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Unexpected Pulmonary Embolism Following Microvascular Head and Neck Reconstruction : Report of Two Cases and Review of the Literature

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유리피판을 이용한 두경부 재건 후 발생한 폐색전증 2예 및 문헌고찰

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= **국문초록** =

유리피판을 이용한 두경부 재건후 폐색전은 드물게 발생한다. 수술후 예상하기 힘들고 갑작스럽게 발생하기 때문에 환자가 아주 위험한 상황에 빠질 수 있다. 즉각적인 발견과 적절한 치료만이 환자의 생명을 구할 수 있다. 유리피판을 이용한 두경부 재건후 발생한 폐색전증 2예를 문헌고찰과 함께 보고하는 바이다.

중심 단어 : 폐색전증 · 재건 · 두경부암.

Introduction

Venous thromboembolism is uncommon in patients following otolaryngologic surgery. Pulmonary embolism(PE), most commonly originating from deep venous thrombosis (DVT) of the legs, may occur unpredictably in patients undergoing microvascular free flap reconstruction. PEs range from asymptomatic state, incidentally discovered emboli to massive emboli which can cause hemodynamic instability and immediate death.[1] Many head and neck surgeons seem to overlook this potential complication because it happens only rarely. However, immediate detection and proper management of this disastrous complication may save a patient's life. In this brief report, we describe two cases of patients who underwent microvascular head and neck reconstruction and subsequently developed PEs several days after surgery. In addition, we review the literature regarding risk factors, diagnostic approaches, treatment, and prevention.

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Case Report

1. Case 1

A 64-year-old woman visited our clinic with history of trismus for several months and history of a neck mass for several years. She was moderately obese at 63.1 kg with a height of 142 cm(body mass index 31.3). Her past medical history was unremarkable except for hypertension and there was no family history of a bleeding disorder or coagulopathy. Pathology reported her buccal mass as an adenocarcinoma ; clinical staging was T4aN2aM0. She received wide excision of the buccal mass and hemimandibulectomy with combined modified radical neck dissection for an oral cavity carcinoma with neck metastases. The oral and mandibular defects were reconstructed using the fibular osteocutaneous free flap.

The patient was placed in a short-leg splint after surgery. We applied stockings routinely on both legs. Nine days following the surgery she experienced a sudden onset of chest discomfort. Her blood pressure was 105/67 mmHg and her pulse was 139 beats/min. Chest radiography was unremarkable and electrocardiography initially showed sinus tachycardia. Twenty minutes later she developed cyanosis. Her partial arterial oxygen pressure(PO_2) was 43.2 mmHg and her O_2 Saturation was 49.5%. Oxygen was supplied immediately. Echocardiography showed right ventricular dysfunction and massive thrombi in the main pulmonary artery. The reported value of the ELISA D-dimer was 42,097 ng/mL. The CT angiogram was unable to be taken due to the patient's unstable vital signs. Her diagnosis was a massive PE with Echocardiography. Low-molecular-weight heparin therapy was started immediately. However, within half an hour her blood pressure was not recordable and there was no palpable carotid pulse. She was ventilated with 100% oxygen and cardiopulmonary resuscitation was initiated. Despite the attempted resuscitation she died of cardiac arrest.

2. Case 2

A 52-year-old woman visited our clinic with history of a tongue base mass for several weeks and history of a neck mass for several months. Her past medical history was unremarkable and there was no family history of a bleeding disorder or coagulopathy. Pathology reported the tongue base mass as squamous cell carcinoma ; clinical staging was T2N1M0. She was treated with wide tongue excision via paramedian mandibulotomy with combined bilateral neck dissection for cure of tongue SCC. The tongue defects were reconstructed using the radial forearm free flap. She was not given anticoagulant prophylaxis preoperatively. She abruptly developed breathing difficulties and chest pain on two days following the surgery. She was alert and oriented with a blood pressure of 134/80 mmHg and a pulse of 86 beats/min. Her physical examination revealed no clinical evidence suggestive of deep vein thrombosis. Chest radiography was unremarkable and electrocardiography showed sinus tachycardia. Her arterial PO_2 was 64.0 mmHg and her O_2 saturation was 93.7%. Echocardiography showed no specific findings. The reported value of the enzyme-linked immunosorbent assay(ELISA) D-dimer was 579 ng/mL. She was diagnosed as having a pulmonary embolism. This was confirmed by a contrast-enhanced CT angiogram(Fig. 1). She was hospitalized for 4 weeks and treated with low-molecular-weight heparin for 3 weeks followed by warfarin. Following discharge from the hospital she was asymptomatic and had no further pulmonary complications.

