

Left Ventricular Mural Thrombus Treated With Dabigatran

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Abstract

Novel oral anticoagulants may be effective and safe alternatives to conventional treatment for left ventricular (LV) mural thrombus. A case of left ventricular mural thrombus successfully treated with dabigatran etixelate is described, and the rationale for undertaking further systematic evaluation of novel anticoagulants for this indication discussed.

Keywords: Mural thrombus; Dabigatran; Subarachnoid haemorrhage; Coronary artery dissection; Anticoagulation

Introduction

In patients with left ventricular (LV) mural thrombus the novel oral anticoagulants, which include the direct thrombin inhibitor dabigatran etixelate, may be effective and safe alternatives to conventional treatment with heparin and a vitamin K antagonist. However their use for this indication has not been reported. We describe a case and discuss the rationale for undertaking further systematic evaluation of novel anticoagulants for this indication.

Case Report

A previously well 65 year old woman collapsed while sweeping the floor, hitting her head with brief loss of consciousness. She gave no history of chest pain or other symptoms either before or after the blackout. Full blood-count, creatinine, electrolytes, serial troponins and electrocardiograms (ECG) were all normal. A computed tomography (CT) brain scan demonstrated a traumatic subarachnoid haemorrhage (SAH) in the left precentral sulcus (Figure 1). After 24 hours observation she was discharged, but 4 days later she re-presented complaining of palpitations. Admission ECG demonstrated sinus rhythm and new 1-2 mm ST elevation and then T wave inversion in the anterior leads. Serial measurement of troponin T demonstrated a rise and fall (152, 718, 289 ng/L, 99% for normal 14ng/L) consistent with an acute coronary syndrome (ACS).

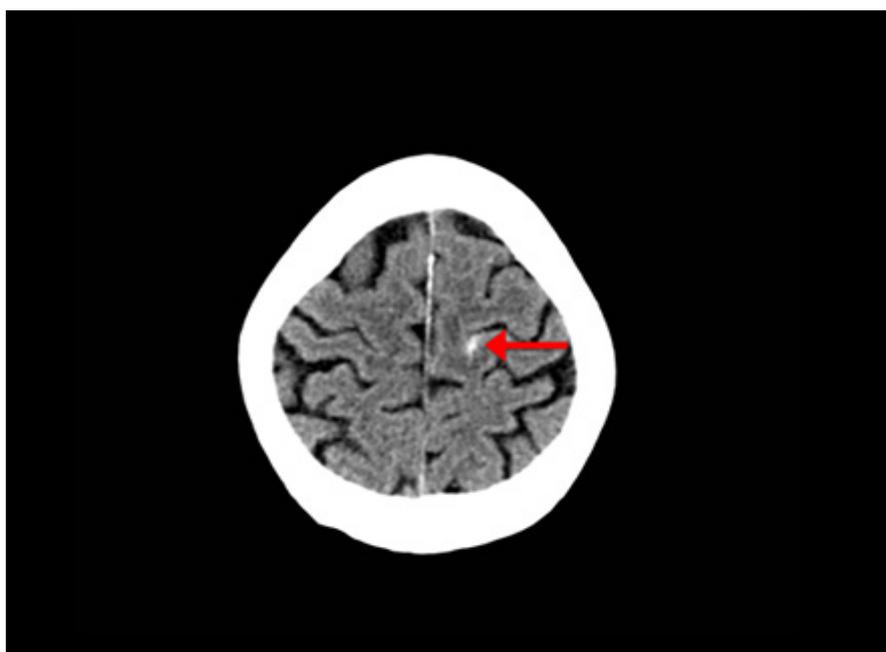


Figure 1: Computed tomography showing a subarachnoid haemorrhage in the left precentral sulcus, (arrow).

Because of the recent SAH and stable clinical status she was not treated with anticoagulant or antiplatelet drugs, and coronary angiography was postponed. On day 7 repeat CT head scan showed resolution of the SAH, and coronary angiography identified spontaneous dissection of the mid and distal left anterior descending coronary artery, with good blood flow to the distal vessel (Figure 2). Left ventriculography demonstrated antero-apical akinesia, and a 1.4 x 1.0 cm apical mural thrombus. The mural thrombus was confirmed by echocardiography (Figure 3).

