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Idiopathic Postoperative Paresis: Know The Unknown

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Case Report(s)

Postoperative paresis can be frustrating when aetiology is unclear. Herein, after obtaining written and informed consents, we are presenting perioperative hospital course of two patients with â€œidiopathicâ€• postoperative paresis.Â

Case One: 31-year-old multigravida female presented to labour and delivery suite in active labour. Patient received continuous-infusion labour epidural analgesia for sixteen hours and delivered vaginally, three hours after rupture of membranes. On postoperative day one, patient began complaining cramping lower body pain interfering in ambulation efforts. On postoperative day two, patient completely stopped lower limb movements. Metabolic panel (sodium, potassium, calcium) was normal. Neurology was consulted. Per neurology, bilateral hip flexion/extension/abduction/adduction was 3/5 and bilateral knee flexion/extension was 4/5 with bilateral patellar/ankle reflexes as 3+ extensor responses. Tenderness was noted but tone/touch/sensation/bowel/urinary functions were normal. Urgent magnetic resonance imaging ruled out suspected radiculomyelopathy/paraparesis/cord compression/myelitis. Per physical therapy team, patient seemed distracted in initiating efforts to ambulate. Per psychiatry consult, patient was not malingering. As patient refused sub-acute rehabilitation facility admission as recommended by physical therapy team, patient was discharged home on postoperative day eight after demonstrating improvements in ambulation efforts with assisted mobility on cane/walker and sustained pain-relief on acetaminophen, ibuprofen and cyclobenzaprine.

Case Two: 54-year-old male presented for robotic-assisted laparoscopic ventral herniorraphy in steep Trendelenburg position which was intraoperatively converted to open ventral hernia repair with mesh placement. On postoperative day one, neurology was consulted for new-onset left-sided weakness (2-3/5), sensory deficits with facial droop and reflex 2+ flexor responses. At this time, patient elaborated on past history about nocturnal seizures complicated by Toddâ€™s paresis. Magnetic resonance imaging head was unremarkable. Metabolic panel (sodium, potassium, calcium, magnesium, phosphorus) was normal. On postoperative day two, left-sided weakness improved (4/5) with normal muscle tone and dull sensations. Transthoracic echocardiogram demonstrated delayed appearance (after eight beats) of bubble contrast in left atrium suggesting small pulmonary arterio-venous malformation. On postoperative day three, patient was discharged home. However, on postoperative day six, patient returned to emergency department for evaluation of recurrent seizures and Toddâ€™s paresis secondary to non-compliance with carbamazepine due to its side effects. Computed tomography head and electroencephalogram were within normal limits. Patient was started on sodium valproate and discharged from emergency department to follow up with outpatient neurology.

Both cases eventually unravelled common causes as contributors to â€œidiopathicâ€• postoperative paresis. In first case, patientâ€™s unrelenting lower body pain interfered with patientâ€™s efforts to move thus presenting as postoperative paresis until postoperative pain control was ensured and postoperative physical therapy was initiated [1-2]. In second case, Toddâ€™s paresis happened after postoperative seizures, although postoperative seizures are uncommonly reported after non-intracranial surgeries [3]. In first case, we had additionally considered upright pelvis skiagram to elucidate whether exaggerated symphysis pubis dysfunction/separation due to pregnancy was a potential cause for pain [4]. Similarly, in second case, we had considered investigating pulmonary arterio-venous malformation with transesophageal echocardiography with bubble contrast/pulmonary angiogram/transcranial Doppler with/without bubble contrast as a potential cause for recurrent cryptogenic stroke-like symptoms after seizures [5-9]. Additionally we had considered theorizing and exploring whether post-ictal paralysis can be explained by seizures creating a Valsalva-like physiology/phenomenon inducing increased intra-pulmonary shunting and spontaneous gas embolism across the shunt leading to prolonged paralysis after seizures almost as if maybe seizures creating nitrogen bubbles on the lines of decompression sickness due to pressure changes happening within the body systems due to a Valsalva-like physiology/phenomenon during seizures [10-13].

Summarily, the â€œidiopathicâ€• aetiology-pathogenesis warrants exploring/delving into common/rare causes/diagnoses underlying such â€œidiopathicâ€• presentations.

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