Staphylococcus lugdunensis Endocarditis

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Staphylococcus lugdunensis infective endocarditis (SLIE) is a rare disease with an aggressive and fulminant course carrying a high mortality rate. Systemic embolizations occur with a high complication rate. Surgery is almost always necessary. Though reports of successful SLIE cases with antibiotics are documented, complete eradication is difficult as possible embolization may have already occurred. We report herein a 44-year-old male whose SLIE was complicated with central nervous system and mesenteric artery embolization.

Key Words: Bowel ischemia • Infective endocarditis • Staphylococcus lugdunensis

INTRODUCTION

Staphylococcus lugdunensis (SL) was first described in 1988.1 Since then, sporadic cases have been reported totaling 77 over a 20-year period worldwide. As of this writing, this is the first case to be reported in Taiwan and is only the second case reported in Southeast Asia. The first case was reported in Singapore.2 To the best of our knowledge, this is also the first case to demonstrate in writing, distal embolization to the mesenteric arteries causing bowel ischemia.

CASE REPORT

A 44 year-old male was admitted to our hospital due to fever for 10 days. Past history showed hypertension, hepatitis B-related liver cirrhosis and uremia with regular hemodialysis for 7 years. He has a 20 pack years of smoking history but does not drink alcoholic beverages. Family history was unremarkable. 10 days prior to admission, the patient started having fever and chills that usually occurred in the evenings. This was accompanied by productive cough of yellowish sputum and sore throat. There was no dyspnea or abdominal pain. No medical consultation was done and no medications were taken. Symptoms occurred every day until the day of admission when the symptoms became intolerable. Physical examination revealed a conscious, coherent and ambulatory patient. Blood pressure was 114/44 mmHg, pulse rate was 118/min with a regular rhythm, respiratory rate was 18/min and temperature was 37.2°C. The neck was supple with no lymphadenopathy. The precordium was dynamic with a non-displaced or sustained apex beat. A 3/6 pansystolic murmur was heard over the apex and a 3/6 diastolic murmur was heard over both upper parasternal borders. There were fine crackles over the bibasilar lung fields.

The patient was treated as a case of pneumonia with levofloxacin 500 mg every after hemodialysis sessions. Laboratory data revealed the following: hemoglobin: 7.7 g/L, hematocrit: 24.2%, white blood cell count: 5,230/uL, (seg: 83%, lymph:7%), platelet count: 114,000/µL, serum glutamic oxaloacetic transaminase: 24 U/L, glutamic pyruvate transaminase: 12 U/L, blood urea nitrogen: 48.6 mg/dL, creatinine: 8.3 mg/dL, fasting glucose: 123 mg/dL, sodium: 133.3 meq/L, potassium: 3.02 meq/L, C-reactive protein: 25.14, prothrombin time: 12.6/11.3 (INR: 1.25), albumin: 2.3 g/dL, and cortisol level 8 am: 23.3 ug/dL, 4 pm: 27.1 ug/dL.
Electrocardiogram showed sinus tachycardia with non-specific ST changes, left atrial enlargement and left ventricular hypertrophy by voltage. Abdominal ultrasonography showed liver cirrhosis with ascites, splenomegaly, bilateral renal atrophy and bilateral pleural effusion. Cardiac ultrasonography showed vegetation over the noncoronary cusp (NCC) of the aortic valve (Figure 1A), moderate eccentric mitral regurgitation (MR), moderate pulmonary hypertension with pulmonary artery pressure (PAP) of 67 mmHg with mild tricuspid regurgitation (TR). Ejection fraction was 55%. Four sets of blood culture grew Staphylococcus lugdunensis that was sensitive to moxifloxacin, oxacillin, and penicillin. Levofloxacin was switched to oxacillin 2 gm every 4 hours with moxifloxacin 400 mg once a day.

Despite 2 weeks of antibiotics, fever persisted. Two sets of blood cultures drawn later still grew Staphylococcus lugdunensis. Antibiotics were shifted to vancomycin 1 gm every week and imipenem + cilastatin 500 mg every after hemodialysis session. Follow-up cardiac ultrasonography showed bilateral atrial dilatation, and dilated aortic root. The NCC vegetation was larger and shaggy echo was noted at both right and left coronary cusps. There was paravalvular leakage or abscess formation (Figures 1B-C) with severe eccentric AR. There was also shaggy echo at the mitral valve with severe MR and severe pulmonary hypertension with a PAP of 90 mmHg and moderate TR. Minimal pericardial effusion was also noted. Ejection fraction was 56%. Cardiovascular surgeon was consulted again and double valve replacement was performed 22 days after admission. St. Jude Medical 27MJ-501 and 21AHPJ-505 mechanical valves were placed over the mitral and aortic valve areas respectively. Blood cultures drawn 3 days post-surgery were negative.

Five days after surgery, the patient became drowsy with right hemiparesis. Brain computed tomographic scan showed post-contrast enhancement over the parieto-occipital area near the skull bone (Figures 2A-B) and ring-like enhancement over the left parietal area (Figure 2C). Cerebrospinal fluid routine and culture were unre-

Figure 1. (A) Short transthoracic echo view shows hyper-echogenicity over the non-coronary, right and left coronary cusps. (B) Parasternal long axis view shows the vegetation over the aortic valve area. (C) Parasternal long axis view shows a hypoechoic density over the aortic paravalvular area (arrow), consistent with abscess formation resulting in paravalvular leak.
Cardiac ultrasonography showed a mechanical aortic valve with non-critical aortic stenosis (estimated pressure gradient: 27 mmHg) and mild AR; and a mechanical mitral valve with a mitral valve area of 2.5 cm² and trivial MR. Histopathology showed myxoid degeneration, fibrosis and focal calcification of the aortic valve with numerous vegetations. The mitral valve also showed myxoid degeneration and fibrosis. Both valves showed large numbers of inflammatory exudates containing bacterial colonies.

