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CASE REPORT

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A case of asymptomatic acute pulmonary embolism due to deep venous thrombosis after total knee arthroplasty in a patient with rheumatoid arthritis

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Abstract In the field of orthopedics, acute pulmonary embolism (APE) associated with deep venous thrombosis (DVT) is a serious complication following surgery and leads to death if undetected. Although an examination for the presence of APE after surgery has been well established in other countries, there are few reports on APE after orthopedic surgery in Japan. Here, we describe a case of asymptomatic APE associated with DVT after total knee arthroplasty (TKA) in a patient with rheumatoid arthritis (RA). Because it is difficult to determine the clinical features of APE prior to the initiation of angiography, we used a perfusion lung scan, which is a useful tool for detecting asymptomatic APE. We successfully identified APE in the TKA patient with RA, and continuous intravascular infusion of a thrombolytic agent and an anticoagulant was an effective treatment in this case. Our report clearly shows that a well-established procedure for diagnosis, as well as therapeutic guidelines, are essential for detecting systemic thromboembolism in patients after orthopedic surgery.

Key words Acute pulmonary embolism · Deep venous thrombosis · Rheumatoid arthritis (RA) · Thrombolytic therapy · Total knee arthroplasty (TKA)

Introduction

Clinically, deep venous thrombosis (DVT) occurs in patients after surgery, particularly in the orthopedic field. In Europe and the United States, the DVT frequency has been reported to be 34%–63% after total hip arthroplasty (THA),[1–7] and 41%–88% after total knee arthroplasty (TKA)[8–12] in the absence of prophylaxis.

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In Japan, the frequency of DVT or acute pulmonary embolism (APE) after an operation such as THA and TKA has gradually been increasing as a serious complication. Although the continuous low-dose intravenous infusion of anticoagulants such as low-molecular-weight heparin has been shown to be an effective treatment for DVT, systematic therapy has not yet been well established. In this report, we describe one case of DVT in a patient following TKA. This report describes the risks of developing APE or DVT after TKA. The condition is apt to be misdiagnosed, since a patient with APE does not always exhibit any subjective symptoms.

Case report

A 56-year-old woman presented with pain and swelling in the joint of her right knee of 9 months' duration. A diagnosis of rheumatoid arthritis (Steinbrocker's classification,[13] stage IV, class III) had been made 5 years previously based on the presence of morning stiffness, systemic arthritis, and a high rheumatoid factor titer. There was no deficiency in antithrombin-III, protein C, or protein S. The patient was negative for the presence of anticardiolipin antibodies or lupus anticoagulants.

We performed a right TKA using an air tourniquet and bone cement for severe RA. The operating time was about 1.5 h, with little blood loss. Arterial blood gas samples obtained at atmospheric pressure had a PaO_2 of 43–46 mmHg, a $PaCO_2$ of 33–35 mmHg, and oxygen saturation of 80%–82% for 3 days after the TKA (Fig. 1). Arterial blood gas samples obtained when the patient was receiving 6 l oxygen per minute revealed a PaO_2 of 66 mmHg, a $PaCO_2$ of 35 mmHg, and oxygen saturation of 85%. Moderate pitting edema of the right lower extremities, Homans' sign, and severe pain following compression of the lower legs (Lowenberg's sign) were observed immediately after surgery. We assumed that they were clinical signs of DVT. However, she presented no specific DVT symptoms such as dyspnea, short-windedness, cough, bloody sputum, chest

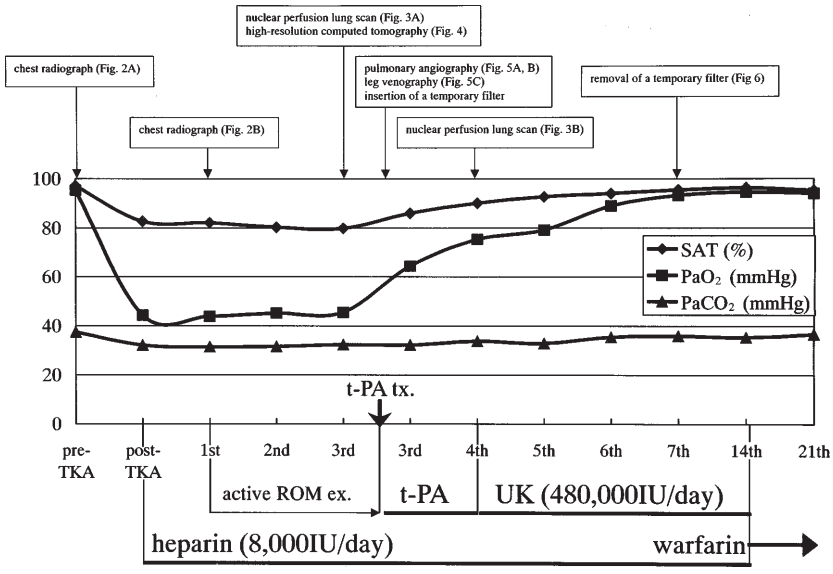

Fig. 1. Blood-gas analysis showing hypoxemia before thrombolytic treatment with tissue-type plasminogen activator (*t-PA*), urokinase (*UK*), and warfarin. Hypoxemia was indicated by a PaO_2 of 43–46 mmHg, a $PaCO_2$ of 33–35 mmHg, and oxygen saturation (*SAT*) of 80%–82% for 3 days after total knee arthroplasty (*TKA*)

