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LETTER TO THE EDITOR Open Access

Perioperative arrhythmias and metabolic status: an elephant in the room

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It is not rare for an anesthetist to encounter heart rhythm disturbances in day-to-day practice of anesthesia and critical care and instead of getting intimidated and frightened, following basic principles eventually gets us to the shore. The foremost step is to find out the cause, whether physiological, structural, or any electrolyte imbalance. Being aware of the cause will not only guide us to the management but will also ensure that reoccurrence doesn't happen. The next step is to be aware of any hemodynamic instability as ventricular arrhythmias are notorious to cause low cardiac output states. Lastly, being observant of the arrhythmias throughout the perioperative period as they have the potential to cause cardiac compromise even though haemodynamics may seem stable at presentation. Based on these above-stated basic principles we present a clinical scenario encountered in a patient undergoing an emergency appendectomy.

A 48-year-old male reported to our hospital. After being evaluated for his symptoms by the surgical team a diagnosis of acute appendicitis was made and the patient was prepared for an open appendectomy. On examination by the anesthesia team, his pulse was found to be irregular and 12 lead electrocardiography showed ventricular bigeminy. 2D echocardiography was done which ruled out any structural abnormality of the heart. Investigations were as follows: hemoglobin: 12.3 gm%, TLC: 11,200, platelet: 2.3 lakh/μl, PTI: 100%, INR: 1.0, Na: 138 meq/L, K: 3.9 meq/L, urea: 23 mg/dl, creatinine: 0.9 mg/dl which were normal.

Considering the above-stated facts plan was to give general anesthesia, with lignocaine infusion (MacIntyre & Hillis, 2000). An arterial line was placed before induction and a standard rapid sequence induction was done. Rapid sequence induction was done with injection thiopentone 250 mg/dl, Injection succinylcholine—100 mg/dl, followed by intubation, following this injection atracurium: 30 mg, injection morphine 6 mg was given. The anesthesia was maintained on isoflurane and nitrous titrated to a MAC value of 1–1.2. Although arrhythmia reverted for around 15 minutes after giving lignocaine bolus of 2 mg/kg followed by infusion of 2 mg/kg/h, it swiftly reoccurred. At that time point, it was considered wise to get done an arterial blood gas analysis to rule out any metabolic and electrolyte disturbances. The ABG revealed metabolic acidosis with a pH of 7.18, base deficient of − 12, bicarbonate of − 11.8, rest of the findings were normal. As the arrhythmias were persistent, we decided to correct the acidosis with 150 meq of sodium bicarbonate. This metabolic acidosis could be due to sepsis because of the surgical condition as patient had abdomen pain for past 1 week. After the bicarbonate infusion, the arrhythmias were resolved. A repeat ABG showed no abnormality in the blood gas analysis. The patient was extubated and followed up with a postoperative 12 lead ECG. Which showed normal sinus rhythm with no recurrence of arrhythmias.

Metabolic acidosis is one of the important yet overlooked causes of arrhythmias with no other obvious signs. The previous published literature does give us insight into whether acidosis can cause various types of arrhythmias (Gandhi & Akholkar, 2015). The scenario of metabolic acidosis is mostly encountered in critical care, acute resuscitation, sepsis, or hemorrhagic shock where patients are mostly hemodynamically unstable. In this particular case, we came across a situation where even though the patient was not being critically ill or hemodynamically unstable yet he had underlying metabolic acidosis due to the low grade sepsis, and correction of acidosis reverted the arrhythmias. Relating to a similar clinical scenario a case report demonstrated that ECG changes of pseudo myocardial infarction can present with conditions causing acute abdomen. As low-grade

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sepsis causes reduced effective volume, increased venous capacitance, insensible water loss, hypodynamic state of perfusion would reduce coronary perfusion and oxygen delivery to the myocardium and cause these transient changes (Liao et al., 2008). One of the studies proposed that delayed after depolarization caused in myocardial muscles due to cellular acidosis can be the mechanism behind these arrhythmias during reperfusion after an ischemic event (Bai et al., 2017). These delayed after depolarization can be a potential cause in this case for causing arrhythmias. It has also been reported that rise in inflammatory markers in acute abdomen along with vagal stimulation has led to plague rupture and myocardial infarction (Moniuka, 2021). The cause of arrhythmias may be multiple but finding the cause when obvious routine causes are unlikely is also important. Hence, structural abnormality and functioning of the heart must be evaluated in patients with ECG changes and acute abdomen as done in present case.

One must consider evaluation of metabolic status, volume status in patients with abnormal ECG findings in background of acute abdomen after ruling out structural heart disease. The above factors must be given a thought as one of the differential diagnosis especially in seemingly stable patients.

Abbreviations

2D: Two-dimensional; TLC: Total leukocyte count; PTI: Prothrombin time index; INR: Internationalized normal ratio; MAC: Minimum alveolar concentration; ABG: Arterial blood gas analysis; ECG: Electrocardiogram

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