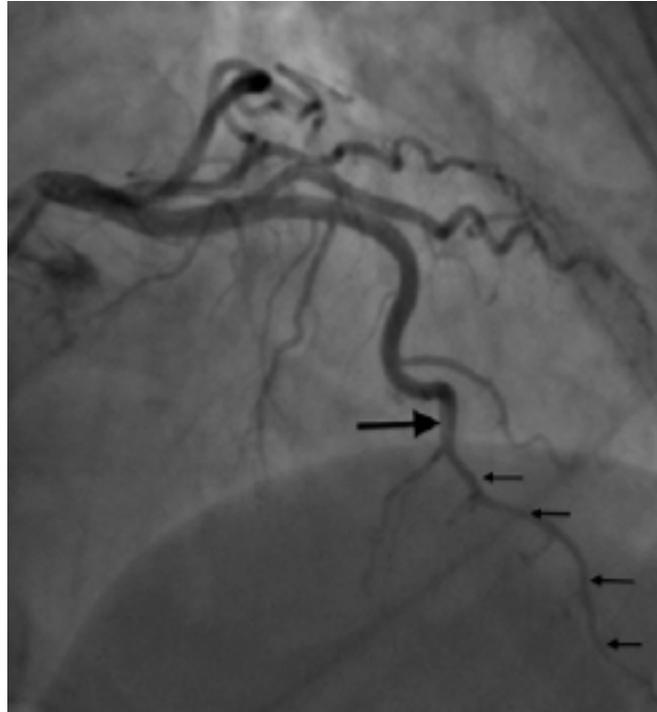


Figure 2: Coronary angiography demonstrating a linear lucency in the mid left anterior descending coronary artery (thick arrow) and long segment of the more distal vessel with decreased lumen diameter but good flow (small arrows), consistent with spontaneous coronary artery dissection. There was no evidence for atherosclerotic disease in other vessels.



Figure 3: Two dimensional echocardiogram showing left ventricular antero-apical akinesia and the apical mural thrombus (large arrow), which resolved completely after 30 days treatment with dabigatran.

Because of the recent SAH she was anticoagulated with dabigatran etexilate 150mg bd rather than warfarin. Neither low molecular weight heparin nor antiplatelet drugs were given. She remained well with no thrombo-embolic event or bleeding. Echocardiography at day 30 showed recovery of LV function and no mural thrombus, so the dabigatran was stopped.

Discussion

There is currently little or no information on use of novel oral anticoagulants to reduce thrombo-embolic risk from LV mural thrombus. In our patient avoidance of anticoagulant and antiplatelet therapy for one week after presentation with acute anterior myocardial infarction (MI) was unusual and predisposed to mural thrombus formation. The cause of MI was spontaneous coronary artery dissection, which was managed conservatively without stenting because there was no evidence for ongoing myocardial ischemia, and there is no established benefit from antiplatelet therapy for this indication [1]. Dabigatran was chosen to treat the LV mural thrombus because in the RE-LY trial [2] the risk of intracranial haemorrhage was lower for patients treated with dabigatran compared to warfarin, and the estimated 10-15% thromboembolic risk from the mural thrombus [3] was thought to be greater than the low risk of further intracranial bleeding >1 week after a traumatic SAH [4].

Dabigatran and novel oral factor Xa inhibitors have several potential advantages for treatment of LV mural thrombus. A lower risk of intracranial hemorrhage [1,2] may be important in elderly patients and when dual antiplatelet treatment is also indicated after coronary stenting. Initiation and monitoring of early therapy is simpler than for vitamin K antagonists which require several days of intravenous heparin or subcutaneous low molecular weight heparin until therapeutic INRs are achieved. Patient education and laboratory monitoring are also simpler during the early period when both the bleeding and thrombo-embolic risk are greatest. [5,6] These advantages may be particularly relevant when anticoagulation for mural thrombus is only indicated for 3 months, or less, as in this case if the thrombus resolves and LV function improves [4].

Conclusion

Guidelines recommending that patients with or at high risk of LV mural thrombus after acute MI are treated for 3 months with an oral vitamin K antagonist are based on limited data from observational studies [4,7]. This case report supports the rationale for a randomised clinical trial to evaluate the benefits and risks of novel anticoagulants compared to conventional treatment with heparin and an oral vitamin K antagonist in patients with or at high risk of LV mural thrombus complicating MI.

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