CASE REPORT

Coronary thromboembolic acute myocardial infarction due to paroxysmal atrial fibrillation occurring after non-cardiac surgery

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SUMMARY

Acute myocardial infarction is a well known precipitant of atrial fibrillation, but it is also becoming increasingly recognised that atrial fibrillation is a direct and indirect cause of acute myocardial infarction. Current guidelines do not recommend anticoagulation therapy in patients undergoing cardiac surgery who have a brief episode of atrial fibrillation lasting less than 48 h. However, recommendations for the management of atrial fibrillation following non-cardiac surgery are less clear. We describe the case of a 70-year-old man undergoing non-cardiac surgery, who developed a short episode of perioperative atrial fibrillation and later presented with thromboembolic acute myocardial infarction due to a thrombotic occlusion of the right coronary artery.

BACKGROUND

Atrial fibrillation is a well-recognised complication of acute myocardial infarction (MI). However, it is becoming increasingly recognised that atrial fibrillation (AF) can also directly cause acute MI. We present a case of a patient presenting with an embolic MI as a result of new-onset paroxysmal AF, which first manifested itself following non-cardiac surgery. The management of patients with new-onset paroxysmal AF occurring in the setting of non-cardiac surgery is unclear. The guidelines are confusing, as they state that patients presenting with a brief episode of AF following cardiac surgery do not require long-term anticoagulation. However, no recommendations are provided regarding the management of patients presenting with new onset AF in the setting of non-cardiac surgery.

CASE PRESENTATION

A 70-year-old man presented to the emergency department (ED) of a referring hospital with localised sharp epigastric pain and was diagnosed with acute cholecystitis. His medical history was significant for arterial hypertension (treated with perindopril/indapamide 5/1.25 mg) and dyslipidaemia (treated with atorvastatin 20 mg). Laparoscopic cholecystectomy was performed 4 days after admission in the peripheral hospital following a trial of conservative management. Baseline ECG revealed normal sinus rhythm, with left ventricular hypertrophy strain pattern (figure 1A). Preoperative transthoracic echocardiography revealed normal left ventricular systolic function and concentric left ventricular hypertrophy consistent with

hypertensive heart disease. Two hours following the procedure, the patient complained of worsening dyspnoea and a 12-lead ECG revealed new-onset AF with rapid ventricular response (figure 1B). The episode of AF lasted approximately 1 h before reverting to normal sinus rhythm following the administration of intravenous metoprolol 5 mg. A blood sample for troponin T taken 2 h following the episode of AF was found to be elevated at 146 ng/L (upper reference limit (URL) 50 ng/L). A diagnosis of non ST-segment elevation MI (NSTEMI) was suspected by physicians in the peripheral hospital and the patient was referred to our centre for urgent coronary angiography. The latter investigation revealed widely patent coronary arteries with only minor non-obstructive disease and preserved left ventricular systolic function (figure 2A–C, video 1). The patient was noted to be back in sinus rhythm. The clinical presentation was interpreted to be ‘demand ischaemia’ secondary to perioperative AF with rapid ventricular response in combination with left ventricular hypertrophy (ie, type 2 MI). The patient was discharged back to the referring hospital for postoperative recovery. Differential diagnosis included coronary artery thrombus and immediate reperfusion, severe coronary artery spasm (which may be associated with cholecystitis and operative stress) and takotsubo cardiomyopathy, although there was no evidence of apical ballooning on the ventriculogram. The CHA₂DS₂VASc score was 2 (age >65 (1 point) and arterial hypertension (1 point)) suggestive of a moderate-high risk of a thromboembolic event. However, because AF occurred for <48 h duration in a postoperative setting (albeit non-cardiac surgery), a decision was made not to initiate oral anticoagulation. Instead, aspirin and a β -blocker were prescribed and a 24 h Holter ECG was scheduled in 4 weeks time. Oral anticoagulation was to be initiated if AF was detected on the follow-up Holter monitor.

