

Case Report

Cardiopulmonary Collapse after Tourniquet Deflation Following Orthopedic Surgery

Yu-Hsuan Shih¹, Ping-Hsin Liu¹, Wei-Hung Chen¹, Shao-Wei Hsieh¹, Kuo-Chuan Hung²

Intraoperative cardiopulmonary collapse following tourniquet deflation is rare. A 42-year-old woman with anesthesia risk belonging to American Society of Anesthesiologists class II (ASA II) was admitted for fixation of an open humeral shaft fracture and closed tibia and fibula shaft fractures. Following uncomplicated general anesthesia and surgery, the tourniquet was deflated after 84 min of use. Approximately five minutes after tourniquet deflation, a rapid decline in end-tidal carbon dioxide partial pressure (EtCO₂) from 35 mmHg to 5 mmHg was observed with PaCO₂ of 93.7 mmHg and PaCO₂-to-EtCO₂ gradient of 88.7 mmHg. This was followed by unstable hemodynamics and cardiac arrest. The patient died despite immediate and vigorous cardiopulmonary resuscitation. Autopsy was refused by her family. Massive pulmonary embolism (PE) was suspected based on the marked increases in dead space and cardiopulmonary collapse. In the light of the fulminant course of this complication, we suggest that the PaCO₂-to-EtCO₂ gradient should be more intensively monitored if cardiopulmonary profiles become destabilized during deflation of the tourniquet. If symptomatic PE is suspected, the tourniquet may be temporarily re-inflated to avoid more emboli being released into the circulation. A diagnostic work-up should be immediately performed to exclude the possibility of PE.

Key words: tourniquet, pulmonary embolism, orthopedic operation

Introduction

Although the occurrence of venous thromboembolism in the circulation after tourniquet deflation is not uncommon,¹⁻⁴ only isolated cases with fatal or near-fatal pulmo-

nary embolism (PE) immediately after tourniquet deflation have been reported.^{5,6} Here, we report a case of cardiopulmonary collapse after tourniquet deflation following orthopedic surgery. We suggest that the PaCO₂-to-EtCO₂ gradient should be monitored more intensively if cardiopulmonary profiles become destabi-

From the ¹Department of Anesthesiology, E-Da Hospital, and I-Shou University, Kaohsiung, and ²Department of Anesthesiology, Chi Mei Medical Center, Tainan, Taiwan.

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Address reprint request and correspondence to: Kuo-Chuan Hung, Department of Anesthesiology, Chi Mei Medical Center, No. 901, Zhonghua Road., Yongkang City, Tainan County 71004, Taiwan.

Tel: +886-7-6150011 ext. 251312, Fax: +886-7-6155352, E-mail: ed102605@gmail.com

lized during deflation of the tourniquet. Written informed consent was obtained from the patient's family with regards to the publication of this report.

Case Report

A 42-year-old woman (American Society of Anesthesiologists class II; height, 154 cm; body weight, 68 kg) was admitted to our hospital with a left open humeral shaft fracture and closed tibia and fibular shaft fractures following a road traffic accident. Her underlying diseases included hypertension and diabetes mellitus without past history of thromboembolic events or myocardial infarction. Preoperative assessment revealed a Glasgow Coma Scale of 15, and the patient was hemodynamically stable with no respiratory depression. Preoperative electrocardiography and chest radiography were unremarkable. Radiograph of the left lower limb revealed a transverse fracture with displaced fragments of the left tibial and fibular shafts (Fig. 1), while the X-ray for the left humerus showed a fracture of the left humeral shaft. Furthermore, the platelet count and coagulation analysis were normal.

Intraoperative monitoring included electrocardiography, capnography, pulse oximetry, temperature monitoring, and non-invasive blood pressure measurement. Anesthesia was induced using intravenous fentanyl (75 µg) and thiopental (225 mg), followed by tracheal intubation facilitated with cisatracurium (10 mg). Anesthesia was maintained with 2% – 3% sevoflurane in oxygen and air. Open reduction with plate and screws was performed for the humeral shaft fracture. Next, reamed interlocking intramedullary nailing was performed for the tibial fracture. Prior to lower limb surgery, a pneumatic thigh tourniquet was applied to the limb and inflated to a pressure of 350 mmHg. The intraoperative course was unremarkable with minimal blood loss.

