Non-infectious fever in cerebral arteriovenous malformation: Central fever or paroxysmal sympathetic hyperactivity

Sir,

Fever is defined as a core body temperature exceeding 38°C.[1] Non-infectious fever is considered in patients without any evidence of infection or drug fever. We report two cases of non-infectious fever observed

during the management of cerebral arteriovenous malformation (AVM) patients in the neuro-intensive care unit. Central fever (CF) results from a disturbance of thermoregulation[2] which involves complex interactions of thermoreceptors, spinal cord, midbrain and hypothalamus.[3] Injury to the frontal lobe, hypothalamus, basal ganglia, thalamus and presence of blood in the ventricles are the risk factors of CF.[3] Paroxysmal sympathetic hyperactivity (PSH) is a clinical syndrome associated with acute brain injury, characterised by tachycardia, hypertension, tachypnoea, hyperthermia, diaphoresis and dystonic posturing because of episodes of unbalanced sympathetic surges.[4] Often PSH is associated with traumatic brain injury (79.4%), hypoxic brain injury (9.7%) and cerebrovascular accident (5.4%).[4] Association of CF and PSH with cerebral AVM is seldom reported in the literature.[5]

In our first case, a 26 years old male patient, with residual right parietal AVM was posted for elective embolisation. Post-intervention computed tomography (CT) of the brain [Figure 1] revealed right frontal hematoma with mass effect, which warranted an emergency evacuation. Post-evacuation Glasgow Coma Scale (GCS) was E1VTM2. On the 6th postoperative day (POD) his nasopharyngeal temperature was 38.3°C. Chest radiograph, blood investigations, serum procalcitonin level and cultures (tracheal aspirate, blood and urine) were unremarkable. On POD-9 episodes of tachycardia, hypertension, tachypnoea and motor posturing along with fever were noticed. Ultrasonography of abdomen ruled out foci of infection and venous Doppler of limbs ruled out deep venous thrombosis. Hence all antibiotics were stopped and the patient was prescribed with oral propranolol, intravenous morphine and baclofen for spasticity. The following day temperature reduced to below 37.7°C and he was gradually weaned off from mechanical ventilation.

In our second case, a 37 years old female admitted with altered sensorium with a GCS of E3V1M5 with subsequent CT brain revealed right frontal lesion with oedema, hydrocephalus and midline shift of 10 mm, for which emergent ventriculoperitoneal shunt was done. Diagnostic angiography revealed right basifrontal AVM. The venous pouch was seen in right basal ganglia and posterior thalamus region. Later, the patient developed high grades of fever for which second-generation cephalosporin and aminoglycoside were prescribed. On POD-3, patient underwent decompressive craniectomy. All cultures including the cerebrospinal fluid were sterile and serum procalcitonin was unremarkable. The nasopharyngeal temperature was of high-grade, reaching up to 40°C. Oral propranolol twice daily was prescribed, following which, febrile episodes reduced and the temperature never crossed 37.7°C. A diagnosis of CF was made by exclusion.

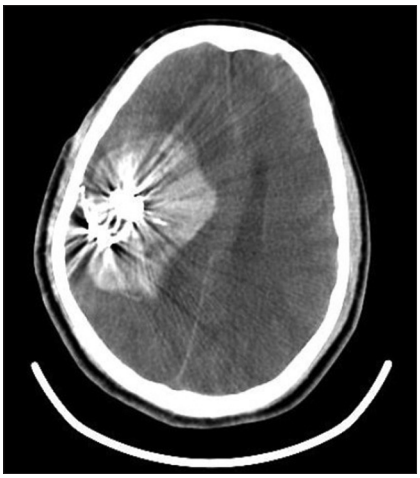

Figure 1: Post embolisation computed tomography scan showing right-sided temporoparietal haematoma with embolised artefacts with mass effect

In this case series, we describe two different presentations of non-infectious fever in a similar spectrum of illness. In the first case, the likely diagnosis was PSH, whereas in the second case the likely diagnosis was CF. The PSH assessment measure (PSH-AM), based on clinical feature, severity and diagnostic likelihood tool helps in identifying PSH.[6] In the first case, fever with hypertension, tachycardia, tachypnoea and motor posturing persisted for more than three consecutive days, thus increased the likelihood of PSH. However, in the second case, the typical episodes of PSH were not observed and high-grade fever early in the course of illness pointed more towards CF. In both conditions, management involves controlling the symptoms. In PSH, pharmacological agents such as non-selective β-blockers like propranolol, centrally acting α-2 agonists namely clonidine, dexmedetomidine and morphine are usually prescribed.[3] As a protocol, we start propranolol and morphine to abort the episodes. The decision to add baclofen depends on the severity of spasticity. In CF antipyretics such as acetaminophen and ibuprofen are often prescribed along with non-invasive and invasive cooling. In CF, many drugs such as morphine, baclofen and propranolol have successfully been tried.[2] CF is a diagnosis of exclusion,[2] but early recognition and treatment is the key to reduce the poor outcome. Although CF, and PSH, have different pathophysiology, the line of diagnosis and treatment of these remain the same.

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