

Central Venous Catheter induced Atrial Fibrillation-A Case Report

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Abstract:

Though a common procedure, central venous access is related to morbidity and mortality of patients. Common cardiac complications caused by central venous catheters include premature atrial and ventricular contractions. But development of atrial fibrillation with haemodynamic instability is quite rare. We are reporting a patient who developed atrial fibrillation with hypotension while inserting central venous catheter through right subclavian

vein by landmark technique. Patient was managed with DC cardioversion. Careful insertion of central venous catheter & prompt management of its complication is crucial to avoid catastrophe.

Keywords: Central venous catheter, Atrial fibrillation, General anaesthesia.

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Introduction:

Establishment of central venous catheter (CVC) in critically ill patients of intensive care unit, operating room, emergency & casualty and in renal dialysis centre is now a routine procedure both for monitoring and therapeutic purposes. But it can invite some serious complications like cardiac dysrhythmias, pneumothorax, vessel/nerve injuries, thromboembolism and infection.¹ Strict attention to insertion technique and correct line tip position can reduce the risk of dire complications like cardiac arrest and even death if not immediately intervened. So, clinicians must remain aware and prepared with management plan if there is any emergency. Here we report a case of first diagnosed atrial fibrillation (AF) with haemodynamic instability during placement of a CVC in an anaesthetized patient

scheduled for emergency craniotomy for intracerebral haemorrhage. Informed written consent of the patient's next of kin (wife) and approval from institutional review board were obtained.

Case report:

Mr. X aged 46 years (weight – 73 kg, height – 174 cm) with no known comorbidity or history of cardiac events, scheduled for emergency craniotomy to remove blood clot for right sided intracerebral haemorrhage (ICH). In the operating room his baseline parameters were as follows: pulse – 44/min (regular), blood pressure (BP) – 186/113 mm of Hg with Glasgow coma scale (GCS) score – E₁M₃V₂ (8/15). There was left sided hemiparesis (2/5). All routine investigations including electrocardiogram (ECG) were within normal limit except serum potassium level which was slightly low (3.2 mmol/L). General anaesthesia was administered following the standard protocol. After left radial arterial cannulation, junior resident tried inserting CVC (7Fr/20cm /tri channel) through right subclavian vein using landmark technique under ECG monitoring. While introducing guidewire there were irregular RR intervals on ECG monitor. So, guidewire was withdrawn approximately 6 cm and CVC was fixed at 12 cm. But dysrhythmias persisted. On monitor his heart rate was 150 -190 /min (irregular) and

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radial pulse was 40–50/min (irregularly irregular, feeble). Patient was diagnosed as a case of first diagnosed fast AF (Fig. 1). Other parameters like oxygen saturation (98%), end tidal carbon dioxide (32 mm of Hg) were within normal limit for the patient. After switching off isoflurane, patient was ventilated with 100% O₂. All other possible predisposing factors were excluded. But within minutes his blood pressure (BP) fell to 67/31 mm of Hg. So synchronized direct current cardioversion (120 J biphasic) done with immediate establishment of sinus rhythm (SR) - 94/min (regular) (Fig. 2) and BP - 168/96 mm of Hg, which subsequently settled at around 140/90 mm of Hg. Potassium supplementation was started by syringe pump (20 ml/hr).

After stabilization of cardiac status patient was observed for fifteen minutes when 12 lead ECG was done and found to be normal (Fig. 3). Blood was sent for evaluation of cardiac enzymes. As it was an emergency case and

patient was haemodynamically stable with no recurrence of dysrhythmias, surgery was allowed to start. The duration of surgery was 155 minutes which was uneventful. At the end of surgery patient was shifted to intensive care unit where his cardiac status was evaluated by cardiologist and found to be normal as per clinical findings, cardiac enzymes, 12 lead ECG, chest skiagram and echocardiography reports. Anticoagulant was not prescribed as it was a case of ICH and surgical intervention. No antiarrhythmic drug (AAD) was given as prophylaxis of AF recurrence. He was successfully weaned from ventilator on 8th postoperative day. Patient was followed for four weeks after the incidence and there was no recurrence of dysrhythmia or cardiac related event. His BP is now under control with three antihypertensive drugs (Ramipril, Cilnidipine, Prazosin hydrochloride) and after four weeks he underwent neuro rehabilitation for low GCS (14/15) and left sided weakness (4/5).



Fig.-1: ECG tracing just before cardioversion.

