Internal Jugular Thrombophlebitis Caused by Dermal Infection

Hisao Yoshikawa, Makoto Suzuki, Naohiko Nemoto, Hidehiko Hara, Go Hashimoto, Takenori Otsuka, Masao Moroi, Masato Nakamura and Kaoru Sugi

Abstract

A 29-year-old man presented with complaints of fever and pain and itching of his left neck with atopic dermatitis and abrasion. These symptoms had persisted for two days and dullness and fever developed, but the patient did not consult a physician. On the following day, he had a fever of $40^\circ$C and redness and swelling of the left neck. He visited a local clinic and was hospitalized with suspected cellulitis. A thrombus was detected in the internal jugular vein on MRI, and he was referred to our hospital. Neck ultrasonography showed the presence of an immovable thrombus in the area from the left internal jugular vein to the left brachiocephalic peripheral vein. Blood analysis indicated a major inflammatory response and juvenile idiopathic thrombophlebitis was suspected. A filter was carefully inserted into the superior vena cava, and anticoagulant therapy and medication with antibiotics led to remission. We present this case as a rare example of a condition mimicking Lemierre syndrome that was caused by dermal infection, and we include a review of the literature.

Key words: internal jugular vein, thrombophlebitis, lemierre syndrome, dermal infection


(DOI: 10.2169/internalmedicine.50.4612)

Introduction

Thrombophlebitis in the neck may be caused by trauma, carcinoma, infection and central vein catheter implantation (1, 2). Lemierre syndrome is a form of thrombophlebitis that is now relatively uncommon due to treatment with antibiotics (3-8). We present the case as an example of a disease mimicking Lemierre syndrome, since it involved jugular thrombosis and accompanying phlebitis with dermal infection.

Case Report

A 29-year-old man presented with complaints of fever and pain and itching in his left neck with atopic dermatitis and abrasion. Two days later, he developed dullness and fever, but did not consult a physician. On the following day he had a fever of $40^\circ$C, and redness and swelling of the left neck. He visited a local clinic and was subsequently hospitalized with suspected cellulitis. A thrombus in the jugular vein was detected by MRI, and he was referred to our hospital.

The patient had a history of atopic dermatitis and bronchial asthma, but had not received treatment. He did not smoke and he only occasionally drank alcohol. His family history was unremarkable. At the initial examination, his height was 172 cm, weight 62 kg, temperature $38.4^\circ$C, blood pressure 118/76 mmHg, and pulse rate 98/min. A dermal inspection indicated abrasion of the left neck that was in remission, but pain occurred on pressing. A physical examination indicated no abnormalities.

A blood test indicated elevation of the white blood cell count to 10,400/μL and C-reactive protein (CRP) was elevated to 22.9 mg/dL, indicating inflammation. A coagulation test indicated PT 12.4 sec., PT activity 77%, Fib. 661 mg/dL, AT-III 84%, D-D 1.2 μg/mL, and FDP 7.2 μg/mL, suggesting slight coagulopathy. There were no other abnormal data indicating coagulopathy or collagenosis. Testing for viruses, such as EB virus, was negative. Staphylococcus

Division of Cardiovascular Medicine, Toho University Ohashi Medical Center, Japan

Received for publication September 29, 2010; Accepted for publication November 1, 2010

Correspondence to Dr. Hisao Yoshikawa, hisao_yskw@yahoo.co.jp
aureus was positive in both aerobic and anaerobic bottles in a blood cultures. Electrocardiogram and chest radiography were normal.

Neck and chest CT demonstrated expansion of the left internal jugular vein, the subclavian vein and the left brachiocephalic vein (Fig. 1). There were also low density areas extending from the left internal jugular vein and from the subclavian vein to the brachiocephalic vein. The subcutaneous tissue density was increased, suggesting edema (Fig. 1). Neck ultrasonography showed the thrombus in the area from the left internal jugular vein to the left brachiocephalic peripheral vein (Fig. 2). The vein was expanded and the thrombus had an echo level that was not equal from the low to high echo profile (Fig. 2). T2-weighted MRI of the neck detected a high signal intensity lesion that extended from the skin surface to the posterior cervical space and the neck carotid sheath. A high signal area was also detected in the left brachiocephalic vein extending to the left internal jugular vein. These finding were consistent with inflammatory lesion. There were no abnormal findings in echocardiography, abdominal ultrasonography, superior/inferior limb vessel ultrasonography, and lung perfusion scintigraphy.

At the time of hospitalization, the patient had itching of the left neck, abrasion, pain upon pressing, and fever. A diagnosis of left internal jugular thrombophlebitis induced by infection of the left neck with abrasion was made based on the following evidence: increased inflammation in blood tests, expansion of the jugular vein and thrombosis on neck and chest CT and neck ultrasonography, inflammation extending from the neck carotid sheath and posterior cervical space to the skin surface on MRI, the absence of another infection source, and no hemorrhagic irregularities.

