Ventricular bigeminy during balloon occlusion of carotico-cavernous fistula

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Sir,

A 12-year-old, 39 kg American Society of Anaesthesiologists (ASA) grade I, male patient presented with proptosis of right eye ball since 6 months and tinnitus in right ear since 2 months. He had a history of head injury 8 months ago. He did not have any signs or symptoms of cardio-respiratory abnormality and his systemic and haematological examination were normal. He was diagnosed to have right carotico-cavernous fistula (CCF) and was scheduled for balloon occlusion of right CCF under general anaesthesia. In the neuroradiological suite pulse oximeter, Electrocardiography (ECG) and non-invasive blood pressure were attached. A standard anaesthetic technique using propofol, fentanyl and rocuronium for induction followed by sevoflurane and nitrous oxide for maintenance via laryngeal mask airway (LMA) was used. Following infiltration with 2% lignocaine, femoral access for the procedure was secured. The patient had stable haemodynamics for initial 45 minutes. However, during the inflation of balloon in the cavernous sinus, sudden bradycardia (heart rate decreased to 35 beats per minute from the initial value of 92 beats per minute) with hypotension (Blood Pressure 86/47 mmHg from the initial value of 126/78 mmHg) and the ECG showed ventricular bigeminy (VB). The neuroradiologist was informed but brief interruption of the procedure could not correct the arrhythmia and hypotension. The bradycardia and hypotension responded to 0.6 mg of intravenous (IV) atropine but the VB persisted. The arterial blood gas analysis was normal and there were no signs of inadequate depth of anaesthesia or hypoxemia. To treat the VB, we administered 2% lignocaine 1.5 mg/kg followed by infusion @ 2 mg/min. The VB resolved within next 10 minutes. Rest of the procedure was uneventful and the patient was shifted to Intensive Care Unit (ICU) further management.

Balloon occlusion is one of the therapeutic options for CCF.[1] The trigemino-cardiac reflex (TCR) is defined as the sudden onset of parasympathetic dysrhythmia, sympathetic hypotension, apnoea or gastric hypermotility resulting from stimulation of receptors on trigeminal afferent fibres including those of the peripherical branches.[2] The different risk factors for TCR are hypercapnia, hypoxia, lighter plane of anaesthesia or drugs (beta blockers, calcium channel blockers or narcotics like sufentanil and alfentanil).[2] In our case, the patient did not have any risk factors for TCR or VB. Stimulation of receptors in the ophthalmic and maxillary division of trigeminal nerve situated in the lateral wall of cavernous sinus can initiate TCR during neurointerventions for CCF and dural arteriovenous fistula[3] and VB can occur during oculo-cardiac reflex,[4] which is a variant of TCR. Ventricular bigeminy is a cardiac arrhythmia characterized by the occurrence of a normal heartbeat followed by a premature ventricular contraction. Sudden onset VB has an increased potential for ventricular fibrillation,[5] and hence should be treated promptly. Lignocaine (1.5 mg/kg followed by 1-4 mg/min, if needed) is the treatment of choice for VB which is unresponsive to correcting the underlying abnormality.[5] To the best of our knowledge, this is the first report of ventricular bigeminy resulting due to TCR during balloon occlusion for CCF. With this case report, we want to emphasise upon the fact that VB can occur as a part of TCR during balloon occlusion of CCF and may require lignocaine for resolution.

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