

Clinical Notes

Suspected fatal pulmonary embolism following application of intermittent pneumatic compression during cranial surgery

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The optimal prophylaxis against venous thromboembolism (VTE) in patients undergoing neurosurgical procedures remains controversial. Over the years the subject has motivated several studies however, the results were frequently conflicting. Hence, practices still vary widely and appear to be more influenced by the power of a recent anecdotal morbidity as much as of convincing scientific evidence. Many neurosurgeons fear the use of pharmacological prophylaxis early after intracranial surgery as it may increase the risk of intracranial hemorrhage (ICH). More than half of neurosurgeons prefer to use mechanical prophylaxis with elastic stockings (ES) and intermittent pneumatic compression (IPC) devices as they are safe even though they might not be highly effective as a sole preventative therapy.^{1,2} Massive intra-operative pulmonary embolism (PE) occurring after the application of an Esmarch bandage, a device that is used to exsanguinate the limb to achieve a bloodless operative field in orthopedic surgery, is well reported in the literature.³ In this article, we reported a patient that died of a suspected intra-operative PE, which occurred following the application of an IPC device, a complication that is not well documented in the literature. It is hoped that the presentation of this case will serve to remind clinicians of the necessity of preoperative VTE prophylaxis in high-risk patients, and to make them aware of the possibility of PE occurring after the application of an IPC device.

A 57-year-old woman presented to our neurosurgical unit with a 4 months history of headache, forgetfulness, and occasional incontinence. She became weak and unsteady on her feet, however, she was still able to ambulate independently. Her past medical history included hypothyroidism, hypertension, osteoporosis, and rheumatoid arthritis. On examination, she was fully conscious with mild papilledema and normal visual acuity. Her gait was ataxic, and she had no limbs paresis. Computerized tomography (CT) of the brain (Figure 1) demonstrated a 7 cm midline, well circumscribed, contrast enhancing anterior basal lesion, most likely an olfactory groove meningioma with peritumoral edema. Carotid angiography confirmed that the tumor was considerably vascular. Her preoperative medical work up included thyroid function tests, ECG, CT chest, echocardiography, and pulmonary function test, all of which did not reveal any findings that would have suggested a pre-existing PE such as vascular filling defects, right heart strain, and diffusion abnormalities. She was started on dexamethasone and phenytoin, fitted with ES, and was encouraged to ambulate. She



Figure 1 - Computerized tomography of the brain showing a well-circumscribed contrast enhancing bifrontal basal tumor (arrowed) most likely an olfactory groove meningioma with peritumoral edema.

was taken to the operating room 4 days after admission. After induction of anesthesia, an IPC device was applied on her calves and thighs, which were not noted to be swollen or warm. A bi-coronal scalp flap and a bifrontal craniotomy were started. Twenty minutes after the start of the operation, the patient's blood pressure suddenly deteriorated from a systolic of 120 mm Hg to a systolic of 60mm Hg with a drop in oxygen saturation to 50%, and in the end tidal carbon dioxide pressure to 10 mmHg. Attempts to resuscitate the patient by giving her more fluids, oxygen, inotrope and blood failed to improve her condition. The ECG showed S1Q3T3 pattern suggestive of a PE, which was not evident on the preoperative ECG. She quickly worsened to a state of asystole. All efforts at cardiac resuscitation failed, and she was pronounced dead.

Deep venous thrombosis (DVT) is perhaps the most significant preventable cause of morbidity and mortality in today's neurosurgical patient population. Although the lack of standard definition for DVT (clinically silent or symptomatic) and differing screening methods make interpretation of the current research difficult, average estimates place the incidence of DVT in neurosurgical patients at approximately 25%. The DVT in isolation is not life threatening, but between 1.5 and 5% of patients with DVT developed PE, a condition that is fatal in 9-50% of patients.^{1,2} Patients with brain tumors have among the highest VTE rates of all cancer patients.¹ The propensity towards clotting in these patients has been attributed to both clinical and biochemical factors including prolonged operative times, hemiparesis, non ambulation and release of brain-derived tissue factor, which may cause chronic, low grade disseminated intravascular coagulation. Other reported risk factors include advancing age, tumor size, grade, location, and use of corticosteroids.^{1,4} Among intracranial tumors, meningioma is associated with a high risk rates of VTE as high as 70%.⁴ Such patients can be at risk of a thrombotic event not only postoperatively and also at any time throughout their clinical course.¹ Hence, it

