Case Report

Cardiac arrest after tourniquet deflation in tibial plateau fracture surgery in a healthy man

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ABSTRACT

Surgical tourniquets are commonly used in orthopedic and trauma surgery. Although the pneumatic tourniquet has been classified by the US Food and Drug Administration as a class I medical device, there are still reports of hazards related to its use. Here, we describe an unusual complication experienced by a healthy man after he underwent surgery for trauma to the lower extremity. The patient experienced cardiac arrest 5 minutes after tourniquet release and was resuscitated immediately. He regained consciousness without any symptoms, and the electrocardiographic signs of arrhythmia completely resolved within 30 minutes of leaving the operating room. The patient recovered completely and was discharged 5 days later. The mechanism underlying the incidence of cardiac arrest after tourniquet deflation is still not clear. By ruling out other common intraoperative triggers as possibilities and upon reviewing the literature, we conclude that reperfusion injury is a plausible and the most likely explanation. Injuries resulting from tourniquet use are commonly pressure-related, and can also be caused by excessive tourniquet time. To reduce these injuries, an adequate pressure setup (a cuff pressure not exceeding 300 mmHg) and tourniquet ischemia time less than 2 hours should always be the preferred option. Even then, it may be impossible to eliminate all associated complications.

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1. Introduction

Surgical tourniquets are commonly used in orthopedic and trauma surgery, but their complications and contraindications are well recognized. Tourniquets are required to facilitate good exposure of the surgical field, in which case it is often necessary to prolong the duration of the procedure. Surgeons and anesthetists always pay attention to the duration of tourniquet use, which is associated with an increased risk of complications. Here we describe an unusual complication of trauma surgery performed on the lower extremity of a healthy man.

2. Case report

In November 2008, a 54-year-old man, a heavy worker and smoker (12 cigarettes per day) but with no past history of ischemic heart disease, presented to our emergency department after a motor vehicle crash. He reported pain in his swollen right knee. Physical examination of the right knee revealed diffuse tenderness and swelling compatible with hemarthrosis. The distal peripheral pulses were palpable, and no neurological deficits were noted in the right lower limb.

Radiography of the right knee showed a Schatzker type V fracture of the tibial plateau (Fig. 1). The results of the preoperative laboratory evaluations, including a complete blood count, blood coagulation profile, and blood chemical parameters, were all within normal limits. The results of evaluations using electrocardiographic (ECG) and roentgenographic imagings of the chest were also normal.

Under general anesthesia, open reduction and internal fixation (Fig. 2) were performed after a pressure of 300 mmHg had been applied to the right thigh using a tourniquet. The patient’s blood pressure during the tourniquet inflation was 146/88 mmHg. The tourniquet was used for 2 hours 12 minutes. The patient experienced arrhythmia 5 minutes after the tourniquet was released, which was clearly noted on the ECG tracing (ST segment depression, and then widening of the QRS complex) in leads II and III (Fig. 3). Cardiac arrest then occurred, and the patient was resuscitated immediately.

After 30 minutes of resuscitation, the patient’s vital signs became stable. Blood samples were then sent for a complete blood cell count and analyses to determine the levels of urea, arterial blood gas, cardiac enzymes (creatine kinase 116 U/L, creatine...
kinase-MB 36.1 U/L, troponin I: 0.02 μg/L), D-dimer (2.28 mg/L), and electrolytes (potassium 5.7 mmol/L) during resuscitation. The patient was taken to the recovery room for 12-lead ECG and further management.

During recovery, a cardiological opinion was sought. An echocardiogram was obtained, which showed hypokinesis of the anterior wall and of the apical, middle, and basal septa of the left ventricle, with mild-to-moderate impairment of left ventricular systolic function. The patient regained consciousness without any symptoms, and the arrhythmia recorded on the ECG (Fig. 4) completely resolved within 30 minutes of his leaving the operating room.

In the recovery room, an arterial blood gas sample was immediately taken for analysis and showed metabolic acidosis (pH 7.23, base deficit 2). The electrolytes revealed a serum potassium level of 5.7 mmol/L. The patient left the recovery room 3 hours after the operation with a completely normal ECG and normal repeat blood sample results, indicating resolution of the previous discrepancies.

