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Pulmonary Embolism as a Consequence of Applying Sequential Compression Device on Legs in a Patient Asymptomatic of Deep Vein Thrombosis

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INTRAOPERATIVE pulmonary embolism (PE) is an uncommon complication of emergency intraabdominal surgery. Best practice recommendations for longer surgical cases include the fitting of antithrombotic leg devices such as stockings and pneumatic compression devices.[1,2] Although these devices certainly prevent thrombosis in deep vein thrombosis (DVT)-prone surgical patients,[1,2] we were unable to find reports of any significant complications caused by them (besides minor skin bruising, sores, *etc.*), namely, dislodgment of a preexisting thrombus, causing a life-threatening PE. Although PE has been observed after tourniquet deflation in orthopedic surgery cases,[3] we report a case of PE possibly related to pneumatic sequential compression device.

Case Report

A 56-yr-old woman who had been in bed for 2–3 days came to the emergency department with signs and symptoms of a small bowel obstruction. She had recently been diagnosed with lymphoma. The working diagnosis in this woman, who had undergone no prior abdominal surgery, was obstruction caused by a secondary deposit of tumor. Although she was in considerable abdominal pain, there was no indication of either pulmonary insufficiency or of lower-extremity swelling.

After fluid resuscitation and administration of antibiotics, the patient was taken to the operating room, where her initial vital signs and oxygen saturation (Sp_{O_2}) were within normal limits. A sequential compression device with long sleeves (Sequel model 6325; Kendall Company, Mansfield, MA) was applied to both legs as part of routine practice for any surgery lasting over 3 h. The sequential device was turned on just before the induction of anesthesia with inflation pressures around 45 mmHg. General anesthesia was induced using a rapid-sequence technique. The medications included thiopental and succinylcholine. After injection of induction agents but before insertion of an endotracheal tube, the pulse oximeter reported a near instantaneous decrease from 100% to approximately 75% (despite the administration of 100% oxygen). Her systolic blood pressure also decreased to 90 mmHg from a preanesthesia value of 130/70 mmHg. Prompt intubation, ventilation with oxygen at a fraction of inspired oxygen of 1.0 atm, and confirmation of tube placement, both by auscultation as well as by positive end-tidal carbon dioxide, did not succeed in restoring her Sp_{O_2} to the preanesthesia level. She also was noted to have high peak airway pressures (> 40 cm H_2O), a low end-tidal carbon dioxide level (approximately 22 mmHg), and Sp_{O_2} around 95%. An arterial blood gas in the operating room later revealed *p*H of 7.25, carbon dioxide partial pressure of 55, oxygen partial pressure of 83, and HCO_3 of 25 on 1.0 fraction of inspired oxygen, and positive end-expiratory pressure of 10 cm H_2O. Desaturation was initially attributed to an unobserved

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Key words: Pneumatic compression; prophylaxis; trauma.

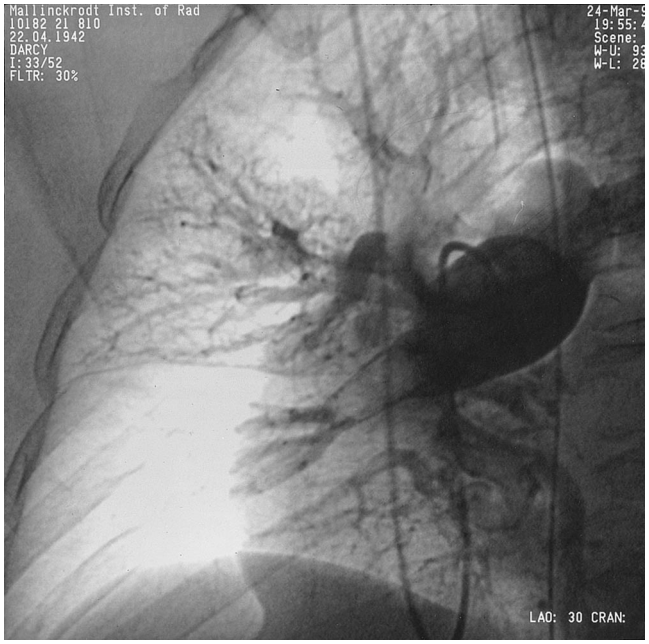

Fig. 1. Pulmonary arteriogram showing significant compromise of perfusion to right middle lobe and right lower lobe.

aspiration. When the operation concluded, she remained intubated and mechanically ventilated and was transferred to the surgical intensive care unit. In the surgical intensive care unit, she was hemodynamically stable on minimal doses of dopamine infusion but still had an unexplained requirement of relatively high fraction of inspired oxygen (0.6–0.7 atm) and positive end-expiratory pressure of 7.5–10 cm H_2O, to maintain an arterial oxygen partial pressure of 65–70 mmHg. The chest radiograph did not show evidence of pulmonary edema or any infiltrates suggesting aspiration. The patient became oliguric despite increasing central venous pressures, prompting us to obtain a transthoracic echocardiogram to assess better her cardiac function. This suggested dilatation of right atrium and ventricle with pulmonary artery pressures of 46/20 mmHg with a relatively underfilled left heart. A pulmonary artery catheter was inserted and confirmed these findings. We suspected an acute PE. The interventional radiologists performed an emergency pulmonary arteriogram (figs. 1 and 2), confirming huge emboli in the proximal tree with multiple smaller emboli in the more distal branches. Heparin therapy was initiated, and a mechanical filter was placed in the inferior vena cava just below the level of the renal veins after her Doppler study revealed DVT involving distal and proximal veins in bilateral lower extremities. Her oxygen requirement gradually improved over 2–3 days of heparinization. Because this patient seemed to have a variant of cancer-associated thrombosis and embolism (Trousseau's syndrome), warfarin therapy was eschewed in favor of long-term low-molecular-weight heparin therapy. She was transferred to the ward 4 days after the surgery and PE, after her Sp_{O_2} was $>$ 95% on 2 l of O_2 *via* nasal cannula; a few days later, she was discharged home without supplemental O_2. The leg sequential compression device was discontinued as soon as the diagnosis of embolism was suspected.