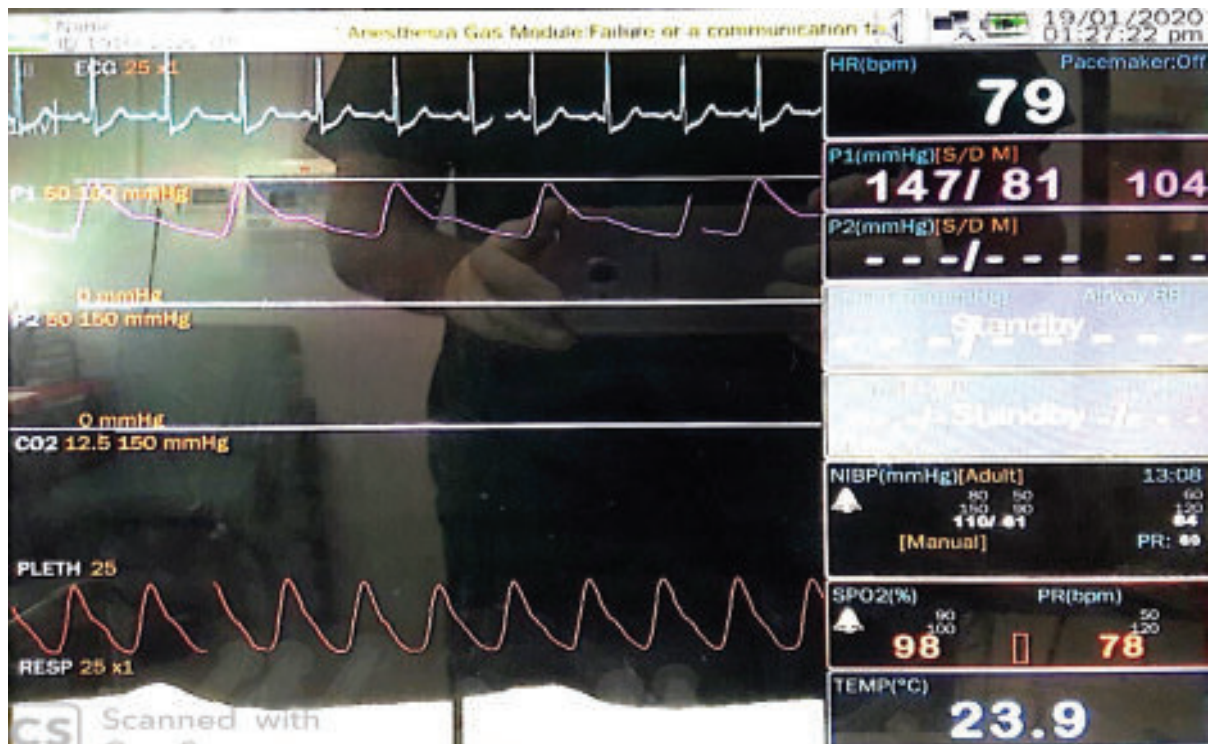


Fig.-2: ECG tracing just after cardioversion.

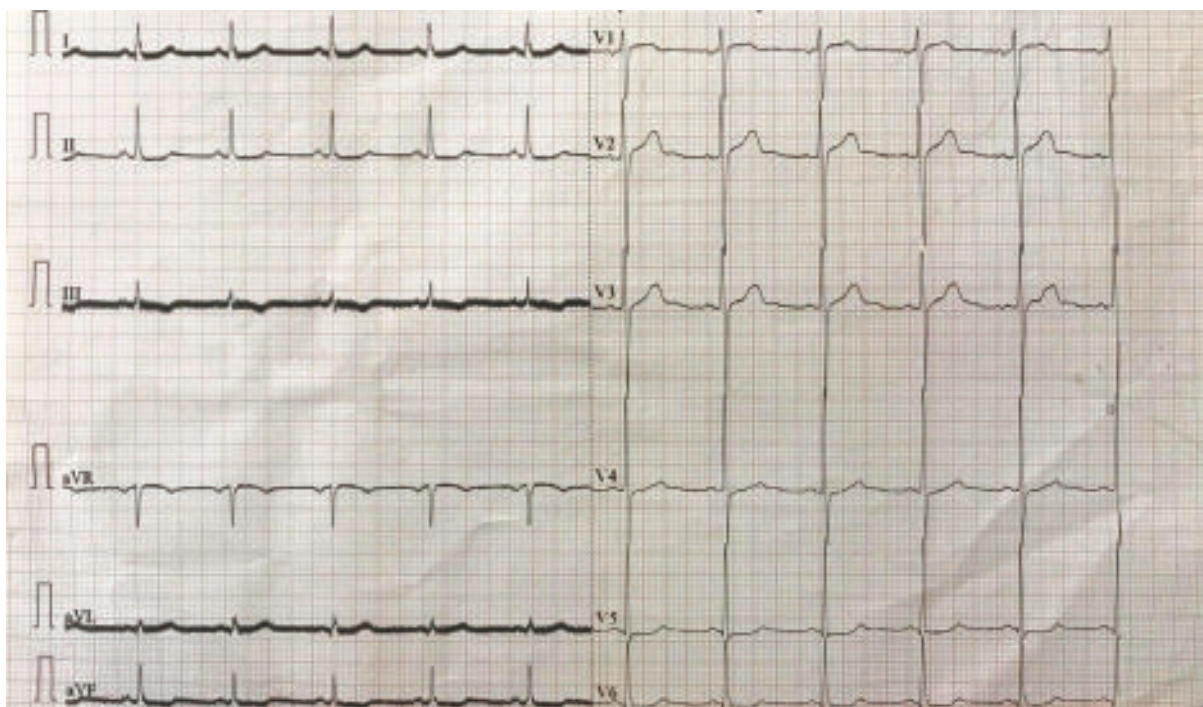


Fig.-3: 12 lead ECG after cardioversion.