The patient made a full recovery and was discharged 5 days later. An intensive cardiovascular follow-up examination was conducted in the outpatient department after the patient had been discharged, and no cardiovascular disorders, such as coronary heart disease, were found. The implants were removed after 1 year because of bony union.

3. Discussion

The use of a tourniquet in the treatment of fractures is somewhat controversial. In the early years of the Association for Osteosynthesis/Association for the Study of Internal Fixation, it was suggested that internal fixation should be performed using a tourniquet because it would maintain a relatively bloodless surgical field during extremity surgery, reduce operating time, minimize blood loss, aid in the identification of vital structures, and expedite the procedure. Moreover, animal studies have shown no delay in bone healing with the use of a tourniquet. Nevertheless,
the interruption of the blood supply leads to transient local ischemia, which might be less of a problem in elective musculoskeletal surgery.6

However, in fracture treatment, the use of a tourniquet can be associated with postoperative complications, such as pressure-related nerve injury,7,8 post-tourniquet syndrome, intraoperative bleeding, compartment pressure syndrome, higher rates of infection,9,10 pressure sores and chemical burns, thrombosis, tourniquet pain, rhabdomyolysis, and metabolic changes.10,11 Injuries resulting from pneumatic tourniquet use are commonly pressure-related and can be caused by excessive tourniquet time. Whereas most of these problems may be transient, perhaps even unnoticeable, some are permanent, irreversible, and even life-threatening.12 The actual requirement for tourniquet application in acute fracture treatment must always be considered.

Within the last 30 years, important improvements in the technology of tourniquet instruments and tourniquet cuffs have been made, and the US Food and Drug Administration has classified pneumatic tourniquets as class I medical devices. Despite these improvements, there are still reports of hazards related to their use.13,14 The mechanism underlying the incidence of cardiac arrest after tourniquet deflation is still not clear. In this case, we excluded other possibilities, such as myocardial infarction, pulmonary embolism, and anesthetic agent-induced reperfusion injury. Reperfusion injury after tourniquet release may be the most important cause of cardiac arrest after tourniquet deflation.

In setting up an adequate cuff pressure, the surgeon must consider the following factors: blood pressure (the occurrence of hypertension, which demands a higher tourniquet cuff pressure), cuff design (curved and wider tourniquet cuffs occlude blood flow at a lower inflation pressure than straight or narrow cuffs), limb circumference (a higher tourniquet cuff pressure is required for limbs with a large mass of fatty or muscular subcutaneous tissue), the state of the tissue (folds and puckers in the underlying flaccid tissue can cause skin injury and uneven pressure on the vessels), and vascular status (the presence of atherosclerotic vascular disease or a similar disease that occludes the artery demands a higher tourniquet cuff pressure).15

Limb occlusion pressure (LOP) is defined as the minimum pressure required at a specific time by a specific tourniquet cuff applied to a specific patient limb at a specific location to stop the flow of arterial blood into the limb distal to the cuff. LOP is usually determined by gradually increasing tourniquet pressure until distal blood flow is interrupted.16 The currently established guideline for

Fig. 3. ECG series. The patient experienced arrhythmia 5 minutes after the tourniquet was released.
setting tourniquet pressure on the basis of LOP is used with an additional safety margin of pressure to the measured LOP.

In the 2009 Recommended Practices for the Use of the Pneumatic Tourniquet, the US Association of Perioperative Registered Nurses recommended that tourniquet pressure for normal adults should be set on the basis of the LOP, as measured with any validated method, with an additional safety margin of 40 mmHg for an LOP of <130 mmHg, 60 mmHg for those of 131–190 mmHg, and 80 mmHg for those of >190 mmHg.15 Many procedures are required for measuring LOP, and these are time-intensive. Therefore, earlier heuristic recommendations, such as adding 50–75 mmHg and 100–150 mmHg to the arm systolic blood pressure for tourniquet pressure during upper and lower limb surgery, respectively,4 may not be ideal but are quite simple for clinical use.

In this patient, after administering general anesthesia, the blood pressure was 144/88 mmHg, so a cuff pressure set-up of 300 mmHg was acceptable. Higher levels of tourniquet pressure are associated with a higher risk of tourniquet-related injury. Cuff pressure should not exceed 300 mmHg and 100–150 mmHg to the arm systolic blood pressure for tourniquet pressure during upper and lower limb surgery, respectively,4 may not be ideal but are quite simple for clinical use.