Discussion

Modern care of the critically ill patient dictates an aggressive approach to preventing DVT, the precursor to PE. These preventive interventions include but are not limited to compression boots, long sequential pneumatic compression stockings, subcutaneous heparin, low-dose warfarin, low-molecular-weight heparin, aspirin, *etc.* Although all of these interventions are associated with intervention-specific risks, the risks have generally been held to be small in comparison to the event that they intend to prevent.

Among the interventions, the compressive devices applied directly to the legs have been argued to be among the safest, with proven efficacy for DVT prophylaxis. Review of the professional literature, discussion with manufacturers, and review of the reports to the Food and Drug Administration revealed no significant complications of these devices when used appropriately. Use of pneumatic compression devices in prophylaxis of DVT/PE was found to be very effective and equivalent to low-dose subcutaneous heparin therapy in reducing incidence of DVT (and possibly PE) in moderate to high-risk surgical patients, including those with malignancy.[2,4,5] However, their use is contraindicated in patients with a history of DVT/PE within the previous 6 months.[6]

These devices should not be used in patients who are

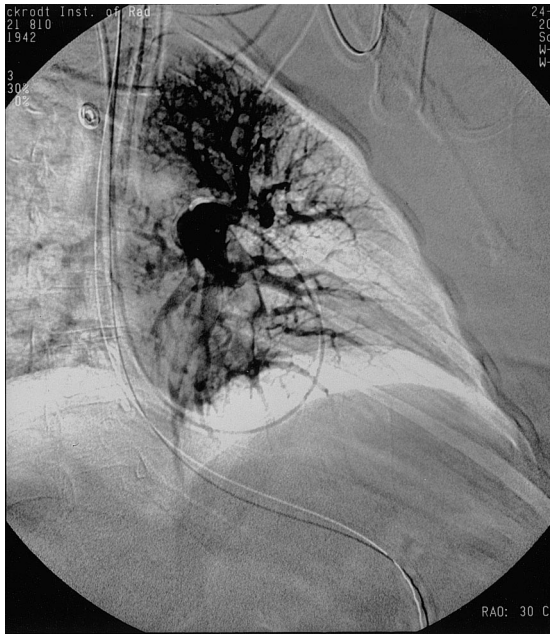

Fig. 2. Pulmonary arteriogram showing almost complete occlusion of left upper and lower lobe arteries by significant thromboembolism.

immobilized for more than 72 h without any DVT prophylaxis.[6] Conspicuous by its absence is any report of such devices precipitating an embolic event during general surgery.

We cannot establish causality. It is possible that the embolism would have occurred irrespective of the placement of the sequential devices because of the presence of multiple risk factors in our patient, including age, obesity, malignancy, estrogen replacement hormones, and relative immobility (because of illness). Nevertheless, the temporal association between activation of the pneumatic device and sudden onset of symptoms at least suggests that the application of preventive measure may have paradoxically triggered a life-threatening complication.

Such an anecdotal report should not be held up as the sole basis for changing clinical practice, particularly when benefits of these compression devices outweigh the risk of complication. However, we suggest that these well-intentioned prophylactic measures may have adverse consequences, particularly if they are not used appropriately. Clinicians who care for patients with moderate to high risk for DVT who are fitted with these devices should remain alert for signs and symptoms of acute embolism, especially during the first few minutes and hours after their application.

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Reversal of Intraoperative Myocardial Ischemia with a Hemoglobin-based Oxygen Carrier

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THE hemoglobin-based oxygen-carrying solutions currently under investigation have oxygen transport and exchange properties similar to blood. However, their cardiovascular effects are still a subject of controversy. In particular, there have been several reports of extensive vasonconstriction,[1–4] and several authors have suggested that this effect may be deleterious for the myocardium in patients with coronary artery disease.[5,6]

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Key words: Cardiovascular effects; coronary artery disease; tissue oxygenation.

Case Report

A 64 yr-old man with abdominal aortic occlusive disease was admitted for an occlusion of the right lower limb of an aortobifemoral graft. The patient had previously undergone several radiologic and surgical lower-limb revascularization procedures. The patient reported an uncomplicated myocardial infarction after a previous aortobifemoral grafting procedure. However, there was no subsequent angina or cardiac insufficiency. On admission, his electrocardiogram showed a atrial fibrillation longstanding with a ventricular rate of 90 beats/min. Intravenous heparin was started but was complicated by a significant lower gastrointestinal hemorrhage leading to a decrease in hemoglobin