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Prolonged Sympathetic Storming Caused by Spontaneous Subarachnoid Haemorrhage

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Introduction

The pathophysiology of sympathetic storming has been refined since it was originally described in 1929, and it was initially thought to be resulted from epileptic discharges from thalamic nuclei. However, based on EEG studies and the current model, it is recognized that the interruption of autonomic pathways results in an imbalance between sympathetic and parasympathetic nervous systems, which leads to PSS. There is no clear pattern of injury that increases the likelihood of sympathetic hyperactivity. However, it is more common in patients with diffuse axonal injury. Sympathetic storming occurs most frequently in patients with traumatic brain injury. However, this unique phenomenon has also been associated with hypoxic injury, brain tumors and hydrocephalous [1]. Although excessive release of catecholamine and increased sympathetic activities resulting in cardiac and pulmonary manifestations have been well reported in subarachnoid haemorrhage, for example, myocardial infarction and hypertension, there was no report with specific description of paroxysmal sympathetic storming in spontaneous SAH found after extensive literature search. Here, we report a case in which a patient developed prolonged sympathetic storming after a spontaneous subarachnoid hemorrhage secondary to a posterior communicating aneurysm rupture [2]. An urgent ventriculostomy was performed as well as craniotomy with posterior aneurysmal clipping. Despite Nimodipine for vasospasm prevention, the patient developed episodes of vessel spasms and increased intracranial pressure. Due to these complications, the patient was placed on a hypothermic protocol with paralytics. Mannitol and 3% sodium chloride were started for ICP elevation. Levetiracetam was added for seizure prophylaxis [3]. After his ICP was normalized, the patient was weaned off all paralytics and rewarmed from the hypothermia protocol on day 16 of hospitalization. Overnight on day 16 of hospitalization, however, the patient developed acute onset of episodic tachycardia up to 200, hypertension with SBP up to 220, increased respiratory rate into the mid 30's, and hyperthermia with a temperature of 101 F. Meanwhile, he had severe diaphoresis, tremor, and spontaneous extensor posturing. All of this occurred with a normal ICP and no change in gag reflex or pupil size. The clinical episodes were consistent with sympathetic storming. He was given Labetalol IV 10 mg twice, and his tachycardia, hypertension, posturing, and tremor quickly resolved within several minutes while his hyperthermia and diaphoresis improved gradually [4]. This rapid response to Labetalol treatment confirmed a diagnosis of sympathetic storm. There was no evidence indicating possible acute onset infection, hyperthyroidism, pheochromocytoma, or hypercortisolism. Despite this initial treatment, similar episodes recurred with frequency of 3–6 episodes/day with His storming symptoms completely resolved on day 46 of his hospitalization. Although not fully oriented, the patient resumed normal spontaneous movement of extremity and eye opening with the ability of simple command following. His Bromocriptine and Propranolol were then switched to PRN and later discontinued. He was then discharged to nursing facility with 24-hour care [5]. Although the general clinical presentation of paroxysmal sympathetic storming has been well recognized for nearly a century, the syndrome has not been described in a non-traumatic subarachnoid haemorrhage [6]. Most well recognized cases of sympathetic storming have been identified in patients who have suffered traumatic brain injury, brain tumors, aqueductal stenosis, or cardiac arrest. Subarachnoid hemorrhage is associated with significant catecholamine elevation and marked sympathetic activation, which has been linked to cardiopulmonary complication other than PSS, such as neurogenic stress cardiomyopathy, arrhythmias, neurogenic pulmonary edema, and neurogenic myocardial injury. While numerous studies have described the above conditions in SAH, in our extensive electronic literature search for English-language articles on neurological and cardiopulmonary complications of SAH up to date, no original article was found reporting the direct association of paroxysmal sympathetic storming with spontaneous SAH [7]. This paper presents the description of PSS caused by spontaneous SAH with a prolonged hospital course that involved comprehensive management of this syndrome. It is essential to distinguish PSS from sympathetic activation-induced cardiopulmonary effects, as their manifestation, mechanism, and treatment are different [8]. While hypertension and cardiac injury are relative common complications of SAH and are a result of catecholamine elevation, PSS is rare and manifested as episodic tachycardia, hypertension, tachypnea, hyperthermia, dystonia, posturing, and diaphoresis in cycles. Instead of simple sympathetic activation, PSS is thought to be caused by the imbalance between sympathetic and parasympathetic nervous systems. The mechanism contributing to its paroxysmal nature is not clear although certain external noxious stimuli may act as triggering factors. As in this case, the initial vasospasm and transiently increased ICP may be triggering factor and the signs of PSS might have been masked by hypothermia initially as storming symptoms started right after rewarming, days after his ICP was normalized [9]. In this case, although PSS symptoms promptly responded to non-selective β-blocker Labetalol upon onset, the episodic relapses did not dissipate till scheduled Propranolol and Bromocriptine were started in addition to sedative agents and the external stimuli including vital and neurologic checks were minimized to medical necessity. Therefore, we think that the scheduled Bromocriptine and Propranolol are much more effective in inducing complete resolution of PSS symptoms. This patient's sympathetic storming did not start until he was weaned from the hypothermia protocol, this frequent association between PSS and rewarming as well as weaning of sedatives and paralytics has been well reported.

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This raised question for potential prophylaxis of PSS in these high-risk patients with medication that has minimal effect on hemodynamics such as scheduled Bromocriptine [10]. The potential prevention of PSS in critically ill patients may significantly reduce related complications, increase their survival, and shorten their ICU stay.

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Conflict of Interest

None

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