We present the case of a 20 year old man who presented to the emergency department of Craigavon Hospital with a one day history of abdominal pain and dyspnoea. He had been involved in a motorcycle accident three days previously and sustained a soft tissue injury to his left leg. Examination revealed lower abdominal tenderness and left calf swelling. Blood pressure was 140/53mmHg and oxygen saturations were 97% on room air. ECG showed sinus tachycardia (137 beats per minute) and 2mm upsloping ST segment elevation in leads V1-V4 (figure 1).

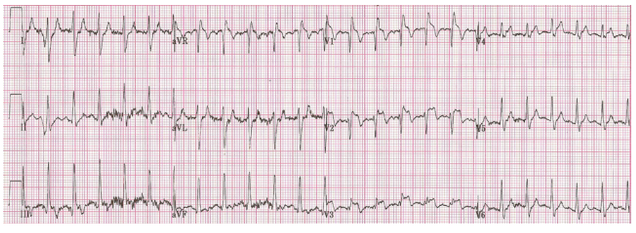

Fig 1.

Ten minutes after arrival, he had an asystolic arrest. Cardiopulmonary resuscitation was commenced, 10 units of intravenous reteplase were administered and he transferred to the cardiac catheterisation laboratory. Myocardial infarction was thought unlikely, thus we proceeded first to pulmonary angiography which showed a large filling defect in the main pulmonary artery extending into left and right branches consistent with a saddle embolism (figure 2). Catheter manipulation and direct intra-embolus injection of further reteplase achieved slight clot fragmentation into smaller sub-branches, but no significant return of pulmonary artery flow or systemic circulation. The resuscitation attempt was discontinued after 90 minutes. Autopsy confirmed a left leg deep venous thrombosis, a saddle-type pulmonary embolism and normal coronary arteries.

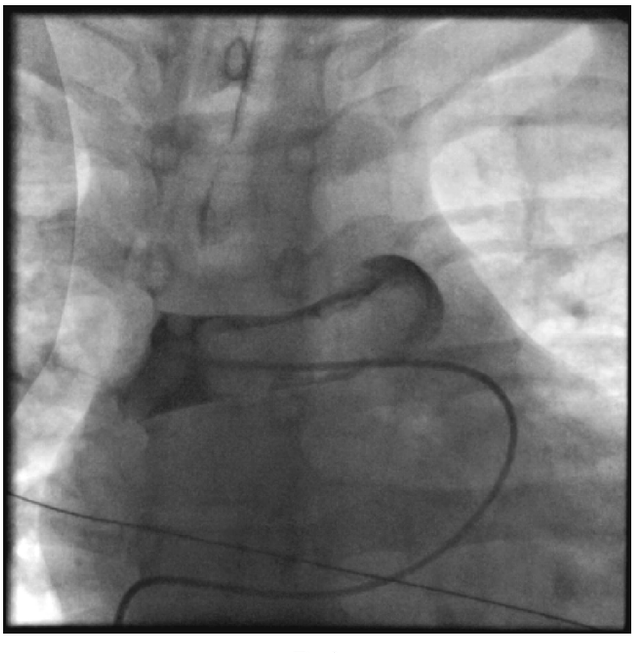

Fig 2.

This case highlights the often atypical presentation of pulmonary embolism[1,2], the feasibility and value of early invasive pulmonary angiography even during cardiac arrest, but also the need for ongoing development of percutaneous techniques/devices for effective large-clot fragmentation or removal.

The authors have no conflict of interest.

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STREAMLINING THE USE OF IHC IN IDENTIFYING GERMLINE MISMATCH REPAIR MUTATIONS IN LYNCH SYNDROME.

Editor,

Colorectal cancer (CRC) is the second most common cause of cancer-related death[1]. Inherited genetic factors are significant in <30% of cases. In ~5% of all cases[2], CRC is associated with a highly penetrant dominant or recessive inherited syndrome. The most common of these is Lynch syndrome (hereditary non-polyposis colorectal cancer, HNPCC), an autosomal dominant cancer susceptibility syndrome caused by a germline mutation in one of the DNA mismatch repair (MMR) genes, namely MLH1, MSH2, MSH6 or PMS2. Affected individuals have a predisposition to developing early onset CRC and a range of other cancers, particularly endometrial in females. The associated lifetime cancer risk is 75%[2]. Early diagnosis enables at risk family members to be enrolled in appropriate cancer surveillance programmes, thus reducing mortality and morbidity. Additionally, recent studies have indicated a role for aspirin in reducing Lynch syndrome tumours[3].

MMR defect leads to instability in microsatellites of tumour DNA. This feature can be found in >90% of colon cancers associated with Lynch syndrome, compared to ~ 15% of cases of sporadic CRC[2]. Using immunohistochemistry (IHC), tumour analysis with antibodies against the four MMR proteins demonstrates loss of protein expression of the causative gene. This investigation thereby provides early, valuable identification of possible HNPCC-related tumours. It furthermore directs germline mutation screening to the gene involved, significantly reducing the time and cost involved in