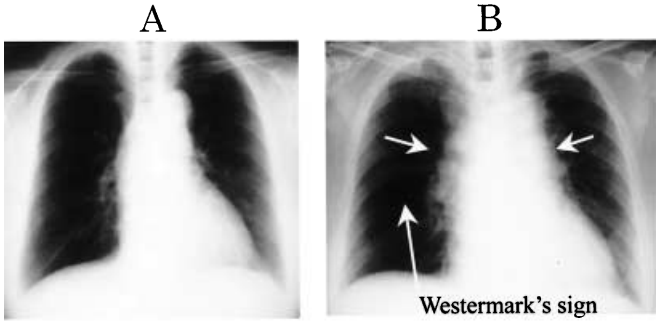

Fig. 2. **A** Chest radiograph showing clear lung fields before TKA. **B** Chest radiograph on day 1 after TKA showing bilateral prominent central pulmonary arteries with Westermark's sign

pain, or other subjective symptoms. Chest radiography performed on day 1 after TKA revealed extensive bilateral cardiomegaly with prominent central pulmonary arteries (knuckle sign); in particular, a bulge in the right pulmonary artery was observed peripherally as a high radiolucent lung field, the so-called Westermark's sign[14] (Fig. 2B). An electrocardiogram performed on day 1 after TKA revealed size and function abnormalities of the left ventricle, with negative T waves (data not shown). A nuclear perfusion lung scan performed on day 3 after TKA showed a significant decrease in the density of the tracer in the upper-right lung upper area (Fig. 3A). High-resolution computed tomography (HRCT) of the thorax performed on day 3 after TKA showed stenosis of the right pulmonary artery consistent with pulmonary embolism (Fig. 4). Right pulmonary angiography performed on day 3 after TKA detected several vessel thrombi in the main upper region corresponding to the result obtained by perfusion lung scan (Fig. 5A). Suction elimination of the pulmonary thrombi was performed following angiography. Many thrombi formed a tight link in the deep right veins, indicating the formation of a chain of DVT (Fig. 5C).

We commenced thrombolytic treatment of the patient by continuous intravenous administration of a tissue-type plasminogen activator (t-PA) following angiography on day 3 after surgery. After urokinase (UK) infusion for 10 days at a rate of 480000 units/day, the patient recovered from hypoxemia (see Fig. 1). On day 7 following the thrombolytic therapy, a lump of DVT was trapped in a temporary filter that had been inserted in the inferior vena cava (Fig. 6). No significant changes in oxygen saturation, heart rate, or systemic blood pressure have been noted since day 7 after surgery. Fortunately, the progressive hypoxemia never recurred as a result of improvement of right lung failure, and the patient was discharged in week 6 after surgery with continued administration of warfarin to prevent the recurrence of thromboembolism.

Pathologic findings

Microscopic examination of the pulmonary thrombi showed a lining composed of endothelial cells. In some areas, the connective tissue was infiltrated, indicating fibrinoid deposition and vasculitis. Focally, there was no formation of concentric circles which suggests periodic recurrence in the past, as observed in a case with chronic thrombi (Fig. 7).

Discussion

The pathologic findings of pulmonary vessel thrombi suggest that they were formed during the acute phase after