Discussion:

Despite the benefits of central venous lines to patients and clinicians, more than 15% patients will have catheter related complications.¹ Benign cardiac dysrhythmias resulting from guide wire or catheter tip in the atrium or the ventricle during the cannulation procedure is usually transient.² However severe arrhythmias have been reported during catheter insertion and use.³

Expert opinion suggests that the tip should be parallel to the wall of a large central vein outside of the pericardial inflection.⁴ This reduces the risk of perforation and the risk of cardiac tamponade if perforation occurs. During insertion the catheter tip position can be adjusted according to electrocardiogram or central venous pressure pattern.⁵

Arrhythmia results from guide wire contact with right atrium. Most frequently this results in premature atrial and ventricular contraction.⁶ However, if the atrioventricular node is in contact with guide wire or tip of CVC for a significant amount of time supraventricular tachyarrhythmias can result and lead to fatal arrhythmia and arrest.⁷

Supraventricular arrhythmias in particular AF are important for anaesthesiologists as it is associated with haemodynamic derangement, postoperative stroke, perioperative myocardial infarction, ventricular arrhythmias, heart failure and longer hospital stay.⁸

AF is the most common pathological supraventricular arrhythmias and is caused by multiple electrical wavelets appearing in the atria simultaneously with an irregular ventricular response.⁹ AF is classified as first diagnosed when occurring suddenly in a patient previously in sinus rhythm as it happened in our patient. Characteristics of ECG findings of AF are the absence of p waves, with fibrillatory waves in their place and irregular RR intervals.⁹

Whenever possible cardiac arrhythmias should be controlled before operation as surgery and anaesthesia can cause marked deterioration.⁹ Upon acute onset of intraoperative arrhythmias, etiology like hypoxemia, hypovolemia, electrolyte imbalance, pneumothorax, pulmonary embolism, acute myocardial infarction, drug effect, cardiothoracic surgery etc. should be considered before therapy is instituted. In our case, patient had mild hypokalemia which was subsequently managed and probably not related with the incidence of AF.

In cases of perioperative AF, the goal of management is rate control. Slowing the ventricular rate lengthens diastole, enhancing stroke volume and reduces myocardial oxygen consumption. The beta blocker esmolol is short acting & easily titratable, and recommended for this purpose. Although the nondihydropyridine calcium channel blocker diltiazem has a less inotropic action than esmolol but it is less easily titrated and not recommended in the perioperative period due to its slow onset of action (six hours) and low efficacy in high adrenergic state such as surgery. Commonly used rate controlling drugs have side effects like hypotension, bradycardia, heart block and heart failure which may deteriorate the haemodynamics under anaesthesia.

For rhythm control in general, electrical cardioversion is faster, more effective and more efficient than pharmacological cardioversion. Furthermore, patients with extreme haemodynamic instability characterized by hypotension, pulmonary oedema or sign symptoms of myocardial ischemia should be treated with immediate direct current electrical cardioversion.⁹

There is risk of thrombus formation in atrium during AF, so use of electrical cardioversion in AF may lead to thromboembolism. The routine cases should be anticoagulated before cardioversion and in emergency transesophageal echocardiography can be done to exclude the presence of left atrial thrombus.¹⁰ All patients should be anticoagulated with warfarin or nonvitamin K oral anticoagulants (NOACs) for at least four weeks after cardioversion because mechanical function of the atrium lags by up to seven days after the restoration of sinus rhythm.¹¹ In this case anticoagulant was not prescribed to avoid rebleeding as it was a case of intracerebral haemorrhage followed by surgical intervention.

Although some complications appear critical, direct current synchronized cardioversion is usually safe and effective if performed under the care of well trained personnel. Troponin I measurements after cardioversion were not elevated in patients with normal and reduced left ventricular function suggesting lack of myocytes injury.¹²

Maintenance of sinus rhythm after cardioversion is challenging with recurrence rates ranging from 63 - 84% in the first year.¹³ Overall the rhythm control strategy

using AADs to maintain SR has not shown any clear benefit on clinical outcomes rather associated with severe adverse effects including the potential induction of life-threatening arrhythmias.¹⁴ No AAD showed superior efficacy compared to others although amiodarone did show a tendency for greater additional rhythm control. But because of its extracardiac side effects it has been recommended to reserve this drug for specific situation such as concomitant heart failure.¹⁵ Treatment of underlying disease, analgesia, oxygenation and correction of haemodynamic or electrolyte derangement may restore sinus rhythm in the majority of cases without the need of AADs.⁹ In our case we were almost certain that the cause of the first diagnosed AF was central venous catheterization and there was no structural heart disease. As there was no precipitating factor and no recurrence of dysrhythmia in our patient, we avoided any AAD in postoperative period.

Dysrhythmias and its grave consequences during central venous catheter insertion can be reduced if not totally eliminated, by improving professional skill. Using ECG monitor for early recognition of such event and its prompt management following standard protocol is crucial to save lives.

Conflict of interest- None.

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