is important for the neurosurgeon to remember that a form of VTE prophylaxis is required for all meningioma patients as soon as they are admitted to the hospital, particularly if they have other risk factors. It is also important to ensure that these patients ambulate regularly before the operation and early postoperatively.⁴ Most neurosurgeons are likely to recommend the application of ES preoperatively and the use of an IPC device during the operation and in the early postoperative period.⁴ ES and IPC are popular for the neurosurgical patient population as they are perceived as safe and well tolerated, not only because they reduce VTE by up to 50%.¹ In addition, the miniaturization, and weight reduction of the IPC devices led to more acceptances by patients.² Pharmacological prophylaxis in the form of unfractionated or low molecular weight heparin (LMWH) is effective and if given with mechanical prophylaxis can provide an additional 38% relative VTE risk reduction.⁴ However, for such benefit to be observed, the heparin has to be given early, within 18-24 hours postoperatively, as it has been shown that when the pharmacological prophylaxis is delayed until 48 hours after surgery, 25% of clotting events occur before the first dose is administered.¹ Conversely, prescribing the heparin perioperatively may increase the risk of bleeding.

In a study in which enoxaparin 30mg was administered subcutaneously to brain tumor patients every 12 hours, with the first dose given before surgery, 11% of patients sustained a significant ICH before a second enoxaparin dose was administered and the study had to be terminated.⁵ In addition, studies of spinal anesthesia have demonstrated increased bleeding complications when enoxaparin is administered within 24 hours before the procedure.¹ Other authors however, reported a lower postoperative bleeding rate of approximately 3% when the pharmacological prophylaxis is started within 24 hours of intracranial surgery.^{4,5} It seems that up to now there is no consensus on what constitutes an acceptable risk when giving heparin peri-operatively, or within the first 24 hours postoperatively. This is because even a low rate of postoperative ICH, still highly significant. In addition, the impact of an ICH in anti-coagulated patients is singularly catastrophic and can be associated with an approximately 45% mortality rate.¹⁹

We believe that the intraoperative clinical course of our patient supports the diagnosis of a massive PE, even though the findings could have been caused by a tension pneumothorax, or a myocardial infarction. Unfortunately, the patient deteriorated very quickly and she was unstable for a CT scan. In addition, transesophageal echocardiography was not available, and an autopsy was not possible. We feel that the timing of the suspected PE, which was 20 minutes after the application of the IPC device, was more than a coincidence. We believe that the patient, who did not have clinical evidence of DVT, may have had a silent DVT, which dislodged after the IPC was started causing the PE. Such development is recognized with the use of Esmarch bandage during orthopedic surgery.³

The unexpected events in our case support the need for an effective VTE prophylaxis for brain tumor patients

in general and meningioma in particular. Pharmacological therapy should be considered preoperatively if the operation is not scheduled for a few days. Some physicians recommend the use of an inferior vena cava (IVC) filter. However, IVC filters have a complication rate that may reach up to 62% including procedure related and thromboembolic.¹ In addition, a DVT surveillance process should be considered for high-risk immobile patients prior to the placement of IPC. It is recognized that DVT surveillance using venous duplex ultrasound scanning (VDUS) allows for early detection of DVT, even if silent and reduces the rates of PE among patients undergoing neurosurgical procedures.⁶ However, the rarity of the association between the application of an IPC device and DVT dislodgement is likely to make the cost-benefit ratio of such surveillance process questionable. Neurosurgeons should expect that VTE related mortality in meningioma patients who otherwise are characterized by long-term survival, is likely to be put under the medico-legal spotlights. The prophylaxis regime should be balanced, and the neurosurgeon should not feel pressurized to use overly aggressive measures and subject the patient to the risk of a devastating ICH. Therefore, until data on primary prevention are available, the most appropriate VTE prophylaxis remains to begin mechanical prophylaxis in the form of ES and IPC before the operation and then to add a pharmacologic agent 18-24 hours afterwards. In addition, there should be low threshold for DVT surveillance using VDUS for high-risk patients preferably before the application of the IPC.

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