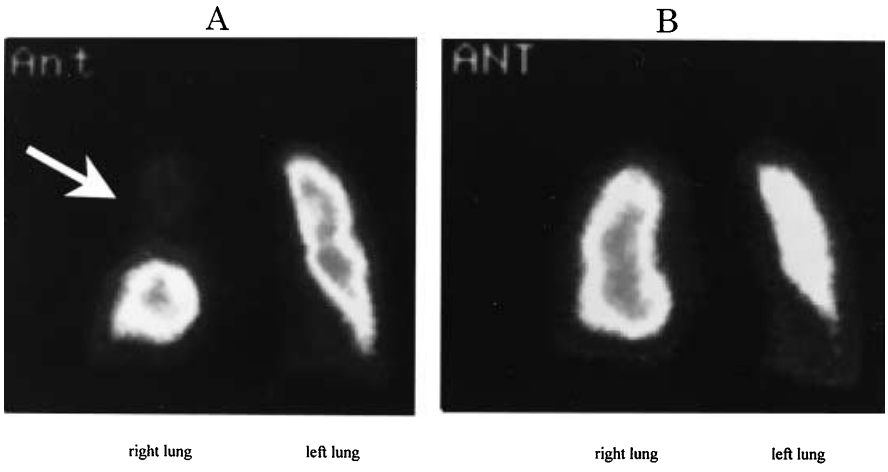

Fig. 3. **A** Perfusion lung scan on day 3 after TKA showing decreased upper perfusion with normal lower perfusion in the right lung before thrombolytic treatment. **B** Perfusion lung scan showing recovery of the blood flow in the right upper lung after thrombolytic treatment

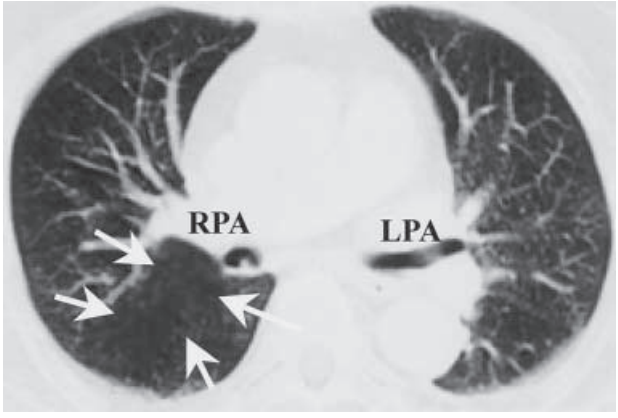

Fig. 4. High-resolution computed tomography of the thorax on day 3 after TKA showing a large mass in the right pulmonary artery (*RPA*). *LPA*, left pulmonary artery

TKA. The chest radiograph showed that the right descending pulmonary artery was enlarged as a result of APE. Enlargement of a descending pulmonary artery is highly indicative of APE. In particular, detection of a sausage-like appearance of the vessel (knuckle sign) as well as Westermark's sign should indicate a high probability of the disease.

A perfusion lung scan showed typical defects in the upper half of the right lung, corresponding to regions of pulmonary arterial thrombi. This finding may be helpful in the differential diagnosis of APE from other respiratory diseases.[15]

Right pulmonary angiography showed several filling defects with stenosis and the abrupt narrowing of peripheral branches. A proximal filling defect indicates the existence of a vessel thrombus. These findings are also highly indicative of APE.[16] Early diagnosis of APE is important, since thrombolytic therapeutic agents such as t-PA immediately improve lung perfusion disturbance in APE cases.[17]

Our patient had no specific subjective symptoms such as dyspnea, short-windedness, cough, bloody sputum, or chest pain that would suggest the presence of APE before thrombolytic therapy. All findings from blood gas analysis, chest radiography, electrocardiography, HRCT, perfusion lung scan, and pulmonary angiography suggested abnormal gas exchange. The low oxygen saturation noted for 3 days following TKA may have retrospectively indicated the onset of APE. In spite of hypoxemia for 2 days after TKA, the patient had few complaints that would lead us to suspect APE. Thus, we commenced t-PA therapy when the APE diagnosis became definitive after clinical examinations.

APE remains the third most common cause of mortality following heart disease and stroke in the United States. In Japan, the frequency of APE after joint surgery is gradually increasing. We assume that there are three reasons for this. First, detailed examinations for APE have rarely been performed by orthopedists in the past unless the patient showed specific symptoms. Second, progress in medical technology means that we can now diagnose APE accurately. Third, patients with APE who have undergone such surgery tend to be elderly.

In summary, we have described asymptomatic APE associated with DVT following TKA. Joint arthroplasty is one of the risk factors for venous thromboembolism.[18] In particular, there are several reports showing that coagulation factors were activated in patients with RA.[19] The lack of typical clinical symptoms of APE at an early stage after surgery in a patient who has undergone TKA does not necessarily indicate that he or she did not contract APE. It is important to perform a blood gas analysis, chest radiography, and electrocardiography routinely for all patients who have undergone joint replacement for at least 3 days and on day 7 postoperatively. We recommend that checks should be carried out and prophylaxis for APE and DVT started as soon as possible (Fig. 8).[20] Any change in oxygenation should lead us to suspect APE even if the patient shows no specific symptoms.