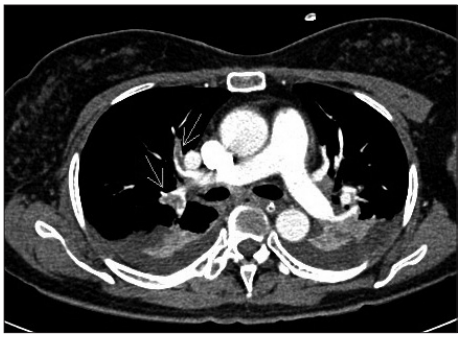

Fig. 1. Contrast-enhanced CT angiogram of the pulmonary arteries with intraluminal filling defects in the segmental artery of the right upper lobe.

Discussion

The incidence of PE following microvascular head and neck reconstruction for the treatment of head and neck cancer is unknown. PE occurs in up to 50% of patients with proximal vein thrombosis.[1)] In the last 17 years(1991−2007) at our institution we have observed the occurrence of significant pulmonary embolism in only 2 of 400(0.5%) patients who underwent microvascular free flaps for reconstruction of oncologic defects.

The pathogenesis of deep vein thrombosis(DVT) is related primarily to hypercoagulability and venous stasis.[2)] Advanced age is another clear risk factor, with the risk increasing after the age of 40 years.[1)] Obesity(body mass index >30) also increases the risk of venous thrombosis.[3)] Major surgery and reduced mobility of the body confer an increased risk.[3)] Interestingly, malignant cells can activate blood coagulation in several ways.[4)] The length of the procedure for microvascular reconstruction of oncologic defects can also increase the risk of DVT and PE. Moreover, as was true in our cases, free composite flap reconstruction needs more immobilization time, thereby increasing the risk for DVT and PE.

Sudden onset dyspnea is the most frequent symptom in patients with PE. Chest pain and fainting frequently occur.[5)] Tachypnea and tachycardia are common but nonspecific findings. The possibility of a massive pulmonary embolism should be considered in patients who have a sudden onset of syncope, hypotension, extreme hypoxemia, or cardiac arrest after surgery.[1)]

Once a pulmonary embolism is suspected, a careful assessment based on the history, physical examination, and known risk factors should be performed. Additional studies including electrocardiography, chest radiography, and arterial blood gas

analysis should also be considered. A sudden or unexplained change in arterial oxygen saturation should raise the clinician's suspicion of PE. D-dimer assays have superior sensitivity (98% or higher) for venous thromboembolism.[6,7] Contrast-enhanced CT angiograms have advantages over ventilation-perfusion scanning, including speed, characterization of non-vascular structures, and detection of venous thrombosis.[7] Echocardiography is valuable in differentiating between massive PE and other causes of hemodynamic compromise.[6]

Initial treatment with low-molecular-weight heparin(LMWH) is recommended in suspicious or confirmed PE.[8] Documented thromboembolism in patients with transient risk factors should be treated for 3 to 6 months.[1]

In patients who undergo microvascular head and neck reconstruction and have dyspnea or chest pain postoperatively, the surgeon should be suspicious of the development of PE. Early identification and immediate management is crucial. If a patient is undergoing major head and neck reconstruction surgery and has risk factors for the development of postoperative PE, parenteral anticoagulation therapy with LMWH may be effective for the prevention of PE.

PE has important clinical implications and a high mortality rate of about 15−18%.[7] To lessen the risk of PE, early mobilization, anticoagulant prophylaxis, and lower limb mechanical prophylaxis should be considered in patients who undergo microvascular head and neck reconstruction. More importantly, the head and neck surgeon should keep in mind the possibility of the patient developing PE after this lengthy procedure.

Conclusions

Because the signs and symptoms of PE are nonspecific, diagnosing PE on purely clinical grounds is very difficult. Knowledge and identification of the risk factors reviewed in our two cases may make it possible to reduce the incidence of DVT and PE following microvascular head and neck reconstruction. Proper treatment of PE may reduce mortality ; postoperative anticoagulant prophylaxis may be effective in these patients.

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