



Case Report

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Ventricular Tachycardia Following Mannitol-Induced Hyperkalemia :A Case Report and Literature Review

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Abstract

Diagnosis of Cardiac arrest in the operating room is relatively simple compared to other situations, as the majority of patients are closely monitored. Anesthetists and neurosurgeons should be aware that fatal arrhythmia can occur due to mannitol-induced hyperkalemia. We report a case of cardiac arrest type ventricular tachycardia on hyperkalemia induced by infusion of mannitol. Evidence is rare when dealing with cardiac arrest in neurosurgery, and more efforts are needed in understanding the etiologies, mechanisms and the specificities of its management.

Keywords: Ventricular tachycardia ; Hyperkalemia ; Cardiac arrest ; Neurosurgery

Introduction

Cardiac complications including Ventricular Tachycardia, Ventricular Fibrillation Pulseless Electrical Activity and Asystole during neurosurgery are rare but dreadful events. Diagnosis of Cardiac arrest in the operating room is relatively simple compared to other situations, as the majority of patients are closely monitored. Anesthetists and neurosurgeons should be aware that fatal arrhythmia can occur due to mannitol-induced hyperkalemia. We report a case of cardiac arrest type ventricular tachycardia on hyperkalemia induced by infusion of mannitol.

Case Report

A 58-year-old woman, 165 cm tall and weighing 78 kg, was suffering from headache and seizures for 4 months. Diagnosis

confirmed a right temporal lobe glioma. She was then scheduled for craniotomy and tumor resection under general anesthesia. The patient had a medical history of hypertension treated by calcium channel blockers; she was also under sodium valproate for the seizures.

The preoperative checkup did not diagnose any problem, in particular the preoperative ECG and the other laboratory tests, blood ionogram, the renal function, the glycemia were all normal.

On admission to the operating room, the patient's hemodynamic status was stable. Anesthesia was induced with Propofol (100 mg) and fentanyl (200 µg/), and for intubation rocuronium was added (50 mg). The anesthesia was maintained with sevoflurane (1.2-

1.5%). Twenty five (25) minutes after the surgical incision, 200 ml of 20% mannitol (40 g) was administered intravenously over 60 min.

Approximately 20 min after the end of mannitol administration, a Ventricular Tachycardia occurred (Figure 1).

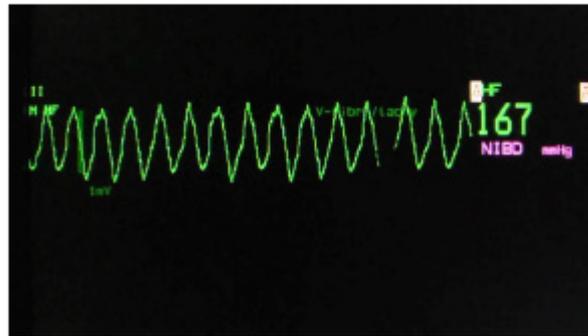


Figure 1: ECG monitor showing ventricular tachycardia at 167 bpm.

At this time, in the operative field, the arachnoid membrane of the Sylvian fissure was separated. Blood pressure decreased to <40 mmHg. We immediately started the chest compression, and the VT recovered to sinus rhythm at a rate of 75 bpm approximately 20 s after the start of the chest compressions. After recovery of sinus rhythm, ECG did not show noticeable abnormalities. Analysis of arterial blood gases showed pH 7.38, PaCO₂ of 40 mmHg, PaO₂ of 350 mmHg, ionogram revealed hyperkalemia of 5.5 mmol/L.

The glucose serum was administered with 10 units of insulin. The control value of kalemia was 3.6 mmol/L. Tumor resection was performed without any further incident nor hemodynamic instability. At the end of the operation, no abnormalities were

described in the ECG and laboratory tests. The duration of the surgical intervention was 4 h 20 min the estimated blood loss during the operation was 300 ml.

The urine output was 2000 ml. The patient had no other postoperative event and was admitted to intensive care for postoperative care. The patient was extubated after full recovery.

Discussion

There are a few reports of fatal arrhythmia, Ventricular Tachycardia and / or ventricular fibrillation (Figure 2) due to mannitol, which are mainly due to hyperkalemia [1,2]. The ECG is then the main tool to predict the event on the appearance of arrhythmias.

