Fatal pulmonary embolism subsequent to the use of Esmarch bandage and tourniquet: A case report and review of literature

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ABSTRACT

We report a case of fatal pulmonary embolism (PE) following exsanguinations of lower limb in orthopedic surgery. A 30-year-old man was posted for fixation of a tibial plateau fracture on the seventh day after injury. Subarchnoid block was performed. Esmarch bandage was used for exsanguination of the limb and tourniquet was inflated. Within three minutes after tourniquet inflation, the patient became unconscious hypotensive, and collapsed. Resuscitation was instituted and spontaneous circulation restored. Right ventricular strain pattern on transthoracic echocardiography, increased levels of D-dimer, and Doppler examination of the lowerlimb confirmed deep venous thrombosis of right femoral vessels and PE. In spite of thrombolytic therapy with streptokinase and heparin, the patient died on the ninth day after the event. Cases of PE secondary to the use of Esmarch bandage and tourniquet are reviewed here to identify the risk groups and management of PE.

Key words: *Esmarch bandage, lower limb fractures, pulmonary embolism, tourniquet*

INTRODUCTION

Esmarch bandage is commonly used for the exsanguination of the limb before applying the tourniquet, to provide a bloodless field for the surgery. Many cases of severe pulmonary embolism (PE) have been attributed to the use of Esmarch bandage and tourniquet.[1-12] We report a case of PE following the use of Esmarch bandage and review the literature relating to PE subsequent to the use of Esmarch bandage and tourniquet.

CASE REPORT

Following a road traffic accident, a 30-year-old male developed right tibial plateau and fibular fracture. There were no other significant injuries. The limb was immobilized in a plaster cast for six days and he was planned for open reduction and internal fixation of the fracture. Due to the presence of limb edema, surgery could not be carried out till the sixth day after injury. Routine monitoring including noninvasive blood pressure, electrocardiography (ECG), and pulse oximetry were started. Spinal anesthesia was performed with 3.5 mL of hyperbaric bupivacaine 0.5%, along with buprenorphine 120 mcg. Block level of T4 was achieved and surgery was started. Approximately 40 minutes after spinal anesthesia, before starting open reduction of the tibia, exsanguination of the limb was carried out using Esmarch bandage and the tourniquet was inflated to a pressure of 300 mmHg. In the next three minutes, the patient suddenly became unconscious and his blood pressure dropped to 60/34 mmHg. Intravenous ephedrine 15 mg was given but the hypotension persisted. Endotracheal intubation was performed and full-support ventilation was initiated. Palpation of carotid artery pulsation and ECG revealed electromechanical dissociation. External cardiac compression was initiated and intravenous adrenaline of 1 mg was given every three minutes. For the brief duration of ventricular fibrillation which developed, defibrillation was done. It was suspected to be due to PE; hence, heparin 5,000 IU was given intravenously about 20 minutes after the arrest. After about 30 minutes of cardiopulmonary resuscitation (CPR), spontaneous circulation returned. Infusion of dopamine was started for hypotension. Surgery was abandoned and the patient was shifted to the intensive care unit (ICU) for further management. In the ICU, transthoracic echocardiography

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revealed right atrial and right ventricular dilatation and tricuspid regurgitation. Serum level of D-dimer was increased (>5,000 ng/mL), and Doppler study of the lower limb indicated the presence of thrombus in right femoral vein, confirming deep venous thrombosis (DVT) and PE as the diagnosis. Thrombolysis was started with intravenous streptokinase 500,000 IU. Intravenous infusion of heparinwas continued at 1,000 IU/hour to maintain activated partial thromboplastin time (aPTT) for around 90 seconds (three times control). Infusion of dopamine was stopped following normalization of blood pressure. Cerebral protective measures like mannitol, phenytoin, and dexamethasone were started from the second day. The patient never had a complete neurological recovery. Four days after the event, magnetic resonance imaging (MRI) of the brain revealed diffuse hyperintense signals in bilateral areas of the cerebral hemisphere, suggestive of diffuse cerebral edema. There was obliteration of the basal cistern and herniation of cerebellar tonsils. His neurological status deteriorated further in the next five days. The patient did not have a neurological recovery and died on the ninth day after the surgery.

DISCUSSION

Trauma, immobility, and surgery are known to predispose to DVT. Immobility of more than three days predisposes a person to DVT and the possibility increases with lapse of time. Fractures of lower limbs are known risk factors for the development of DVT. Although thrombus can embolize spontaneously anytime, mechanical stress caused by Esmarch bandage and tourniquet are thought to cause a sudden increase in the velocity of the venous flow and dislodge the pre-existing thrombus. Many cases of massive PE have been reported following the use of Esmarch bandage and tourniquet.[1-12]

We could find a total of 14 cases of embolism related to the use of Esmarch bandage and tourniquet inflation [Table 1]. Of these, six patients were female and eight were male. So, there appears to be no predilection for either sex. The age of the patients varied from 30 years (in our case) to 90 years. The mean age of patients was 62 years. A higher prevalence of fractures in the older age group may be the reason for a higher incidence of embolism reported in the elderly. Higher age is also considered as an independent risk factor for the development of DVT. Most of these cases were reported in patients undergoing fixation of fracture subsequent to trauma, but PE has also been reported in elective total knee replacement arthroplasty.[9]