Eighteen days later, the patient represented to the ED reporting of epigastric and left-sided chest pain radiating to the back, with symptom onset at 17:30 h. A 12-lead ECG demonstrated AF with rapid ventricular response (~150 bpm), ST segment depression in leads I, II, and aVL and V4–V6, and borderline ST segment elevation in leads III and aVR (figure 3). Initial high-sensitivity troponin T, which was sampled three and a half hours following symptom onset (21.07 h), was mildly elevated (0.035 μ g/L (URL 0.014 μ g/L)) but creatine kinase was normal (43 IU/L (URL <190 IU/L). High-sensitivity troponin T and creatine kinase

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Figure 1 Baseline 12-lead ECG revealed normal sinus rhythm with left ventricular hypertrophy strain pattern. An ectopic supraventricular extrasystole is also apparent (A). A 12-lead ECG performed 2 h following laparoscopic cholecystectomy revealed atrial fibrillation with rapid ventricular response (B).

Figure 2 Initial coronary angiogram revealed a widely patent left anterior descending (LAD) (A), left circumflex (LCX) (B) and right coronary artery (C). The LAD and LCX had separate ostia (A and B).

were again taken 7 h following the first set (at 04:07) were found to be further elevated (high-sensitivity troponin T 0.388 µg/L, creatine kinase 496 IU/L). The consultant cardiologist on call was informed of this result and the decision was made to perform coronary angiography at the start of the day's list at 08:00, as the patient was symptom free at this point. However, at 07:15 (3 h following the second troponin T and creatine kinase), the patient suddenly became bradycardic (heart

rate 35 bpm) and hypotensive, and developed cold sweats; he was, therefore, transferred to the intensive care unit for an isoprenaline infusion. The patient subsequently reverted to normal sinus rhythm with a heart rate of 70 bpm (noted on cardiac monitor—an ECG was not performed at this point) and blood pressure stabilised. High-sensitivity troponin T and creatine kinase were performed a third time at 07:25 (3 h after the second round) and were found to be further elevated at 0.540 µg/L and 784 IU/L, respectively. The patient again developed chest pain and the decision was made to perform urgent coronary angiography (15½ hours following initial symptom onset, 1½ hours following the episode of bradycardia and 19 days after the initial unremarkable coronary angiogram). Vascular access via the right radial artery for coronary angiography was obtained at 08:52.

INVESTIGATIONS

Coronary angiography revealed a widely patent left anterior descending coronary artery but with evidence of retrograde filling of the right coronary artery (RCA; figure 4A). The left circumflex coronary artery was widely patent (figure 4B). The RCA was completely occluded up to the ostium and was filled with echogenic material indicative of thrombus (figure 4C, video 2).

TREATMENT

Emergent percutaneous coronary intervention (PCI) of the RCA was performed via the right radial artery (figure 5). Following the administration of intravenous unfractionated heparin and the successful passing of a guidewire (BMW Universal II Guidewire, Abbott Vascular, Santa Clara, California, USA), the vessel was predilated with the return of thrombolysis in myocardial infarction grade 1 flow (figure 5A). Thereafter, thrombus aspiration was performed. (figure 5B). Two intravenous boluses

Video 1 Coronary angiography performed following the first episode of atrial fibrillation revealed widely patent coronary arteries. The patent right coronary artery is shown.

Figure 3 A 12-lead ECG performed 18 days following a normal coronary angiogram demonstrating atrial fibrillation with rapid ventricular response, ST segment depression in leads I, II, and aVL and V4–V6, and borderline ST segment elevation in leads III and aVR.

Figure 4 Emergent coronary angiography performed 18 days following the initial coronary angiogram revealed a widely patent left anterior descending coronary artery (A) and left circumflex coronary artery (B) but with retrograde filling of the right coronary artery (RCA) (A, arrow). Selective angiography of the RCA confirmed it to be completely occluded to its ostium (C).

of the glycoprotein IIb/IIIa inhibitor eptifibatide were administered at a 10 min interval. The angiographic result was considered satisfactory following multiple passes (~15–20 times) of the thrombus aspiration catheter and the decision was made not to proceed with stent implantation (figure 5C). The patient was transferred to the coronary care unit with an 18 h intravenous infusion of the glycoprotein IIb/IIIa inhibitor, eptifibatide. The maximum creatine kinase value was 1822 IU/L. A 12-lead ECG

performed 1 day following PCI revealed normal sinus rhythm, with no evidence of any pathological Q-waves (figure 6).

