Successful Management of Intraoperative Acute Bilateral Pulmonary Embolism in a High Grade Astrocytoma Patient

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Patient: Female, 39
Final Diagnosis: Acute bilateral pulmonary embolism
Symptoms: Headache • amnesia • seizure • urinary incontinence
Medication: —
Clinical Procedure: —
Specialty: Anesthesiology

Objective: Management of emergency care
Background: Intraoperative pulmonary embolism (PE) is a rare life-threatening complication in patients undergoing surgical intervention. Generally, cancer patients have a higher risk for developing this complication. Unfortunately, there is no standard procedure for its management.

Case Report: We report the case of a 39-year-old woman with high-grade glioma in the right frontal lobe who was admitted to the surgical theater for craniotomy and excision of the tumor. During the general anesthesia procedure and just before inserting the central venous line, her end-tidal CO₂ and O₂ saturation dropped sharply. The anesthesiologist quickly responded with an aggressive resuscitation procedure that included aspiration through the central venous line, 100% O₂, and IV administration of ephedrine 6 mg, colloid 500 mL, normal saline 500 mL, and heparin 5000 IU. The patient was extubated and remained in the supine position until she regained consciousness and her vital signs returned to normal. Subsequent radiological examination revealed a massive bilateral PE. A retrievable inferior vena cava (IVC) filter was inserted, and enoxaparin anticoagulant therapy was prescribed to stabilize the patient’s condition. After 3 weeks, she underwent an uneventful craniotomy procedure and was discharged a week later under the enoxaparin therapy.

Conclusions: The successful management of intraoperative PE requires a quick, accurate diagnosis accompanied with an aggressive, fast response. Anesthesiologists are usually the ones who are held accountable for the diagnosis and early management of this complication. They must be aware of the possibility of such a complication and be ready to react properly and decisively in the operation theater.

MeSH Keywords: Resuscitation • Neurosurgery • Enoxaparin • Anesthesia • Vena Cava Filters

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Background

Intraoperative pulmonary embolism (PE) is a rare complication in patients undergoing surgical intervention. Its incidence ranges from 0.3% to 30% in the different surgical populations, with the highest incidence being in orthopedic surgical patients [1]. For example, the incidence of intraoperative PE in patients undergoing neurosurgical procedures is less than 4%. It has been shown that patients with tumors are at higher risk for developing PE [1]. In addition, it has been encountered in patients undergoing vascular hemodialysis [2].

Despite its rare incidence, PE is still a serious life-threatening problem. It has been reported that about 2–6% of patient mortality can be attributed to stable PE, while more than 30% of patient mortality can be attributed to hemodynamically unstable PE [3]. Gorek Dilektasli et al. [4] reported that PE is the main cause of mortality in patients with right ventricular dysfunction.

Anesthesiologists are usually the ones who are held accountable for the diagnosis and early management of intraoperative PE. However, there is no standard procedure for the management of this complication [5]. Here we present a case of intraoperative PE in a high-grade astrocytoma patient that was successfully managed without any further complications. It should be emphasized that this patient had a higher risk for PE due to her cancer.

Case Report

A 39-year-old female was admitted to King Abdullah University Hospital complaining of recurrent headaches, mainly bifrontal, and amnesia as noticed by family members for 3 months. The symptoms were exacerbated into recurrent episodes of generalized tonic-clonic seizures, urinary incontinence, and behavioral changes 2 weeks before admission. There was no history of recent trauma or falling down. In addition, no fever or weight loss was documented. The patient was a nonsmoker and was not receiving any medication. The only reported finding in her medical history was a cholecystectomy procedure few years ago. The neurological examination did not show any positive sign except for mild left upper limb weakness and pronator drift. However, magnetic resonance imaging (MRI) with contrast media revealed a large right frontal intra-axial well-defined nonenhancing lesion with areas of hemorrhage surrounded by extensive vasogenic edema that compressed the lateral ventricles and induced a midline shift to the left by about 1 cm (Figure 1). The differential diagnosis was consistent with a glial tumor. A surgical operation was scheduled by the neurosurgeon in order to remove the tumor.

On admission of the patient to the operating theater, a brief history was taken by the anesthesiologist for confirmation. Upon connection to monitoring devices (electrocardiogram [ECG], pulse oxymetry, and noninvasive blood pressure), the anesthesiologist noticed that the patient underwent sinus tachycardia. He directly administered 2 mg of midazolam to reduce the anxiety of the patient. Tachycardia persisted, and the anesthesiologist was reluctant to proceed. However, the O₂ saturation was 98% on room air. The anesthesiologist started pre-oxygenating the patient for 3 min, and then he gave her general anesthetic agents through a previously inserted cannula in the dorsum of her hand. The anesthetic agents consisted of 150 µg of fentanyl, followed by 200 mg of propofol, followed by 50 mg of rocuronium.