The most important factor for the development of DVT in these patents is immobilization from the time of injury to the surgical intervention. Most of these patients had a delay before the surgery varying from one day to three weeks (with a mean duration of immobilization of 12 days prior to surgery). One of the patients, who underwent surgery one day after the injury also had an embolic event.[7,8] This implies that even though, most of the patients develop DVT during the period of immobilization, it can happen without a prior history of immobilization. Most of the patients did not receive any anticoagulation during the period of immobilization (9 out of 14). Even in patients where anticoagulation was used, it was stopped inappropriately early before the surgery (warfarin stopped two weeks prior to the surgery; two cases) or was given for only one dose of low-molecular-weight heparin (LMWH) prior to the surgery (one case).[3,4,9] One of the patients had developed PE prior to the surgery, which was treated with subcutaneous heparin. Subsequent venography did not reveal DVT; hence, they proceeded with the surgery, but the patient developed severe PE.[12]

The type of anesthesia is unlikely to influence the possibility of embolizationof DVT which is formed prior to the surgery. The patients developed PE under all three forms of anesthesia. (general anesthesia: Subarchnoid block: Epidural = 3: 7: 3). Most of these cases developed symptoms immediately after the use of Esmarch bandage to five minutes after the inflation of the tourniquet. In most of the patients, Esmarch bandage was used for limb exsanguination, but in two cases, the tourniquet was inflated without prior use of the Esmarch bandage. So, the use of Esmarch bandage is commonly associated with PE, but is not a prerequisite. Increase in the compartment pressure in the limb caused by tourniquet inflation maybe sufficient to dislodge the thrombus and manifest as PE.

Like PE developing following the use of Esmarch and tourniquet inflation, similar episodes of PE have been reported following tourniquet deflation [Table 2].[13-16] These cases were also reported in either sex and in varying age groups, mostly in patients undergoing trauma surgeries. Immobilization prior to the surgery without receiving adequate anticoagulation is a common risk factor even in these cases. Post-tourniquet deflation PE is also reported under all the three types of anesthesia. All these cases reported cardiovascular collapse within the first five minutes of tourniquet deflation.

However, intraoperative cardiac arrest related to tourniquet deflation maybe caused by metabolic acidosis and vasodilation, with resultant myocardial ischemia and left ventricular dysfunction, but PE was demonstrated in all these cases by transesophageal echocardiogram (TEE) or pulmonary angiography. The possibility of bone marrow, cement, or air embolizing after tourniquet

Table 1: Case reports of pulmonary embolism following Esmarch bandage/tourniquet inflation

Ref	Age/sex	Type of surgery	Time since injury (days)	Anticoagulation	Anesthesia	Time after esmarch/ tourniquet inflation (min)	Outcome	Comment
[1]	14/M	Trauma	15	No	GA`	Immediate	Nonfatal	Thrombolysis
[2]	57/M	Trauma	15	No	GA	1	Fatal	Postmortem confirmation
[3]	76/F	Trauma	7wks	Warfarin, stopped 2 weeks prior to surgery	Epidural	No esmarch immediate after tourniquet	Fatal	Postmortem confirmation
[4]	71/F	Trauma	6wks	Warfarin, stopped 14 days prior to surgery	SAB	2	Fatal	Postmortem confirmation
[5]	60/M	Trauma	6	No	GA	5	Nonfatal	TEE Surgical embolectomy
[6]	36/M	Trauma		No	Epidural	Second tourniquet	Fatal	Pulmonary angiography Postmortem confirmation
[7]	76/F	Trauma	1	No	SAB	Immediate	Fatal	Varicose vein TEE
[8]	76/F	Trauma	1	No	SAB	Immediate	Fatal	TEE
[9]	50/F	Trauma	8	LMWH, only 1 dose before the op	SAB	3 min	Fatal	H/o DM Postmortem confirmation done
[9]	51/F	TKR (elective)	Inpatient for 5 days	LMWH	Epidural	Immediate	Fatal	DM, past MI, obese Postmortem confirmation
[10]	71/M	Trauma	3	No	SAB	5	Nonfatal	TEE CPB embolectomy ECMO
[11]	90/M	Trauma		No	GA		Fatal	Revived with anticoagulant, died after 21 days
[12]	63/M	Trauma	11	Heparin	SAB	No esmarch immediate after tourniquet	Nonfatal	H/o PE about 10 days prior, was treated, venography prior to surgery did not show any DVT, heparin treatment
Our case	30/M	Trauma (tibia #)	7 days	No	SAB	3 min	Fatal	Streptokinase thrombolysis

TEE – Transesophagealechocardiography; CPB – Cardio pulmonary bypass; ECMO – Extracoporealmembrane oxygenator; PE – Pulmonary embolism; DVT – Deep vein thrombosis; MI – Myocardial infarction; GA – General anesthesia; SAB – Subarachnoid block; LMWH – Low-molecular-weight heparin; TKR – Total knee replacement; F – Female; M – Male; Wks – Weeks