During the entire procedure, hemo-



Fig. 1 The X-ray findings in the 42-year-old female patient showing a transverse fracture with displaced fragments of the left tibial and fibular shafts (arrow).

dynamics were stable. Upon completion of surgery, the patient was allowed to spontaneously breathe with the tracheal tube in place. The tourniquet was deflated after 84 minutes of use. Approximately five minutes after tourniquet deflation, EtCO₂ suddenly dropped from 35 mmHg to 5 mmHg, accompanied by desat-

uration (SpO_2 , 80%). At the same time, the patient's heart rate decreased from 100 beats/min to 50 beats/min with a blood pressure of 95/55 mmHg. Intravenous atropine (1 mg) and epinephrine (1 mg) were administered for unstable hemodynamics, resulting in a heart rate elevation to 115 beats/min with a blood pressure of 130/90 mmHg. Central venous and an arterial access routes were immediately established via femoral vein and radial artery, respectively. Initial arterial blood gas analysis showed a pH of 6.839, PaO_2 of 50.5 mmHg, PaCO_2 of 93.7 mmHg, a base deficit of 18.3, and hemoglobin concentration of 9.3 g/dL. Physical examination revealed clear and equal bilateral breath sounds. Although there was a short-term improvement in hemodynamic status, the value of EtCO_2 remained low (~14 mmHg) with SaO_2 of 80%. Shortly thereafter, the patient redeveloped severe bradycardia and cardiac arrest. The patient died despite immediate and vigorous cardiopulmonary resuscitation. Consent for autopsy was refused by her family, and a definite cause of death was not established.

Discussion

The findings of hypocapnia and a marked increase in dead space ($\text{PaCO}_2 - \text{EtCO}_2 = 88.7$ mmHg) led to the suspicion of massive PE in our case.⁷ Concurrent metabolic acidosis and vasodilation following tourniquet deflation may have further predisposed our patient to rapid development of unstable hemodynamics.⁸ In previous case reports^{5,6} and the present case, the fulminant progression of PE did not allow the anesthesiologists to make an early diagnosis in hemodynamically compromised patients. A definite diagnosis of PE may be established only by autopsy.

In our case, the thromboembolus may have formed from venous stasis induced by tourniquet inflation. The release of embolic material into the circulation after tourniquet

deflation following lower extremity surgery had been reported in several studies.¹⁻⁴ The use of a tourniquet may predispose patients to a five-fold greater risk of embolism compared with that in orthopedic surgery performed without a tourniquet.⁴ However, it is difficult for clinicians to identify high-risk patients. The incidence of these embolic events may be unrelated to patient characteristics, degree of immobilization, type of surgical procedure, duration of tourniquet inflation, and the use of anticoagulant medication.^{2,3}

Intramedullary nailing is a commonly used method for the treatment of shaft fractures of long bones, but this procedure may be associated with the risk of PE.⁹ When the intramedullary pressure becomes higher than the venous pressure of the feeding vessels of the bones, fat globules can be pushed into the vein and embolize the lung vessels. Therefore, fulminant fat embolism is another possible diagnosis in our patient who underwent intramedullary nailing for tibial fracture. Sathiyakumar et al. reported that the type of surgery may affect the rates of deep vein thrombosis (DVT)/PE in a total of 27,441 hip fracture patients.¹⁰ They found that patients undergoing intramedullary nailing had the highest rates of DVT/PE compared with those receiving other procedures for femoral fractures. In our case, it is unclear whether this complication could be avoided if intramedullary nailing was not performed.

The previously reported cases^{5,6} and the present case share some common features. First, the patients in most cases appeared healthy without notable predisposing factors of PE (e.g., a history of thromboembolism) that required thromboembolic prophylaxis. Second, most of these cases developed cardiopulmonary impairment within the first five minutes of tourniquet deflation, followed by immediate cardiopulmonary collapse. Persistent cardiopulmonary collapse usually indicates a significant increase in mortality.

In a review of all cases reported in the literature (N = 146), Visnjevac et al. assessed the use of different monitoring tools, including capnography, central venous pressures, echocardiography, and vital signs, in the diagnosis of intraoperative PE and the impact of each tool on mortality.¹¹ They observed that the perioperative use of trans-esophageal echocardiography (TEE) did not significantly affect mortality in patients with massive PE. In contrast, detection of reduced EtCO₂ as a sign of massive PE may be associated with lower mortality. In a meta-analysis on totally 2,291 subjects in 14 trials, the use of capnography to diagnose PE had a sensitivity of 0.80 and a specificity of 0.49.⁷ As routine use of TEE for embolic detection is uncommon, the monitoring of the PaCO₂-to-EtCO₂ gradient would be important for anesthesiologists in the management of patients undergoing operation involving the use of tourniquet.