Once the patient lost consciousness and relaxation of muscles started, the anesthesiologist intubated the trachea with a cuffed, reinforced 7.5 mm internal diameter endotracheal tube. The chest was auscultated, and good bilateral air entry was confirmed. After that the anesthesiologist started to insert the invasive monitors. He cannulated the left radial artery after performing Allen’s test. Just before inserting the central venous line through the right subclavian vein, the anesthesiologist noticed a sharp fall in end-tidal CO₂. The O₂ saturation dropped to 81% and blood pressure to 90/50.
The central line was quickly inserted and the anesthesiologist started to aspirate through the cannula. However no air was noticed. Thus, the anesthesiologist suspected the presence of fat or thrombotic embolism because there was no open wound and no gas in the aspirated needle. Resuscitation protocols was performed with (1) 100% O$_2$ via endotracheal tube, (2) IV ephedrine 6 mg, (3) colloid IV infusion 500 mL, (4) IV normal saline 500 mL, and (5) IV heparin 5000 IU. The patient remain sedated in the supine position until the muscle relaxant wore off. After that the patient was extubated, regained consciousness, obeyed commands, and breathed spontaneously. Vital signs returned to normal and O$_2$ saturation reached 100% under O$_2$ face mask.

The patient was transferred to the radiology department for an urgent chest computed tomography (CT) scan. Contrast CT images revealed extensive filling defects in the main right (Figure 2A) and left (Figure 2B) pulmonary arteries and their primary branches. These findings were consistent with massive bilateral PEs. The patient was sent to the intensive care unit (ICU), where she started to receive subcutaneous enoxaparin 1 mg/kg twice daily. A retrievable inferior vena cava (IVC) filter was inserted, and the patient was kept under observation in the ICU for 2 weeks, until her condition was stabilized.

After that, the patient was transferred to the neurosurgical ward and remained there for additional week to acclimatize for reoperation. Enoxaparin was suspended, and 12 hours later the patient underwent an uneventful right frontal craniotomy and excision of the tumor. Histopathological examination revealed grade III anaplastic astrocytoma. Enoxaparin was continued after the operation, and 1 week later the patient was discharged with a scheduled 1-week follow-up appointment.

**Discussion**

The source of PE in general is either thrombotic, gaseous, fatty, or detached tumor fragments. The most probable source of PE in our patient was thrombotic, caused by the presence of high-grade astrocytoma. As mentioned earlier, patients with tumors are at higher risk for developing thrombotic PE. This is due to the general hypercoagulable state in cancer patients that is induced by hormones secreted from the tumor tissue or by some chemotherapeutic agents. In addition, the reduced activity of cancer patients may help predispose them to thrombotic embolism [1].

Fatty emboli most commonly occur after orthopedic surgeries that involve long bone fractures and intramedullary procedures. However, they can occur after other conditions such as fatty liver, bariatric procedures, burns, and sickle cell disease [6]. The main two hypothesized etiopathological mechanisms are the mechanical and biochemical theories. The mechanical theory implies physical obstruction of pulmonary vessels by embolized fat particles from the bone marrow at the fracture site, due to increased intramedullary pressure. The biochemical theory implies the release of free fatty acids that instigate
an inflammatory response in the lungs that leads to endothelial injury accompanied by interstitial hemorrhage and edema. This theory could explain the occurrence of atraumatic pulmonary fatty embolism [7].

Gaseous embolism is due to air entering the systemic circulation, where it travels and lodges in the pulmonary vessels [8]. The cause is usually iatrogenic, including various surgical procedures, insertion and disconnection of a central venous catheter, pacemaker placement, and positive pressure ventilation [8,9]. In addition, it might occur as a result of direct chest injuries [9].

The possibility of air embolism in this patient was excluded, since the complication signs started before the central venous catheterization and no air was detected during aspiration. Furthermore, there was a low possibility of fat embolism since the patient has no orthopedic trauma or other susceptible conditions. The origin of the embolism in this patient was most likely thrombotic, supported by the presence of high-grade glioma. As mentioned earlier, the presence of malignancy rendered the patient highly susceptible to developing blood clots. In addition, PE is considered a major complication in patients with intracranial tumors. It has been reported that patients with high-grade gliomas have a twofold increased risk for thromboembolisms [10].