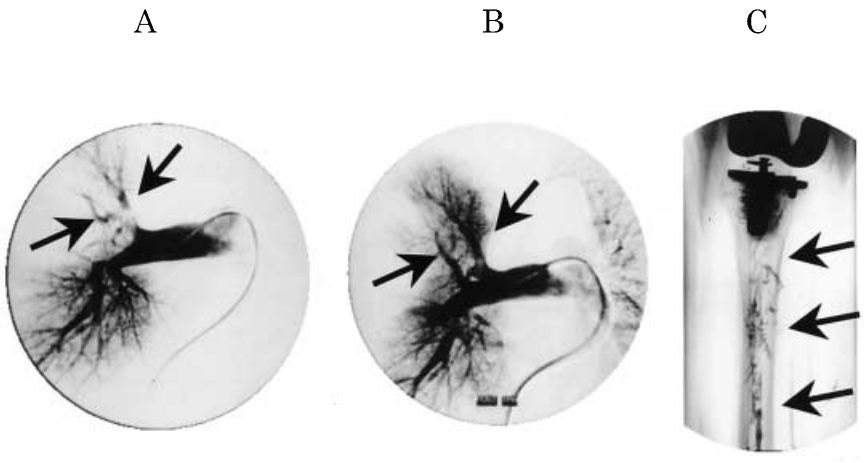

Fig. 5. **A** Pulmonary angiography on day 3 after TKA showing several obstructions in the upward blood flow before thrombolytic treatment (*arrows*). **B** Pulmonary angiography showing restoration of the blood flow after thrombolytic treatment (*arrows*). **C** Lower leg venography on day 3 after TKA showing continuous deep venous thrombosis (*arrows*)

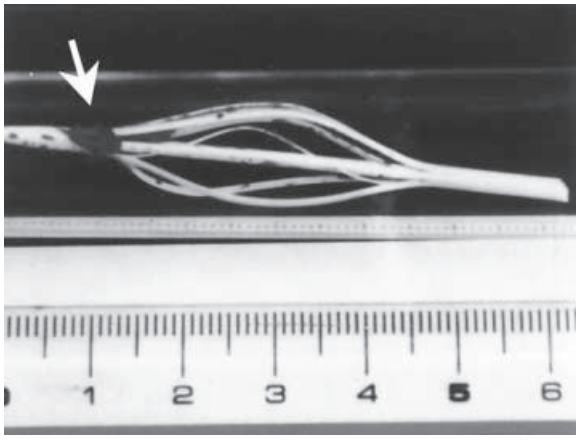

Fig. 6. A temporary filter in the inferior vena cava on day 7 after thrombolytic therapy showing entrapment of the DVT as fresh vessel thrombi

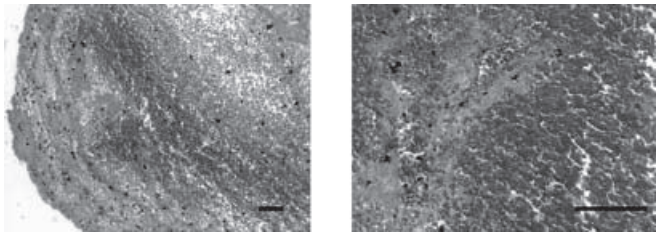

Fig. 7. Microscopic section of the pulmonary thrombi eliminated on day 3 after TKA showing a lining composed of endothelial cells (HE stain, *Bar* 100 μm)

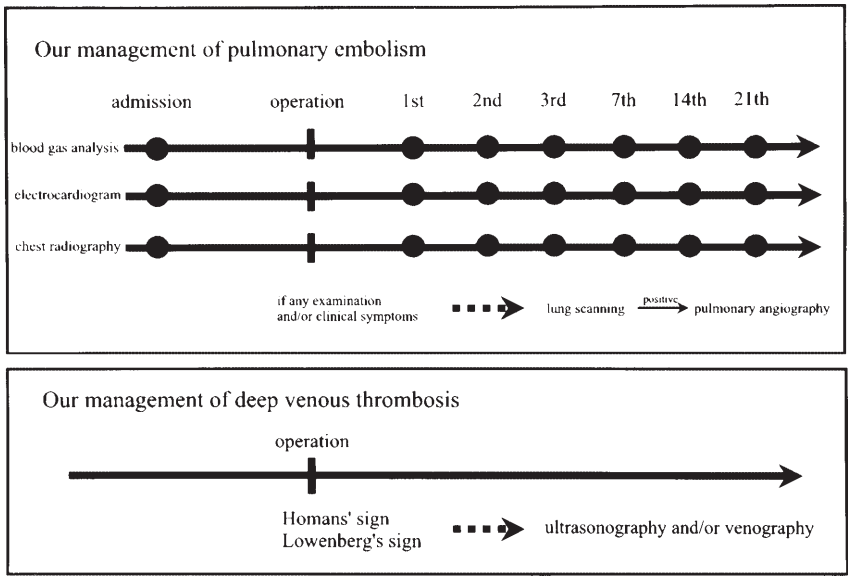

Fig. 8. Recommended routine management of orthopedic patients

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