Intraoperative Atrial Fibrillation Of Unknown Etiology: A Unique Complication
H Prabhakar, S Bhatnagar, S Mishra, R Narang

Citation

Abstract
Cardiac arrhythmias are not uncommon during thoracic and cardiac operations and amongst these, atrial fibrillations are not rare. Their occurrence during non-cardiac surgery in an ASA Grade 1 patient is a matter of concern for the anesthesiologist. We report the case of a 45-year-old male patient diagnosed with left pelvic chondrosarcoma who was posted for left hemipelvectomy. The patient's pre-operative evaluation revealed a normal respiratory and cardiovascular status. The routine investigations were acceptable for elective surgery. Intraoperatively, the patient had atrial fibrillations that continued until the end of surgery. No cause could be attributed to this arrhythmia. As the patient was hemodynamically stable, the surgery was allowed to be continued. Postoperative course of the patient was uneventful with spontaneous return of sinus rhythm.

INTRODUCTION
No arrhythmia is unique to anaesthesia practice. Anaesthesia may per se potentate the risk of developing arrhythmia in an individual, but more particularly in those susceptible. Cardiac arrhythmias are a relatively frequent occurrence during anaesthesia. The possible precipitating factors include: hypoxia, hypercapnia, myocardial infarction, catecholamines, electrolyte abnormalities, acid-base imbalance, drug toxicity and adverse drug reactions. The sudden appearance of any new arrhythmia, regardless of hemodynamic consequences, should be of concern and warrants attention. Specific therapy in the form of drugs, cardioversion, or pacing will have their efficacy greatly enhanced by prior institution of corrective measures. Frequently, drug or electrical therapy will not be required if aggravating factors are removed. We present this case where the patient had atrial fibrillations in the intraoperative period and no known cause could be attributed to it.

CASE REPORT
A 45-year-old male patient weighing 60 kg was scheduled for left hemipelvectomy after being diagnosed with left pelvis chondrosarcoma. His presenting complaint was a 4 cm swelling in the inguinal region and thigh since 1 year. He was a non-smoker and non-alcoholic. Past history was not suggestive of any medical disorder. His pre-operative routine investigations included the hemogram, blood urea and sugar, CXR, ECG, serum electrolytes. All the reports were within normal limits. The patient was pre-medicated with Diazepam 10 mg on the morning of the surgery. He also received Morphine 5 mg iv, Phenargan 25 mg iv and Glycopyrollate 0.2 mg intramuscularly prior to being transferred to the operation theatre. The pre-induction vitals of the patient were pulse of 88 beats/min and regular blood pressure of 140/80 mmHg.

An epidural catheter was placed in the L3-4 space and a test dose of 2 ml of 2% xylocaine with adrenaline was given. The catheter was used for analgesia during and after the surgery. General anaesthesia was induced using morphine 6 mg, thiopentone 250 mg, and vecuronium 8 mg. The patient was intubated with a cuffed portex 8.5-mm tube. The right basilic vein was cannulated using a cavafix 18G 70 cm that served for continuous CVP monitoring. The right radial artery was used for measuring the invasive arterial blood pressure. Anaesthesia was maintained with O2, N2O, isoflurane, vecuronium and epidural sensorcaine 0.25% at regular intervals.

The position of the patient was supine with the legs abducted to facilitate surgery. Intra-operative monitoring included heart rate, 5-lead ECG, SpO2, EtCO2, CVP, NIBP and IBP. Twenty minutes before the incision a bolus of 10 ml 0.25% sensorcaine was given through the epidural catheter. The surgery was started and the patient showed no response to surgical incision, indicating an adequate depth of anaesthesia. Nearly forty minutes after the surgical incision,
the patient began to develop an irregular cardiac rhythm. The ECG was abnormal and pulse irregular but the arterial blood pressure remained stable. The ECG showed a typical atrial fibrillation pattern, (Fig.1) which was confirmed by the cardiologist.

**Figure 1**
Figure 1: A 5-Lead Electrocardiogram showing atrial fibrillation

The surgery was discontinued for some time to see whether a cause could be found. As the patient was hemodynamically stable, the surgery was then allowed to continue. The arterial blood gases and serum electrolytes done before and during the arrhythmia were in acceptable range. Throughout the surgery, CVP was maintained between 10-15 cm H₂O, EtCO₂ between 31-36 mmHg and isoflurane 0.7-0.9. The surgery lasted for about 9 hrs during which the patient was stable. Blood loss and fluids were adequately replaced. At the end of surgery the neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.2 mg. The atrial fibrillations reverted back to the sinus rhythm and once the patient became fully conscious and awake, he was extubated. The patient was then transferred to the ICU and the post-operative period was uneventful.