We suggest that a prediction test must be applied to estimate the risk of PE in tumor patients undergoing surgical intervention. Two scoring tests can be used by anesthesiologists: the Modified Wells Scoring System and the Revised Geneva Scoring System [11,12]. Based on the accumulative score of either of these tests, patients can be classified as having low, intermediate, or high risk for PE. In intermediate-risk patients, as in our case, we recommend the use of a prophylactic anticoagulant (enoxaparin) prior to the operation. However in high-risk patients, the operation must be terminated, and investigation with chest angiographic CT must be performed [13].

Conclusions

Intraoperative PE is one of the major complications expected in brain tumor patients. We recommend that a scoring system test to be applied prior to the operation to determine the PE risk level. Based on the risk level found, the anesthesiologist can either proceed with the standard protocol (low risk), administer a prophylactic anticoagulant (intermediate risk), or terminate the operation and investigate with chest CT (high risk). However, anesthesiologists must be aware of the possibility of such a complication and be ready to react quickly and decisively in the operation theater.

Conflict of interest

None.

References:

1. Desciak MC, Martin DE: Perioperative pulmonary embolism: Diagnosis and anesthetic management. J Clin Anesth, 2011; 23(2): 153–65.
2. Sadjadi SA, Sharif-Hassanabadi M: Fatal pulmonary embolism after hemodialysis vascular access declotting. Am J Case Rep, 2014; 15: 172–75.
3. Klok FA, Zondag W, van Kralingen KW et al: Patient outcomes after acute pulmonary embolism. A pooled survival analysis of different adverse events. Am J Respir Crit Care Med, 2010; 181(5): 501–6.
4. Gorek Dilektasli A, Demirdogen Cetinoglu E, Acet NA et al: Catheter-directed therapy in acute pulmonary embolism with right ventricular dysfunction: A promising modality to provide early hemodynamic recovery. Med Sci Monit, 2016; 22: 1265–73.
5. Hsieh PC, Wang SS, Ko WJ et al: Successful resuscitation of acute massive pulmonary embolism with extracorporeal membrane oxygenation and open embolectomy. Ann Thorac Surg, 2001; 72(1): 266–67.
6. Mellor A, Soni N: Fat embolism. Anaesthesia, 2001; 56(2): 145–54.
7. Newbiggin K, Souza CA, Torres C et al: Fat embolism syndrome: State-of-the-art review focused on pulmonary imaging findings. Respir Med, 2016; 113: 93–100.
8. Soulios JP, Paris P, Braccio M et al: Pulmonary gas embolism after parotid resection. Acta Anaesthesiol Belg, 2015; 66(2): 55–57.
9. Chao CM, Liu WL, Hsieh CF, Lai CC: Pulmonary air embolism. QJM, 2016; 109(4): 283–84.
10. Chaichana KL, Pendleton C, Jackson C et al: Deep venous thrombosis and pulmonary embolisms in adult patients undergoing craniotomy for brain tumors. Neurol Res, 2013; 35(2): 206–11.
11. Douma RA, Gibson NS, Gerdes VE et al: Validity and clinical utility of the simplified Wells rule for assessing clinical probability for the exclusion of pulmonary embolism. Thromb Haemost, 2009; 101(1): 197–200.
12. Klok FA, Mos IC, Nijkeuter M et al: Simplification of the revised Geneva score for assessing clinical probability of pulmonary embolism. Arch Intern Med, 2008; 168(19): 2131–36.
13. Raja AS, Greenberg JO, Qaseem A et al: Evaluation of patients with suspected acute pulmonary embolism: Best practice advice from the Clinical Guidelines Committee of the American College of Physicians. Ann Intern Med, 2015; 163(9): 701–11
14. Desai SS, Naddaf A, Pan J et al: Impact of consensus statements and reimbursement on vena cava filter utilization. J Vasc Surg, 2016; [Epub ahead of print]
15. Bikdeli B, Wang Y, Minges KE et al: Vena caval filter utilization and outcomes in pulmonary embolism: Medicare hospitalizations from 1999 to 2010. J Am Coll Cardiol, 2016; 67(9): 1027–35
16. Davies MG, Hart JP, El-Sayed HF: Efficacy of prophylactic inferior vena caval filters in prevention of pulmonary embolism in the absence of deep venous thrombosis. J Vasc Surg Venous Lymphat Disord, 2016; 4(1): 127–130.e1
17. PREPIC Study Group: Eight-year follow-up of patients with permanent vena cava filters in the prevention of pulmonary embolism: The PREPIC (Prevention du Risque d’Embolie Pulmonaire par Interruption Cave) randomized study. Circulation, 2005; 112(3): 416–22