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Disappearance and Reappearance of Supraventricular Arrhythmia during Aneurysmal Surgery-the Pressure Effect

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Dear Editor,

Electrocardiographic (ECG) abnormalities and rhythm disorders are frequently (40–100%) observed in the acute phase after spontaneous subarachnoid haemorrhage (SAH) (1). These abnormalities are usually benign and transient, and are attributed to catecholamine surge secondary to increased intracranial pressure (ICP). Spontaneous reversion mostly occurs once the ICP is controlled (2).

A 65-year-old female, with known history of hypertension, presented to us with ruptured anterior communicating artery aneurysm with SAH and intraventricular haemorrhage. She had encountered an episode of sudden severe headache four days back and was admitted in a private hospital for three days. However, her sensorium worsened on fourth day and a repeat CT scan of brain showed expansion of haematoma. She was then transferred to our hospital. Because of poor neurological status, her trachea was intubated (Glasgow Coma Scale- E2V2M5). She was categorised as modified Hunt and Hess Grade IV, modified Fisher Grade 4, and World Federation of Neurological Surgeons' Grade IV after complete evaluation. Her preoperative ECG showed supraventricular arrhythmia (heart rate variability [HRV] between 80 and 95 beats min^{-1}), blood pressure (BP) being 150–155/75–80 mmHg. Her blood sugar and serum electrolytes were within normal limits. She was thus taken for emergency clipping of aneurysm. During the initial stage of craniotomy, her ECG rhythm was similar to that in the preoperative period, with HRV between 75 and 90 beats min^{-1}. However, once the duramater was opened, the ECG spontaneously reverted to sinus rhythm (HR between 84 and 86 beats min^{-1}) without change in BP, and the entire period of dissection and clipping showed normal sinus rhythm in ECG. Strangely, ECG arrhythmias appeared again (similar to preoperative period) after the dural closure, and persisted till the end of surgery and for another 48 h in the ICU. No medication was advised by the cardiologist. From the third postoperative day and, subsequently, her ECG rhythm showed sinus pattern.

Existing literature clearly mentions that the incidence of ECG abnormalities correlates with the amount of intracranial blood and SAH grading severity (3). Moreover, changes in HRV after SAH reflect both delayed ischemic and infectious complications. However, no evidence exists that the prophylactic administration of a β-adrenergic or other autonomic antagonist significantly alters the outcome in such patients, and the use of these agents for this purpose is not warranted (4). In our case, since emergency surgery was the need of the hour, it was not delayed by further investigating the arrhythmia. Moreover, it was not associated with haemodynamic instability. The probable hypothesis behind disappearance of arrhythmia after opening of dura could have been the decrease in ICP to the atmospheric level (referred to as zero), and consequently, the downregulation of catecholamine levels and myocardial stunning (5). Additionally, we had administered mannitol at the time of craniotomy, which possibly contributed in lowering ICP. The reverse mechanism might have attributed to the reappearance of arrhythmia after dural clo-

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sure. Thus, it is prudent to closely observe ECG changes in nontraumatic SAH and primary management should revolve around controlling ICP.

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