There are some physiological changes during the ischemic phase and throughout the recovery phase after release of a tourniquet. For example, serum creatine phosphokinase concentration is elevated in response to muscle damage. Furthermore, interruption of blood supply results in cellular hypoxia, tissue acidosis, and potassium release.21

After 2 hours of tourniquet ischemia, the venous pH decreased from a mean preinflation value of 7.40–6.90, the venous PO2 fell from 45 mm to 4 mm, and the PCO2 increased from 38 mm to 104 mm. Upon release of the tourniquet, there was a readjustment phase in which the PH and venous PO2 values gradually returned to their preinflation values.

The readjustment phase after 2 hours of ischemia is always longer than 15 minutes.22 In healthy individuals, no significant adverse effects have been observed after 2 hours of tourniquet ischemia time. However, these changes may be clinically significant in some patients with impaired cardiopulmonary function.22

The risk of tourniquet-related complications increases with longer tourniquet use.24–26 In clinical practice, the safe tourniquet inflation time depends greatly on the patient’s anatomy, age, and physical status, as well as the vascular supply to the extremity.15 Prolonged application of the tourniquet causes severe acidosis and its concurrent problems. One hypothesis suggests that a relatively long tourniquet time and its placement on a patient’s muscular leg leads to muscle ischemia with accumulation and release of lactate, toxic metabolites, and intracellular mediators after tourniquet deflation.14 The return of toxic metabolites to the circulation system results in systemic metabolic dysfunction and is characterized by metabolic acidosis, hyperkalemia, myoglobinemia, myoglobinuria, and even renal failure.4

To date, no prospective randomized clinical trial has defined the optimal duration of tourniquet use in lower limb surgery; however, 2 hours is considered to be relatively safe for upper limb surgery.4 Although there is no completely safe tourniquet time, a routine tourniquet inflation time of more than 2 hours should be avoided.27,28 If the tourniquet ischemia time is longer than 2 hours, it may be approaching some critical points and produce irreversible changes in the metabolic system.14 The surgeon has to balance the potential downside of delayed deflation with the risks involved in prolonging tourniquet inflation times.

Fig. 4. The arrhythmia recorded in the previous ECG series completely resolved after 30 minutes of resuscitation.
In our patient, the tourniquet inflation time was 2 hours 12 minutes. The onset of cardiac arrest approximately 5 minutes after deflation, coupled with the elevated serum potassium level and metabolic acidosis, supports the hypothesis. These circulating mediators can be implicated in causing a degree of coronary vasospasm sufficient to produce the noted ECG appearances and explain the relatively rapid resolution of the problem. Previous studies support the hypothesis of metabolic and hemodynamic insults associated with prolonged tourniquet inflation.

Although we cannot state with certainty that this was indeed the mechanism underlying the cardiac arrest in our patient, we conclude, by ruling out other common intraoperative triggers as possibilities and upon reviewing the literature, that this is a plausible and most likely explanation.

4. Conclusion

According to the US Food and Drug Administration, the pneumatic tourniquet is classified as a class I medical device. However, there are still some reports of hazards related to their use. In our patient, the mechanisms underlying the cardiac arrest after tourniquet deflation is still not clear. Upon reviewing the literature, we have concluded that reperfusion injury is a plausible and the most likely explanation.

There is no clear-cut rule for how long a tourniquet may be safely inflated, although various investigators have addressed the effects of ischemia on muscle and nerve to define a relatively safe period of tourniquet hemostasis. However, adequate pressure set-up (a cuff pressure not exceeding 300 mmHg) and a tourniquet ischemia time no longer than 2 hours should be always kept in mind. In our opinion, in some patients, such as those with impaired cardiopulmonary function, chronic renal insufficiency, and so on, the tourniquet time should be readjusted, although there is no evidence to support this readjustment.

The final decision of when to deflate the tourniquet should be made by the surgeon, after weighing the risks and the benefits of delaying tourniquet deflation until the surgical procedure is complete. Careful monitoring of the patient is essential at this stage of the surgical operation. It is difficult to say how our management of a similar case in the future might change with respect to this experience. Surgeons, anesthetists, and surgical staff should always be attentive to the duration of tourniquet use. However, it may still be impossible to eliminate all associated complications.

References