Case Report

Acute atrial fibrillation in emergency surgery: Is it rare?

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ABSTRACT

Atrial fibrillation (AF) is a common arrhythmia with an estimated clinical prevalence of approximately 1% in the general population and as high as 9% in individuals by the age of 80 years. The aetiology is multifactorial. Systemic disease, e.g., inflammatory processes, sarcoidosis, autoimmune disorders, has also been linked to the development of AF. Myocardial dysfunction observed in sepsis could contribute to arrhythmias and inflammation *per se* could induce or provoke AF. We describe the successful management of an acute AF in an elderly patient scheduled for emergency laparotomy and closure of hollow viscous perforation.

Key words: Acute atrial fibrillation, elderly patient, electrolyte imbalances, sepsis

INTRODUCTION

Atrial fibrillation (AF) is a common arrhythmia with an estimated clinical prevalence of approximately 1% in the general population and as high as 9% in individuals by the age of 80 years.[1] The mortality increases with advanced age and new onset of AF.[2] Myocardial dysfunction observed in sepsis could contribute to arrhythmias and inflammation *per se* could induce or provoke AF.[3] We describe the management of an acute AF in an elderly patient scheduled for emergency laparotomy.

CASE REPORT

A 77 year-old lady (weight: 50 kg) of American Society of Anaesthesiologists (ASA) III was scheduled for emergency laparotomy and closure of hollow viscous perforation. Her past medical history was unremarkable and she was not on any medication. She had an uneventful general anaesthesia for laparoscopic appendicectomy 2 days ago. However, she had abdominal pain and fever postoperatively in the ward for last 24 hours. On examination, she was febrile; her pulse was 110/min, regular; blood pressure was 106/72 mmHg and respiratory rate was 20/min. On auscultation, there was no abnormal heart and breath sounds. Blood investigations done 6 hours prior to surgery revealed haemoglobin of 11.9 g/dl, total counts of 14,000/mm^3 with 13% band forms, serum sodium 140 mEq/l, potassium 3.6 mEq/l, platelets 220,000/mm^3 and creatinine of 1.4 mg%. Chest roentgenogram revealed bilateral mild pleural effusion with normal cardia. Electrocardiogram (ECG) showed sinus tachycardia with heart rate (HR) of 106/min.

In the operating room, baseline monitors (pulsoximetry/non-invasive blood pressure/ECG) were established. ECG showed HR of 156/min in AF, which was not present earlier and confirmed with 12-lead ECG. Invasive blood pressure was 106/72 mmHg. SpO_2 on 60% oxygen was 93%. In view of acute onset AF, intravenous metoprolol was administered to control ventricular rate in aliquots of 1 mg to a total of 5 mg. But there was no change in HR. Defibrillator was kept available. As the patient's blood pressure was stable, patient's trachea was intubated with rapid sequence induction using midazolam 1 mg, fentanyl

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250 mcg, propofol 60 mg and succinylcholine 60 mg. Central line was inserted in right internal jugular vein (IJV) under aseptic precautions. Anaesthesia was maintained with air, oxygen, sevoflurane, fentanyl, hydromorphone and rocuronium. As the HR was refractory to beta blockers, intravenous amiodarone 150 mg was administered. HR reduced to 80–90/min with an improvement in blood pressure (126/86 mmHg) and rhythm reverted back to sinus within 15–20 min of administration. Arterial blood gas and serum electrolytes showed uncompensated metabolic acidosis (bicarbonate = 16 mEq/l) and respiratory alkalosis (PCO_2 = 26 mmHg) with serum potassium of 3 mEq/l. 20 mmol/L of intravenous potassium was administered as infusion over an hour. The repeat serum potassium was 3.4 mEq/l. The rest of the intraoperative period was uneventful. Patient was transferred to intensive care unit (ICU) postoperatively. IV potassium supplementation of 20 mmol/l was administered along with 1 g of IV magnesium sulphate (serum magnesium 1.2 mg/dl) and calcium chloride (serum calcium 6 mg/dl) in the ICU. Serum C-reactive protein (CRP) and procalcitonin was found to be elevated. Thyroid function tests were normal. The patient's trachea was extubated after 12 hours with controlled ventricular rate and sinus rhythm. On consultation with cardiologist, tab. aspirin 81 mg once daily was started after negative transthoracic echocardiogram. She was advised regular follow-up and discharged home on low-dose aspirin therapy.

DISCUSSION

Elderly patients have higher incidence of AF. Fibrosis and loss of muscle mass of the left atrium play a major role in the development of AF. Genetic defects like lamin AC gene mutations might explain the idiopathic development of AF.[4] The aetiology is multifactorial. Systemic disease, e.g., inflammatory processes, sarcoidosis, autoimmune disorders, has also been linked to the development of AF.[5] There is substantial evidence linking inflammation to the initiation and perpetuation of AF. It was initially suspected by the observation that inflammatory states, such as myocarditis, pericarditis, and cardiac surgeries, are frequently associated with AF. There is a greater association between elevated CRP and presence of AF, which is supported by the findings from a large population-based cohort study of 5806 elderly individuals followed for a mean of 6.9 years.[6] Every 1 mg/dl increase in serum CRP was associated with a seven fold increased risk of recurrent AF and a 12-fold increased risk of permanent AF compared with controls.[7] Advanced age, blunt thoracic trauma, shock (notably septic shock), use of pulmonary artery catheter, and previous treatment with calcium channel blockers are the independent risk factors of AF in intensive care patients.[2] Our patient had AF probably due to advanced age, underlying sepsis and electrolyte imbalances.

There are multiple therapeutic strategies for the management of AF, which may confuse individual practitioners.[8] Direct current cardioversion (DCC), pharmacotherapy with antiarrhythmic drugs, and anticoagulation are the mainstay treatment modalities in acute AF. Antiarrhythmic drugs with class I (flecainide, propafenone) and III (amiodarone) agents are used when adopting a rhythm-controlling strategy, whereas class II (beta blockers) and IV (diltiazem, verapamil) agents are reserved for rate-controlling measures, although class III agents share rate-controlling properties.[9] As the blood pressure was stable in this patient, pharmacological conversion to sinus rhythm was attempted initially with beta blocker and then with intravenous amiodarone. Calcium channel blockers were not used in our patient as fatal complete heart block has been described with their use along with beta blockers.[10] Amiodarone was considered for AF of acute onset and to avoid secondary complications of AF like ischaemia, systolic dysfunction and embolism. Airway was secured immediately in our patient to avoid hypoxia, hypercarbia, acidosis, which could further precipitate AF, and to facilitate DCC if needed. In patients with ventricular rates more than 150, ongoing chest pain, or with evidence of critical perfusion: Systolic BP less than 90 mmHg, heart failure, or reduced consciousness, the need to restore haemodynamic stability by the restoration of sinus rhythm is of prime importance, and guideline consensus advocates DCC as the first-line therapy to achieve this, with pharmacologic strategies being used either secondarily or in conjunction with DCC.[9] The patient was discharged on low-dose aspirin therapy for 6 months to prevent thromboembolic complications following AF.

CONCLUSION

Acute AF in an elderly patient coming for emergency surgery with underlying sepsis and electrolyte imbalance needs immediate HR control and conversion to sinus rhythm. Intraoperative vigilant monitoring, intravenous use of amiodarone, correction

of electrolytes and underlying pathology played a vital role in the successful management of this patient.

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