OUTCOME AND FOLLOW-UP

The patient made an excellent recovery and was discharged home 3 days later with lifelong oral anticoagulation (rivaroxaban 20 mg/day) and aspirin 75 mg/day for 1 month. The patient was reviewed 4 months later in the outpatients department and was doing very well with no symptoms being reported. A routine electrocardiographic stress test was performed during the outpatient visit, which was clinically and electrically normal. Transthoracic echocardiography performed at 4 month follow-up revealed a left ventricle of normal dimensions with normal left ventricular systolic function (ejection fraction of 60% measured using Simpsons biplane formula) but with some evidence of inferobasal and inferolateralbasal akinesia. Right ventricular function was normal (tricuspid annular plane systolic excursion (TAPSE) 23 mm)).

DISCUSSION

Although the incidence and prevalence of coronary artery embolism secondary to AF is unknown, it is generally considered to be rare. AF occurs in 6–21% of patients with MI and the latter is a well-known risk factor for AF.¹ Conversely, AF is only recently beginning to be recognised as a risk factor for MI.^{2–9} In a recent prospective cohort of 23 928 participants without coronary heart disease at baseline enrolled from the Reasons for Geographic and Racial Differences in Stroke (REGARDS) cohort study, AF was observed to be associated with an almost twofold increased risk of MI, and this association remained significant after adjustment for traditional cardiovascular risk factors (adjusted hazard ratio 1.70, 95% CI 1.26 to 2.30).² There are several reasons for the association between AF

Video 2 Coronary angiography performed following the second episode of atrial fibrillation revealed a completely occluded right coronary artery up to the ostium.

Figure 5 Emergent percutaneous coronary intervention of the right coronary artery (RCA). Echogenic material was seen in the RCA following initial balloon angioplasty (A). Two intravenous boluses of eptifibatide were administered and thrombus aspiration was performed numerous times (B). The final angiographic result was considered satisfactory following thrombus aspiration only and therefore no stents were implanted (C).

Figure 6 A 12-lead ECG performed 1 day following emergency percutaneous coronary intervention of the right coronary artery demonstrating normal sinus rhythm.

and MI. AF and MI share common pathophysiological processes and risk factors (eg, age, diabetes mellitus, hypertension).² Furthermore, inflammation plays a pathogenic role in AF and MI.² However, as highlighted in the present case, thromboembolism is also a potential mechanism. There have only been a few reports of acute MI secondary thromboembolism caused by AF.²⁻⁹ The present report is unique in that coronary angiography was performed only 3 weeks before the thromboembolic event, confirming widely patent coronary arteries. Given the large burden of thrombus found in the RCA and absence of any

coronary lesions following thrombus aspiration, thromboembolism secondary to AF was the most likely cause of the acute MI.

Contributors CJS was involved in the concept and drafting of the manuscript and performed the percutaneous coronary intervention. MS was involved in the intellectual input and drafting of the manuscript. DT was involved in intellectual input and image preparation. FE was involved in intellectual input and critical revision of the manuscript.

Competing interests None.

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Learning points

- ▶ Atrial fibrillation frequently *complicates* acute myocardial infarction. However, a bidirectional relationship exists between atrial fibrillation and acute myocardial infarction in that atrial fibrillation may also directly cause acute myocardial infarction.
- ▶ Consideration should be given to long-term anticoagulation among patients presenting with new-onset atrial fibrillation following non-cardiac surgery, particularly in the presence of an elevated CHA₂DS₂VASc score, although further evidence is required.
- ▶ Patients presenting with an embolic myocardial infarction may not have underlying coronary artery disease. Therefore, coronary stent placement may not be mandatory in such patients. The present patient was treated with thrombus aspiration alone (together with potent antiplatelet agents and anticoagulants) and made an excellent recovery.

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