D-dimer concentrations are highly sensitive for detecting thrombus formation and are often used to rule out venous thromboembolism (PE or DVT) in patients suspected of having the condition. In patients with a high clinical probability, imaging examination is often warranted to confirm or refute the diagnosis of PE. In our patient with no past history of thromboembolic events, we should have determined D-dimer concentrations when cardiopulmonary profiles became unstable. However, the fulminant course of this complication hindered us from sampling blood for further studies at that moment. We suggest that serum D-dimer concentration should be analyzed routinely in every subject who suffered from cardiopulmonary collapse, severe desaturation, and a widened PaCO₂-to-EtCO₂ gradient.

Current treatment of PE is still conservative. Extracorporeal membrane oxygenation is often used as a bridging treatment for emergent PE when conventional treatment fails and surgical embolectomy may be indicated. As

the fatality rate following massive PE is very high, early detection and prevention of emboli formation must remain a priority in clinical practice. Thromboembolic prophylaxis prior to tourniquet inflation is recommended by some authors to reduce the incidence of fatal PE associated with total knee arthroplasty (TKA). However, other studies have demonstrated that thromboembolic prophylaxis does not affect the occurrence of embolic events^{2,3} as some of these emboli may be composed of fat rather than thrombus. A cumulative meta-analysis on totally 3,482 patients in 14 trials also suggested that the role of thromboembolic prophylaxis in the prevention of symptomatic PE is uncertain after TKA.¹²

In general, the consequences of acute PE become apparent when > 30% – 50% of the pulmonary arterial bed is occluded by thromboemboli. Analysis of isolated cases suggests that significant symptoms of PE usually occur within the first five minutes after tourniquet deflation.^{5,6} As the sensitivity of capnography in the diagnosis of PE is 0.80,⁷ we suggest that the PaCO₂-to-EtCO₂ gradient should be monitored intensively if cardiopulmonary profiles become unstable on deflation of the tourniquet. The severity of PE caused by mechanical obstruction of the lung vessels may correlate with the number of emboli in circulation. If symptomatic PE is suspected, the tourniquet may be re-inflated temporarily to avoid more emboli being released into the circulation. A diagnostic work-up should be immediately performed to exclude the possibility of PE. If a diagnosis of PE is made, a Greenfield filter may be inserted before tourniquet deflation.

In conclusion, physicians involved in treating patients presenting with sudden cardiopulmonary destabilization after tourniquet deflation should keep in mind the possibility of PE. The PaCO₂-to-EtCO₂ gradient should be closely monitored if cardiopulmonary profiles become unstable after tourniquet deflation.

References

1. Parmet JL, Horrow JC, Singer R, et al: Echogenic emboli upon tourniquet release during total knee arthroplasty: pulmonary hemodynamic changes and embolic composition. *Anesth Analg* 1994;79:940-5.
2. McGrath BJ, Hsia J, Boyd A, et al: Venous embolization after deflation of lower extremity tourniquets. *Anesth Analg* 1994;78:349-53.
3. Berman AT, Parmet JL, Harding SP, et al: Emboli observed with use of transesophageal echocardiography immediately after tourniquet release during total knee arthroplasty with cement. *J Bone Joint Surg Am* 1998;80:389-96.
4. Parmet JL, Horrow JC, Berman AT, et al: The incidence of large venous emboli during total knee arthroplasty without pneumatic tourniquet use. *Anesth Analg* 1998;87:439-44.
5. Bharti N, Mahajan S: Massive pulmonary embolism leading to cardiac arrest after tourniquet deflation following lower limb surgery. *Anaesth Intensive Care* 2009;37:867-8.
6. McGrath BJ, Hsia J, Epstein B: Massive pulmonary embolism following tourniquet deflation. *Anesthesiology* 1991;74:618-20.
7. Manara A, D'hoore W, Thys F: Capnography as a diagnostic tool for pulmonary embolism: a meta-analysis. *Ann Emerg Med* 2013;62:584-91.
8. Samii K, Elmelik E, Mourtada MB, et al: Intraoperative hemodynamic changes during total knee replacement. *Anesthesiology* 1979;50:239-42.
9. Högel F, Gerlach UV, Südkamp NP, et al: Pulmonary fat embolism after reamed and unreamed nailing of femoral fractures. *Injury* 2010;41:1317-22.
10. Sathiyakumar V, Greenberg SE, Jahangir AA, et al: Impact of type of surgery on deep venous thrombi and pulmonary emboli: a look at twenty seven thousand hip fracture patients. *Int Orthop* 2015;39:2017-22.
11. Visnjevac O, Pourafkari L, Nader ND: Role of perioperative monitoring in diagnosis of massive intraoperative cardiopulmonary embolism. *J Cardiovasc Thorac Res* 2014;6:141-5.
12. Brookenthal KR, Freedman KB, Lotke PA, et al: A meta-analysis of thromboembolic prophylaxis in total knee arthroplasty. *J Arthroplasty* 2001;16: 293-300.