**DISCUSSION**

The existence of atrial fibrillations in the absence of overt cardiovascular disease has been recognized since the early 1900s. In 1913, Gossage and Hicks (1) first pointed out that auricular fibrillations might arise in a heart previously healthy. Most reports agree that these comprise only 2% to 6% of all cases of atrial fibrillation (2). Evans and Swann proposed the term lone auricular fibrillation for all cases where the etiology of the atrial fibrillation largely remained unknown. This condition has also been described variously as benign, idiopathic, arteriosclerotic, functional, and senile fibrillation, fibrillation of unknown origin and fibrillation without heart disease (3). Auricular fibrillations occur in persons with a clinically normal heart as a result of varied stimuli-toxic, traumatic and reflex-and without any obvious cause. (Table 1)

**Figure 2**
Table 1: Causes of auricular fibrillation

<table>
<thead>
<tr>
<th>Toxic factors</th>
<th>Trauma</th>
<th>Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>(a) Infections: pneumonia, influenza, osteomyelitis, gastro-enteritis, malarial chill, pelvic abscesses</td>
<td>(a) Injuries: to head, chest</td>
<td>(a) Appendicitis, Gallbladder colic, Pepsitis</td>
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<tr>
<td>(b) Drugs: epinephrine, digibals, acetylcholine, acid, ether</td>
<td>(b) Shock, electric</td>
<td>(b) Indigestion and overeating</td>
</tr>
<tr>
<td>(c) Other chemical agents: alcohol, tobacco, arsenic</td>
<td>(c) Instrumentation to the ear</td>
<td>(c) Cough</td>
</tr>
<tr>
<td>(d) Diabetic coma</td>
<td>(d) Burns</td>
<td>(d) Vomiting</td>
</tr>
<tr>
<td>(e) Operation</td>
<td>(e) Electric</td>
<td>(e) Operation</td>
</tr>
<tr>
<td>(f) Exciton</td>
<td>(f) Operation</td>
<td>(f) Operation</td>
</tr>
<tr>
<td>(g) Nervous disturbances</td>
<td>(g) Exciton</td>
<td>(g) Exciton</td>
</tr>
<tr>
<td>(a) Excitement</td>
<td>(h) Emotion</td>
<td>(h) Pain</td>
</tr>
<tr>
<td>Miscellaneous: Hypertension</td>
<td>(i) Hypothesis</td>
<td>(i) Hypothesis</td>
</tr>
<tr>
<td>No apparent cause</td>
<td>(j) Hypersensitivity</td>
<td>(j) Hypersensitivity</td>
</tr>
</tbody>
</table>

Atrial fibrillations present as a grossly irregular rhythm disturbance, with a ventricular rate between 100 and 200 beats/min, except in impaired AV impulse transmission or ventricular pre-excitation syndrome (WPW syndrome), in which ventricular rate may be quite slow or exceed 300 beats/min, respectively. Slower rates are characteristic of idiopathic atrial fibrillation and occur in patients with impaired AV node conduction due to disease or drugs. There are no P-waves, R-R interval is grossly irregular and an undulating ECG base line can be seen. The fibrillating ‘f’ waves are best seen in lead II. (4)

The specific diagnosis of Idiopathic or Lone atrial fibrillations in our case was made as the fibrillations occurred without any pre-existing or co-existing coronary heart disease, congestive heart failure, rheumatic heart disease or hypertensive cardiovascular disease. The postoperative investigations of serum electrolytes (Na, K, Ca²⁺, Mg²⁺) and thyroid functions were within normal limits. A 24-hour Holter monitoring postoperatively did not show any abnormality.

Phillips and Levine (5) suggested that lone atrial fibrillations could be the result of a trigger phenomenon of neurogenic
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Origin in certain patients who are susceptible to functional nervous instability but are otherwise within normal limits. Lone fibrillations may occur as an acute transient episode, as recurrent paroxysmal or less frequent as chronic fibrillations (6). Our case may be a acute transient episode that manifested under anaesthesia. All the possible causes of the fibrillation under anaesthesia like hypoxia, hypercarbia, electrolyte abnormality acid-base imbalance, stress, and pain have been ruled out in our case.

The minimal alveolar concentration of isoflurane and the muscle relaxant vecuronium are both cardiostable and unlikely the cause of initiating the arrhythmia. Bertrand et al (7) reported in their study on 100 persons undergoing surgery under general anaesthesia that 84 patients had supraventricular and ventricular arrhythmias that were particularly common during intubation and extubation. None of their patients had atrial fibrillations or flutter. During our literature search on the subject, we came across only one reported incidence of atrial fibrillation during surgery under anaesthesia. Dodd et al (8) studied the incidence of cardiac arrhythmias in 569 unselected surgical patients. Out of 170 patients who had some or the other form of arrhythmia, only one patient had atrial fibrillation that persisted throughout operation and converted to normal rhythm as the patient emerged from anaesthesia.

Studies have shown that Lone atrial fibrillations are more common in males and in the older age group but sometimes found in younger subjects as well. In an asymptomatic patient, longevity is unaffected by the condition although a study by Phillips and Levine (5) suggests the fibrillations as the reversible cause of heart failure. Wolfe (6) et al studied 20 patients less than 50-yrs of age with atrial fibrillation and found no structural or external cause of their arrhythmia. Godtfredsen et al (7) have shown that Echocardiography is of minor value in patients with lone atrial fibrillation if symptoms and signs of other cardiac disorder are totally absent.

As we were unable to identify the cause of these atrial fibrillations and the patient was hemodynamically stable we have not used β-blockers, Ca²⁺ channel blocker or digoxin or even cardioversion to maintain sinus rhythm.

CONCLUSION

Atrial fibrillations are common arrhythmias occurring in 0.4-5% of adult population and nearly 5% of these are not associated with cardiac disease. It is important to identify the precipitating factor and eliminate it. Anesthesia may trigger such arrhythmias.

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References

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