Recurrent high-pressure supratentorial subdural collections following microvascular decompression: A multifactorial process involving altered cerebrospinal fluid dynamics and anticoagulation - A case report

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ABSTRACT

Introduction: Microvascular decompression (MVD) is the gold-standard surgical treatment for trigeminal neuralgia and is generally associated with low morbidity. While recognised complications are typically confined to the posterior fossa, remote supratentorial haemorrhagic complications following infratentorial surgery are rare. Among these, recurrent supratentorial subdural collections are exceptionally uncommon and poorly understood. Proposed mechanisms include intracranial hypotension following cerebrospinal fluid (CSF) drainage, bridging vein traction, altered CSF circulation, and age-related cerebral atrophy. **Case Presentation:** A 76-year-old male with medically refractory trigeminal neuralgia who underwent elective right-sided retrosigmoid craniotomy and MVD. Preoperative CT demonstrated no subdural pathology but showed generalised cerebral atrophy. Serial postoperative CT imaging, operative findings, microbiological results, and clinical progression were reviewed narratively to characterise the temporal evolution of the subdural collections and explore plausible mechanisms of recurrence. The focus was placed on radiological progression, fluid characteristics at repeat evacuations, associated systemic complications, and later CSF diversion requirements. Within 24 hours of surgery, the patient developed acute neurological deterioration with dense left hemiplegia. CT imaging demonstrated a 17 mm right supratentorial subdural collection causing 9 mm midline shift and subfalcine herniation. Urgent burr-hole evacuation yielded high-pressure "machinery oil" fluid, consistent with chronic subdural haematoma. Despite initial improvement, serial imaging demonstrated persistence and early recurrence. Further deterioration was associated with reaccumulation to 24 mm, 15 mm midline shift, ventricular compression, and transependymal oedema, necessitating repeat evacuation. Intraoperative findings at second surgery revealed lower-viscosity dark red-brown fluid, suggesting progressive evolution of the subdural process. Subsequent imaging showed further recurrence up to 21 mm with recurrent mass effect before gradual evolution into a low-density CSF-like subdural collection. The postoperative course was further complicated by segmental and subsegmental pulmonary emboli requiring therapeutic anticoagulation, likely contributing to persistence and reaccumulation. The patient later developed hydrocephalus requiring ventriculoperitoneal shunt insertion, supporting a broader disturbance in CSF homeostasis. Microbiological cultures were negative aside from an initial likely contaminant. **Conclusion:** This case illustrates a rare but life-threatening complication of posterior fossa surgery characterised by recurrent, high-pressure supratentorial subdural collections with evolving fluid characteristics. The findings support a multifactorial mechanism involving initial bridging vein injury from intracranial hypotension, subsequent chronic subdural membrane physiology, possible arachnoid breach with CSF ingress, impaired CSF resorption, therapeutic anticoagulation, and age-related cerebral atrophy. The later development of hydrocephalus further reinforces the role of global CSF dysregulation. Early recognition, serial neuroimaging, cautious anticoagulation decisions, and a low threshold for repeat surgical intervention are essential. This case expands the limited literature by demonstrating progression from haemorrhagic chronic subdural fluid to CSF-density recurrent collections, suggesting a self-perpetuating subdural space process rather than an isolated postoperative bleed.