On the 8th post-surgical day, the patient’s nasogastric tube drained dark-brown fecal-like material. Abdominal computed tomography showed ascites and air bubbles over the small bowel wall. No hepato-portal venous gas was noted. Possible distal embolization of septic emboli from the cardiac focus, or an aseptic thrombus embolization during the perioperative or postoperative period was entertained. Celiac angiography was planned but was deferred due to the patient’s unstable condition. Paracentesis performed later showed secondary bacterial peritonitis. Exploratory laparotomy was performed and resection of the jejunum 80 cm from the ligament of Trietz up to the entire ileum was performed due to gangrene change. The patient remained on ventilator support but 15 days after surgery, the patient died.

DISCUSSION

SL is a coagulase negative staphylococcus that produces ornithine decarboxylase and pyrrolidonyl arylamidases. It can bind vitronectin and fibrinogen to extracellular matrix proteins and can be misidentified as staphylococcus aureus due to production of clumping factor giving a positive slide coagulase or latex agglutination test. It is usually found on the skin particularly

Figure 2. (A) Pre-contrast brain computed tomographic scan showing a normal image. (B) Post-contrast scan over the same area showing enhancement over the bilateral parieto-occipital areas near the skull bone (black arrows). (C) Post-contrast brain computed tomographic image showing a ring-like enhancement over the left parietal area.
over the perineum and soft tissue. Sources of infection include dental abscesses, cutaneous infections, infected vascular lines, pacemaker leads and recent vasectomies. Systemic embolization with metastatic foci of infection also occur in 30-32% of cases. It has a high complication rate which include valve destruction with para-valvular abscess formation and high peripheral embolic rate. Overall mortality for SL infective endocarditis (IE) was 50% (42% for native valves and 78% for prosthetic valves). Surgery is necessary. Though there are reports of SL infective endocarditis (IE) cases being successfully treated conservatively with antibiotics, complete eradication is difficult as possible embolization may have already occurred earlier. Patients who underwent surgery had a lower mortality rate compared to those who did not [37% vs. 64%; p = 0.05].

In this era of antibiotic resistance, it is surprising to find a virulent microbe causing high mortality being susceptible to penicillin. Despite the minimum inhibitory concentration of penicillin being at least 2 dilutions lower than that of oxacillin or nafcillin, intravenous penicillin was still recommended as the drug of choice. However, despite penicillin susceptibility, SLIE is poorly responsive to antimicrobial therapy alone and the use of penicillin in a patient with septic shock seems somewhat inadequate. This patient suffered from probable embolizations to the brain and intestines on the 5th and 8th postoperative days respectively. However, blood cultures drawn on the 3rd postoperative day were negative. A possible explanation was that these embolizations occurred earlier or during the early course of the disease process when blood cultures were still positive. However, they were not yet severe enough to cause symptoms or organ damage. Surgery and removal of the infecting foci, together with the ongoing use of antibiotics, may have affected or influenced the negative blood culture results. Another possible cause may be due to an aseptic thrombus embolization to both areas during the perioperative or postoperative periods. However, we feel that an aseptic thrombus reaching the brain would manifest differently in the brain computed tomographic scan. This would appear as a hypodense lesion compatible with an acute cerebral infarction and not as a post-contrast enhancement. Regarding the mesenteric infarction, we concede that the cause of the patient’s bowel ischemia cannot be confirmed with absolute certainty that it was secondary to the staphylococcus ludunensis-embolization, since both septic, or an aseptic embolization occurring during the perioperative or postoperative periods are possible causes but may be difficult to prove. Prompt species identification should be performed for all coagulase-negative staphylococcal isolates for early and proper treatment. This specimen was initially overlooked as an ordinary pathogen and the patient was treated as an ordinary case of IE. It was only later that valvular replacement was done, but it may have been too late as brain and mesenteric embolization already occurred. Vascular access during hemodialysis was the most probable cause of infection, but the portal of entry is usually not identified in about 46% of cases.

Aside from endocarditis, SL can also cause peritonitis, osteomyelitis, prosthetic joint infection, brain abscess and catheter related sepsis. But there has been no mention of ischemic bowel disease. To the best of our knowledge, this is the first case to demonstrate SLIE with central nervous system and mesenteric embolization later resulting in bowel ischemia. Usually, no special attention was given to coagulase negative staphylococcus bacteremia at least in our hospital. Patients were usually given empirical antibiotics and later adjusted based on antibiotic susceptibility. Moreover, since this microorganism is usually susceptible to penicillin, one could easily dismiss it as easy to treat, and misses the diagnosis especially if a patient comes in early in the course of the disease with non-specific symptoms. Our patient’s outcome may have been different if we performed valvular replacement earlier, instead of treating him initially with antibiotics alone for 2 weeks.

Finally, this report stresses the value of prompt species identification, since treatment of SLIE differs with conventional IE secondary to Staphylococcus aureus and Streptococcus viridans. Most cases of the aforementioned microorganisms respond well to conventional antibiotics, while SLIE should be treated with immediate surgery even if there is minimal involvement of the valvular structures. This is due to its aggressive nature with a high incidence of distal embolization and difficulty to eradicate with antibiotics alone. Prompt valvular replacement certainly impacts treatment outcome when performed early in the course of the disease. Cardiologists should be aware that antibiotic trial is usually futile.
and valvular replacement is still the best treatment option.

CONCLUSION

In patients with coagulase-negative staphylococcal bacteremia with an audible cardiac murmur, prompt species identification, cardiac ultrasonography, broad spectrum antibiotics and immediate surgery may be life-saving especially if cultures are SL positive.

REFERENCES