Because of the high risk of pneumothrombosis, a filter was carefully inserted into the superior vena cava, and the absence of a thrombus in the superior vena cava was confirmed. After insertion of the filter, urokinase (240,000 units), heparin and warfarin were administered from day 2 to day 9 of hospitalization. Heparin (15,000 U/day) was
started and the dose was adjusted according to the activated partial thromboplastin time (maximal dose of heparin 24,000 U/day). Warfarin (2.5 mg) was started, maintaining the PT-INR between 2.0 and 3.0. On day 10, neck CT and neck ultrasonography showed no marked changes, and therefore the filter was withdrawn on day 13. After filter withdrawal, Warfarin administration was continued. Clindamycin 1,200 mg and imipenem 2 g were administered until day 24 to treat infection. Fever remitted as inflammation decreased, and CRP turned negative on day 19. Neck CT (Fig. 3) and neck ultrasonography on day 28 indicated shrinkage of the vein and thrombus, and the patient was discharged on day 30.

**Discussion**

This patient was diagnosed as juvenile idiopathic thrombophlebitis mimicking Lemierre syndrome. Lemierre syndrome is mainly caused by tonsillitis and pharyngitis, and is a form of internal jugular thrombophlebitis induced by an anaerobe, mainly F. necrophorum (3-8). If the correct diagnosis is delayed, Lemierre syndrome may progress to poly-infection, resulting in complications and a high fatality rate (3-8).

The Lemierre syndrome is thought to progress from a primary infection that advances through the tonsillar vein, internal neck lymph system, and pharynx side wall fascia (8, 9) to the parapharyngeal space and neck carotid sheath, leading to acute jugular thrombophlebitis (5, 10). A thrombus in the jugular vein may cause metastatic infection, resulting in serious disease. In the present case, we thought that dural infection of the left neck caused internal jugular thrombophlebitis, based on MRI showing that inflammation extended from the skin surface to the posterior cervical space and the neck carotid sheath. Acute internal jugular thrombophlebitis caused by dural infection has been reported (1, 10). Based on our search, it seems that there was only one report of internal jugular vein thrombophlebitis caused by blunt neck injury (11).

The diagnosis of jugular thrombophlebitis can be performed by ultrasonography, CT and MRI. Ultrasonography does not require radiation exposure and can be performed easily, but is limited to certain areas and may miss a new thrombus. CT and MRI allow visualization of a thrombus in a vein and changes in surrounding tissues, and these methods are particularly useful for diagnosis (1, 7, 12). In the present case, expansion of the vein and thrombus were evident in ultrasonography and CT, and extension of inflammation to the surrounding tissue was detected by MRI, resulting in identification of the source of infection. So we conclude that the skin surface inflammation extended to the posterior cervical space and the neck carotid sheath resulting in thrombophlebitis.

*Staphylococcus aureus* was identified in a blood culture. Serious cases of jugular thrombolysis due to *Staphylococcus aureus* have also been reported (13-15). Standard treatment for jugular thrombophlebitis and Lemierre syndrome has not been established, but high dose penicillin, metronidazole, clindamycin, and chloramphenicol are recommended as antibiotics. However, these drugs may require 3 to 6 weeks to exert an effect (5, 10). β-lactamase-producing bacteria are increasingly being identified as causative bacteria, and thus monotherapy with penicillin alone is not recommended (10). In the present case, imipenem and clindamycin were administered since the patient had a serious infection. Since the antibiotic medication was started early, remote infection may have been avoided, and this approach resulted in remission.

Anticoagulation therapy is controversial (2, 6, 8, 10). Golpe et al reported that it may be useful if septic emboli persist despite antibiotic therapy or if thrombosis propagates to the cavernous sinus (10). Since the present patient had an acute condition and a high risk of pneumoembolism, antithrombotic drugs were administered after filter insertion in the superior vena cava. Such filter insertion has been reported to be useful, but also has a high risk of vessel damage, cardiac tamponade, and falling of the filter into the heart (2, 16). Thus, this approach to the treatment of thrombolysis requires further evaluation. However, the antithrombolytic drugs used in our case were effective in shrinking the vein and thrombus, as confirmed by neck CT and ultrasonography. This permitted successful treatment of the internal jugular thrombophlebitis caused by dermal infection.

**The authors state that they have no Conflict of Interest (COI).**

**References**

4. Iwata N, Komiya N, Uchiyama-Nakamura F, Onishi K. Lemierre...

© 2011 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html