Persistent Ventricular Tachycardia After Tracheal Intubation

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Abstract

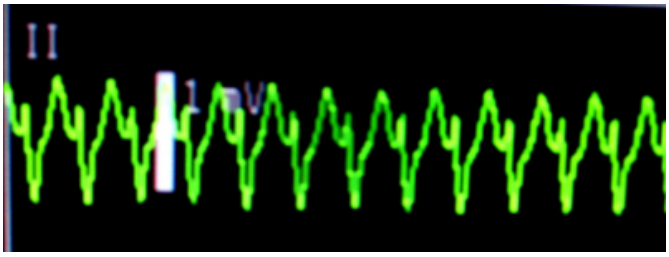
To the Editor,

Numerous reports indicate that cardiovascular disturbances during endotracheal intubation do occur. Takeshima et al mentioned in their article that majority of investigators reported transitory electrocardiographic (ECG) changes during tracheal intubation ([1]). These include changes in the direction of the electrical axis, sinus bradycardia, sinus tachycardia, auricular fibrillation, nodal rhythm, heart block, ventricular tachycardia, premature ventricular contractions, depression of P wave, increase or shortening of the P-R interval, decreased QRS voltage, shortening or prolongation of the Q-Tc interval, decrease in voltage of the T wave and depression on the ST segment ([1]).

We report a case of a 55-year-old male, weighing 60 kgs undergoing elective craniotomy for right temporo-parietal glioma. His medical history was unremarkable and routine investigations within normal limits. Inside the operation theatre, besides routine monitoring equipments, bispectral index (BIS, Aspects medical system) and arterial pressure monitoring was also started. General anesthesia was induced with thiopentone sodium 300 mgs and fentanyl 150 mcg. Rocuronium bromide 60 mg was used to facilitate tracheal intubation. The BIS value at the time of laryngoscopy was 42 indicating adequate anesthetic depth.

The duration of laryngoscopy and intubation was less than 15 seconds. Immediately after intubation, the ECG showed supraventricular tachycardia that soon converted to ventricular tachycardia. Lignocaine 100 mg was given intravenously. Within two minutes the cardiac rhythm returned to normal. The anesthesia was maintained with isoflurane (MAC 1.0) in a mixture of nitrous oxide and oxygen (2:1) along with boluses of fentanyl and vecuronium bromide as required. However, five minutes later, the normal ECG pattern again converted to ventricular tachycardia.

Figure 1



Following a bolus injection of lignocaine 100 mg, the infusion of the same was started at the rate of 1 mg/kg/hr. Arterial blood gas analysis and blood electrolytes at this stage revealed normal values. As the patient remained hemodynamically stable after initiation of lignocaine infusion, no other active intervention was done and surgery allowed to continue. The rest of the intraoperative course was uneventful. At the end of the surgery the neuromuscular block was reversed and trachea extubated. The patient was shifted to the neurosurgical intensive care unit. A 12-lead ECG and echocardiogram was done which showed normal studies. The patient was discharged five days later, neurologically intact and after thorough cardiac evaluation that was normal.

The pressor response to laryngoscopy and endotracheal intubation contributes to the electrocardiographic changes that are usually transitory. However, we encountered changes in the cardiac activities that were not transient but persisted for longer duration than expected, after intubation and requiring infusion of lignocaine for controlling the dysrhythmias. The pressor response during tracheal intubation can be either sympathetic ([2]) or parasympathetic mediated ([3]). The sympathetic response may be in the form of increased blood pressure and heart rate and various

tachyarrhythmias. Physiologic effects of parasympathetic stimulation result in decreased rate of discharge of sinoatrial pacemaker fibers, prolongation of atrioventricular conduction velocity, and heart block along with bradyarrhythmias.

Factors like hypoxia, hypercarbia, anaesthetic overdose, electrolyte imbalance and myocardial ischemia as probable causes of this arrhythmia were ruled out in our case. Even though our patient was premedicated with glycopyrrolate 0.2 mg intramuscularly one hour prior to surgery, the complication could not be prevented. Techniques that have been shown to modify the pressor response should be considered. Since none of the pharmacological approaches to blunt pressor response have proved entirely satisfactory, a logical approach would be to minimize stretching of the tissues of epipharynx and laryngopharynx. Our report emphasizes on the severity of the complication that can occur due to tracheal intubation. It also suggests the vigilant role and timely intervention by the anesthesiologist.

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