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Fatal Pulmonary Embolism Secondary to Limb Exsanguination

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Immobilization and late operative fixation are common orthopedic practices following compound fractures. Exsanguination and application of a tourniquet frequently are performed prior to limb surgery. We report a case referred for delayed internal fixation in which the application of an Esmarch® bandage to exsanguinate the limb precipitated a fatal pulmonary embolus.

REPORT OF A CASE

A 57-year-old miner sustained a compound fracture of his left tibia and fibula. Six hours later, he underwent debridement and suturing of the wounds and manipulation of the frature under general anesthesia. The leg was then splinted in a plaster of paris cast. The intraoperative course was uneventful. For the next 15 days he remained in the hospital with the leg immobilized. The position of the fracture subsequently was deemed unacceptable, and he was scheduled to undergo internal fixation under general anesthesia.

Preoperative assessment revealed no abnormality of the cardiovascular or respiratory systems, except for mild hypertension treated with a thiazide diuretic and potassium supplements. The chest radiograph and ECG were normal and the arterial blood pressure was 140/90 mmHg with a heart rate of 84 beats/min.

Anesthesia was induced with 500 mg thiopental, iv, and muscular relaxation obtained with 45 mg *d*-tubocurarine, iv. The trachea was intubated, and anesthesia maintained with 66% nitrous oxide in oxygen. Ventilation was controlled using a Manley ventilator. Phenoperidine (a synthetic meperidine derivative) 3 mg, and droperidol 5 mg were administered iv to supplement anesthesia. Monitoring consisted of an ECG, and the arterial blood pressure was measured every minute using a Dinamap®.

The plaster of paris cast was removed from the left leg, and the entire leg was cleaned and wrapped in a sterile towel. A tourniquet was applied to the thigh. Before inflation of the tourniquet, the leg was exsanguinated by the application of an Esmarch bandage. Prior to this, cardiovascular parameters had been stable, but within one minute of the application of the Esmarch bandage cyanosis occurred and the arterial blood pressure could no longer be obtained. External cardiac massage was instituted immediately, and attempts at resuscitation were continued without success for one-half hour. Subsequent postmortem examination showed the cause of death to be multiple pulmonary emboli completely blocking both pulmonary arteries. The veins of the injured leg were found to be filled with antemortem thrombus, and we concluded that the traumatized left leg had been the source of the emboli.

DISCUSSION

The risk factors involved in the pathogenesis of deep vein thrombosis (DVT) have been well-documented,[1,2] among which trauma, surgery, and immobility are prominent. Enforced bed rest and immobility in an individual who has been previously fully active predisposes to venous stasis. A period of bed rest of as little as three days may allow the development of a DVT,[3] and beyond three days, this incidence steadily rises.[3] When stasis is combined with another predisposing factor such as trauma, the likelihood of development of a DVT is greatly increased.[1] Fractures of the lower limbs are particularly prone to the development of a DVT.[4]

The diagnosis of DVT may be difficult. The symptoms and signs of pain, tenderness, and swelling are present in only one-third[5] to one-half[6] of patients. The majority of DVTs, therefore, are clinically silent. In the patient reported, encasement of the limb in a plaster of paris cast also complicated the diagnosis.

The presence of a bloodless field for certain orthopedic procedures is often advantageous to the surgeon. This usually is accomplished with a tourniquet, following exsanguination of the limb using an Esmarch bandage. In our case, although embolism may have occurred spontaneously at any time, the application of the Esmarch bandage most probably dislodged the venous thrombus, leading to massive pulmonary embolism and cardiac arrest.

Following acute massive pulmonary embolism, about 50% of patients die within the first 15 minutes,[7] and only about 33% survive over two hours.[8] The mortality of massive pulmonary embolism thus is very high. This is therefore a complication where prevention is of paramount importance. Early detection of thrombosis and reliable prophylactic therapy are two interrelated approaches in the achievement of this goal.

The standard test for diagnosis of DVT is ascending contrast phlebography, but this test is invasive. Noninvasive methods of diagnosis include augmented Doppler ultrasound examination, electrical impedance phlebography, and radiolabeled fibrinogen techniques. A

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combination of impedance phlebography and radiofibrinogen leg scanning is reported to be of comparable sensitivity to invasive contrast venography.[9]

The application of non-invasive techniques in the early diagnosis of DVT has demonstrated the prophylactic value of low-dose heparin given subcutaneously. There is little alteration of clotting time and partial thromboplastin time, and little hemorrhagic hazard when administered in a dose of 5,000 units twice daily.[9] At present, the use of platelet function inhibitors such as aspirin and dipyridamole remains unclear.[10] Dextran, which also impairs platelet aggregation, is an alternative to low-dose heparin, but Smith *et al.*[11] have reported postoperative bleeding and allergic reactions, and it must be used with caution in patients with limited cardiovascular reserve due to the danger of fluid overload.[10]

In conclusion, we suggest that if delayed internal fixation requiring limb exsanguination is performed, a high index of suspicion for the presence of deep vein thrombus must be maintained. Active prophylaxis against the development of venous thrombosis should therefore be considered.

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Intravenous Verapamil to Relieve Pulmonary Congestion in Patients with Mitral Valve Disease

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In mitral valve disease, the development of pulmonary congestion is dependent on factors increasing resistance to flow in the left atrium, notably tachycardia (usually as the result of rapid ventricular response to atrial fibrillation), which shortens diastole during which the left atrium must be emptied.[1] Drugs that prolong AV nodal conduction time have been used in patients with atrial fibrillation to slow a fast ventricular response, *e.g.*, digitalis, propranolol, guinidine, and procainamide.

Cells in the AV node are probably slow-channel-dependent, and AV nodal conduction can be slowed by drugs that interfere with the slow inward current.[2,3] Verapamil (a calcium channel blocker) is effective in terminating supraventricular tachycardia or slowing the ventricular rate during atrial fibrillation.[3,4]

In two patients with mitral valve disease and pulmonary congestion due to the rapid ventricular response to atrial fibrillation, we administered verapamil

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