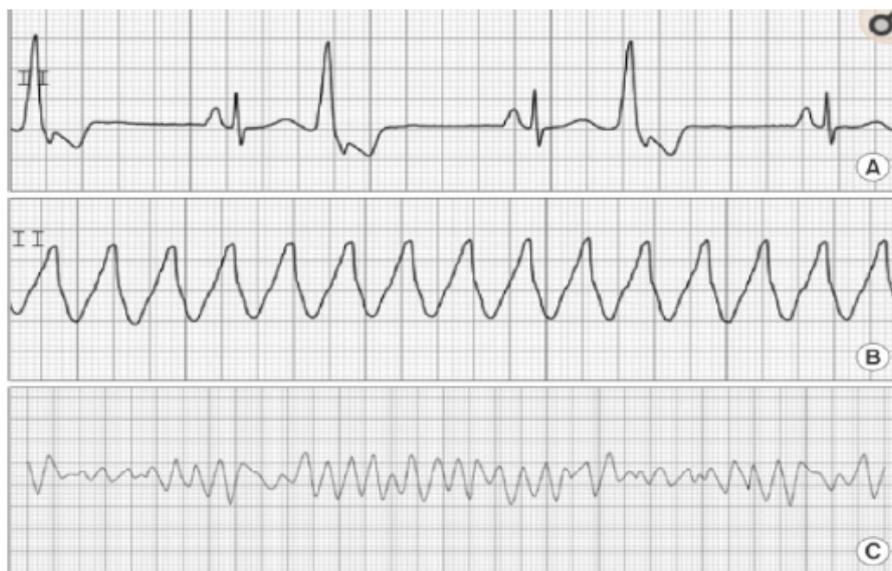


Figure 2: Difference between Premature Ventricular Contractions (A), Ventricular Tachycardia (B) and Ventricular Fibrillation in ECG (C).

Mannitol is widely used to reduce intracranial pressure due to increased osmolality of plasma. However, it has several side effects including osmotic nephrosis and electrolyte disorders [3]. Rapid infusion of mannitol may cause hyponatremia or hyperkalemia. Hyponatremia is mainly due to transient dilution. During mannitol-induced hyperkalemia, intracellular potassium (K) is released into the extracellular space due to an acute increase in plasma osmolality, rhabdomyolysis, hemolysis, and acidosis. In this case, preoperative renal function was normal and there was no evidence of acidosis, rhabdomyolysis or hemolysis. The frequency of mannitol-induced hyperkalemia is 7.7%, and the total dose of mannitol as well as its rate of infusion and decreased renal function may have a role in the development of mannitol

induced hyperkalemia [4]. We used a total dose of 40 g of mannitol, which was the recommended dose (0.25-1 g/kg) [5], And the infusion rate was 60 minutes, which was not rapid. In most of the published cases, the change in the ECG - elevation of T waves - was the main finding.

All cases of lethal arrhythmia reported so far have been preceded by a change in the ECG, including elevated T waves and a wide QRS complex. However, no prior ECG changes were observed in our patient, and even after returning to sinus rhythm after VT. Regarding ECG changes in hyperkalemia, such as elevation of T wave (temporary T wave), decrease of P wave, prolongation of PQ interval due to an increase in the kaleemia the extension of the width of the QRS, the disappearance of the P wave and its subsequent increase which leads to an unclear distinction between the QRS and T waves, leading to the cessation of activity cardiac mechanics [6].

However, an ECG without any abnormalities consistent with hyperkalemia is noted in more than 50% of patients with a K^+ > 6.5 mmol/L, and cases of arrhythmia and cardiac arrest have been reported in patients with of hyperkalemia without ample T wave [7,8].

Ventricular tachycardia should be quickly detected by the anesthetist on the ECG Cardioscope and propose the adequate protocole. The immediate task is to correct any reversible etiology that can include (Hypotension, Hypoxemia, Acidosis and other electrolyte imbalances, Surgical stimulation, light anesthesia)

then to propose rapid defibrillation. Concerning pharmacological interventions, AHA recommends the use of Amiodarone or Lidocaine in Ventricular tachycardia and Ventricular Fibrillation resistant to defibrillation [9].

Conclusion

In our case It is not clear why the VT occurred, but in addition to the transient elevation of potassium and mannitol, the craniotomy itself may have influenced it. Evidence is rare when dealing with cardiac arrest in neurosurgery, and more efforts are needed in understanding the etiologies, mechanisms and the specificities of its management.

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