Table 2: Case reports of pulmonary embolism following tourniquet deflation

Ref	Age/ sex	Surgery	Days of immobilization	Anticoagulation	Anesthesia	Time after tourniquet deflation (min)	Tourniquet time (min)	Outcome	Comments
[13]	55/M	Trauma	15	No	GA	5	45	Nonfatal	Pulmonary angiography, heparin anticoagulation
[13]	63/F	TKR elective	3 months after other TKR	No	GA	5	99	Fatal	HTN, CCF, hypothyroid, venography prior to sx superficial calf thrombus, no deep vein thrombus, TEE, TPA, postmortem confirmation
[14]	56/F	Trauma	10 days	No	GA	Immediate	90	Nonfatal	DM, thrombolysis failed, doppler and venography did not reveal thrombus, pulmonary angiography showed embolus, open pulmonary embolectomy showed fresh emboli
[15]	48/F	Achilles tendon repair		No	Epidural	5		Fatal	
[16]	55/M	Trauma		No	SAB	5	57	Fatal	Postmortem confirmation

CCF – Congestive cardiac failure; TEE – Transesophagealechocardiograhy; TPA – Tissue plasminogen activator; GA – General anesthesia; SAB – Subarachnoid block; TKR – Total knee replacement; M – Male; F – Female

deflation maybe suspected, but in all these reported cases, composition of the emboli was shown to be blood clots either during surgical embolectomy or during the postmortem examination. As with PE at tourniquet inflation, these patients may have developed DVT during the period of immobilization, which embolized after

deflation of the tourniquet. It is also possible that local rheologic changes produced by the pneumatic tourniquet resulted in venous thrombosis during the period of stasis with subsequent embolization once the tourniquet was deflated.

PE may have various manifestations under anesthesia like sudden onset of breathlessness, loss of consciousness, hypotension, and a fall in the end-tidal carbon dioxide (CO_2). The usual ECG changes are ST-T changes seen on the right-sided leads, right bundle branch block, and electromechanical dissociation. TEE has high sensitivity and specificity in detecting PE and right ventricular dilatation. Even transthoracic echocardiography can show right ventricular hypokinesia and dilation, but is not sensitive in identifying the embolus. Increase in plasma level of D-dimer can help in correlating the diagnosis, even though it is not specific for DVT. Normal levels of D-dimer will help to rule out DVT.

Massive PE has a very high rate of fatality; 13 of the 19 patients in these reports succumbed to the PE. Three of the patients who survived, had early diagnosis by TEE and underwent surgical embolectomy using cardiopulmonary bypass.[5,10,14] One of the patients needed 10 days of extracorporeal membrane oxygenation (ECMO) before the recovery.[10] In two of the patients who survived, early diagnosis was made and anticoagulation with heparin was given. One of the three patients receiving thrombolysis survived.

As the rate of fatality subsequent to severe PE is very high, prevention of DVT should be given priority. Preoperative anticoagulation should be used as routine and diagnostic workup should be performed to exclude the possibility of DVT. Many of the diagnostic tests are invasive, not sensitive, and expensive, making it difficult to use routinely. Diagnostic workup should be considered at least in patients who are immobilized for more than three days and where the use of tourniquet is being planned.

In a study comparing the use of limb elevation with the use of Esmarch bandage as a means of achieving exsanguination, it was noted that operating field visibility or requirements of transfusion did not differ between the groups, even though intraoperative loss of blood was more when limb elevation was used.[17] Another recent study has demonstrated that better leg hemodynamic changes were attained during the first month after knee arthroplasty in patients where Esmarchwas not used.[18] The use of Esmarch did reduce arterial flow to the lower limb for a longer period of time, hence raising the question about the need for exsanguination with Esmarch bandage before elective knee procedures. In a meta-analysis of elective knee procedures, it was shown that even though intraoperative loss of blood was significantly greater in the nontourniquet group compared to tourniquet-assisted surgery, there was no significant difference between the groups for total loss of blood or rate of transfusion.[19] There was a trend for greater complications in tourniquet compared to nontourniquet patients. Hence, it was concluded that there is no advantage in using a tourniquet in knee replacement surgery for the reduction of transfusion requirements. All these studies were reported in elective knee replacement surgeries; similar results can be expected in lower limb trauma surgeries.

Therefore, we conclude that patients who are immobile due to fractures of lower limbs are at agreater risk of developing DVT. The possibility of PE due to the detachment of venous thrombus by mechanical stress caused by the application of Esmarch bandage and tourniquet inflation should be kept in mind. Immediate diagnosis using TEE and rapid surgical embolectomyare the most important factors for successful resuscitation. As PE has a high rate of mortality, greater attention should be given for preoperative anticoagulation and diagnostic workup to prevent DVT. In view of the established risk of using Esmarch bandage, it is suggested that the use of this method in orthopedic surgery may not be justified. Even the use of the tourniquet maybe avoided in high-risk patients.

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