CASE REPORT

Achromobacter causing a thrombophlebitis and osteomyelitis combination: a rare cause

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SUMMARY

Achromobacter xylosoxidans is a Gram-negative, aerobic bacillus, present in normal human flora of the skin and gastrointestinal tract. Infections due to Achromobacter are infrequent and have mostly been reported in immunocompromised patients. Rarely, however, the microorganism can cause soft tissue infections even in healthy subjects with a history of trauma. We report thrombophlebitis complicated with osteomyelitis secondary to *Achromobacter* in a 15-year-old girl with a history of purulent discharge from the ankle due to local trauma caused by tight fitting shoes.

BACKGROUND

Achromobacter xylosoxidans is an uncommon opportunistic pathogen causing many serious infections, especially in patients with cystic fibrosis and malignancies, and in immunocompromised individuals. It is a genus of bacteria in the Alcaligenaceae family; it is strictly aerobic and found in water, soil and intravenous fluids.[1 2] *Achromobacter* has rarely been associated with human infections, and its clinical significance remains unclear. To the best of our knowledge, ours is the second thrombophlebitis case, secondary to *Achromobacter*, reported in the literature.[3] There are also a few reports of osteomyelitis following thrombophlebitis, but none of them were caused by *Achromobacter*.[4 5] We aim to focus on this rare opportunistic water pathogen and emphasise the combination of osteomyelitis and thrombophlebitis.

CASE PRESENTATION

A 15-year-old girl was admitted to our clinic with a 3-day history of fever, ankle pain and difficulty walking. She had a 1-week history of purulent discharge from a lesion on her right ankle, as a result of wearing tight shoes. She was otherwise healthy, and her physical and neurological developmental stages were normal; she had been completely vaccinated. She had non-consanguineous parents with no known diseases.

On physical examination, the patient's body temperature was 37.6°C, pulse 90 bpm and blood pressure 110/70 mm Hg. She had a tendered swelling with redness and oedema of the right ankle joint region. Physical examination of other systems and joints was completely normal. The complete blood count revealed a total leucocyte count of 10 300/mm^3, haemoglobin 12.2 gr/dL and platelet count of 201 000/mm^3. Erythrocyte sedimentation rate was 66 mm/h and C reactive protein level was 18.1 mg/dL. Laboratory analyses of electrolyte, liver and renal function tests were all within normal range. Antinuclear antibody test was nucleolar positive and serum immunoglobulin levels were normal. Urinalysis revealed no pyuria, and the culture was sterile. The X-ray of the ankle joint showed no enthesopathic lesions or erosive joint damage. An MRI taken on the day of admission revealed radiographic signs of inflammatory process and infection. Empirically, we started the patient on intravenous cephalosporin and amikacin. We aspirated synovial fluid from the patient's right ankle, from which the cell count revealed 1200 leucocytes/mm^3, with neutrophil predominance, but Gram staining and culture did not yield any microorganisms. *A. xylosoxidans* was identified by blood culture taken on the day of admission, and cephalosporin was changed to intravenous meropenem. In her follow-up, the deep venous pulse of the ankle became weak. With a suspicion of septic thrombophlebitis, Doppler ultrasonography was performed, displaying increased wall thickness and obliteration of the vascular lumen for 16 mm at the distal great saphenous vein on the perimalleolar area. Echocardiography showed no pathology other than minimal mitral insufficiency.

To seek an underlying coagulopathy, coagulation panel tests were performed. The investigations revealed the following: prothrombin time: 13.4 sec, prothrombin activity 65.5%, international normalised ratio 1.2, activated partial thromboplastin time 42.5 sec, antithrombin III 110.4%, protein C 120.1%, and protein S 71.4%. All the results were normal. Lipid profiles were normal (total cholesterol: 105 mg/dL, low-density lipoprotein 60 mg/dL, high-density lipoprotein 15 mg/dL, triglyceride 79 mg/dL). We did not use any anticoagulant therapy.

On the second week of admission, the patient had no significant clinical improvement; however, distention of the peripheral veins of the affected ankle improved. The MRI was repeated, revealing signal increase in talus, navicular and cuneiform bones, which was consistent with osteomyelitis (figure 1). Vancomycin was added to the meropenem and amikacin treatment in case of *Staphylococcus aureus* infection. Surgical intervention was not considered. With the combined antibiotics treatment, the patient responded well and completely recovered on the fourth week of treatment.

TREATMENT

The patient received antibiotics for 4 weeks.

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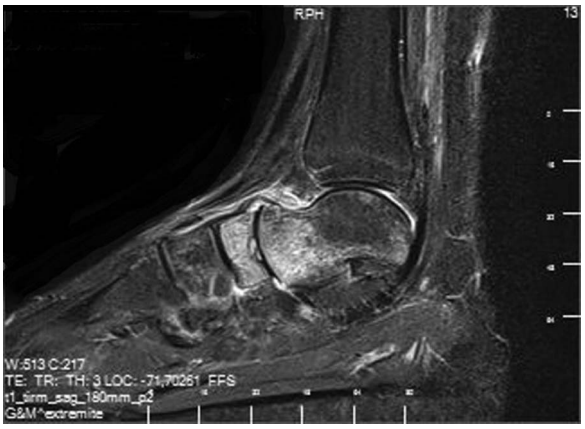

Figure 1 MRI findings of osteomyelitis.

OUTCOME AND FOLLOW-UP

The patient achieved complete recovery after antibiotics treatment.

DISCUSSION

Superficial thrombophlebitis is an inflammation of the vein, and commonly associated with thrombosis. The clinical findings are induration, erythaema, pain and tenderness along the course of the affected superficial vein. The estimated incidence of thrombophlebitis is between 3% and 11%. Although it is generally considered a self-limiting benign disorder, it can be complicated by deep vein thrombosis or pulmonary embolism.[6 7] Our patient had none of these complications. The predisposing conditions that can increase the risk of thrombosis are coagulation or fibrinolysis disorders, endothelial dysfunction, venous stasis, intravenous therapy, and drug usage and infection. Our patient did not have any family history of thrombosis, and the coagulation tests were normal. The most probable cause in our case appeared to be *Achromobacter*, which was isolated in the blood culture. Achromobacter species are uncommon opportunistic pathogens causing nosocomial infections, especially in patients with underlying malignancy and immunosuppression, and in premature infants. The most common cause of nosocomial cases is contaminated intravenous lines. Our patient was previously healthy and immunoglobulin levels and peripheric blood lymphocyte subsets were normal. Community-acquired infections have been reported, but to our knowledge this is the second case of thrombophlebitis caused by *Achromobacter*. The other case of septic thrombophlebitis due to a species of *Achromobacter*, published in 1960, involved a 4-year-old boy.[3]

This bacteria is present in normal human flora of the skin and can be isolated in water sources. Our patient had a history of purulent discharge from her ankle after prolonged wearing of tight shoes with sweaty and wet socks in rainy weather, which was further contaminated by home water supply containing the microorganism.

The combination of acute osteomyelitis and deep venous thrombophlebitis has been reported, but osteomyelitis complicating superficial thrombophlebitis is very rare in the literature.[4] Most of the cases were in adults and none were caused by *Achromobacter*.

In conclusion, we want to emphasise that this rare opportunistic pathogen can be a potential cause of infection even in previously healthy people. Also, osteomyelitis should be considered in cases of slow resolving thrombophlebitis.

Learning points

- Osteomyelitis can be considered in cases of unresolving thrombophlebitis.
- MRI can be repeated in cases of disease unresponsive to treatment.
- Even rare and opportunistic pathogens can cause infection in healthy people with a predisposing condition.

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