

Case Report**Portal vein thrombosis as a complication of vancomycin resistant *Staphylococcus aureus* infection in an infant**Kathwate J¹, Shah I¹*Sri Lankan Journal of Infectious Diseases 2015 Vol.5 (1):28-31*DOI: <http://dx.doi.org/10.4038/sljid.v5i1.7613>**Abstract**

Pylephlebitis usually occurs as a result of an abdominal infection draining into the portal venous system. This infection is usually polymicrobial. Emergence of vancomycin resistant strains of *Staphylococcus aureus* (VRSA) has led to a dreadful scenario where the options available for treating serious infections due to these organisms are very seriously limited. We report a unique case of an infant who suffered from VRSA mandibular osteomyelitis and developed portal vein thrombosis (PVT) with a hepatic abscess which responded well to antibiotics and anticoagulation therapy.

Key words: Portal vein thrombosis, vancomycin resistant S. aureus, infant.

Introduction:

Both *S. aureus* and coagulase negative staphylococcal sp.(CoNS) have remained sensitive to glycopeptides except for a few case reports of vancomycin resistant strains of *S. aureus* (VRSA), vancomycin intermediate sensitive strains of *S. aureus* (VISA) and vancomycin resistant CoNS.¹ However, there are no previous reports of VRSA causing multiple abscesses in the neonatal period.

Case summary:

A one and half months old male infant presented to the casualty department in March 2013 with gasping respiration and required immediate mechanical ventilation in view of severe respiratory distress. He was born at term to a non-consanguineous couple by an emergency caesarean section due to non progression of labour. Birth weight was 4.1 kg. On day six of life, he developed fever with an abscess on the anterior chest wall, for which he received IV antibiotics for 10 days in the local hospital. Pus culture grew *Staphylococcus aureus*. As the parents removed the child from hospital against medical advice no further investigations were done. He was readmitted at age of 6 weeks to our hospital with respiratory distress as described. On

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examination at admission, he had tachycardia (heart rate 160/min) and tachypnoea (respiratory rate of 52/min), with oxygen saturation of 60% in room air. On systemic examination, there were bilateral crepitations and soft hepatomegaly. He was suspected to have sepsis. Investigations showed a hemoglobin of 7.3g/dl, leucocytosis (white cell count 49,700/mm³), platelet count of 570,000 /mm³ with raised C-reactive protein (63mg/dl). Liver function tests were normal. Blood cultures were sent before starting piperacillin-tazobactam and ofloxacin. He developed tender right cheek swelling on the second day of hospitalization and ultrasonography (USG) showed multiple abscesses in both parotid glands and the liver. USG of brain and echocardiography of heart were normal. MRI revealed right mandibular ramus osteomyelitis with same side masseter abscess. Mandibular abscess drainage was attempted but could not be done as the pus was very thick and loculated. Blood culture grew VRSA and antibiotics were changed to co-trimoxazole with rifampicin and clindamycin as per antibiotic susceptibility report (Table1). Vancomycin MIC ≤ 2 μ g/ml was considered susceptible, 4-8 μ g/ml as intermediate and ≥ 8 μ g/ml as resistant for *S. aureus*.

Table 1 : Antibiotic susceptibility of *S. aureus* isolates from blood culture

Antibiotics	MIC μ g/ml	Susceptibility
Penicillin	≥ 0.5	R
Clindamycin	2	S
Moxifloxacin	≥ 8	R
Oxacillin	≥ 4	R
Tetracycline	2	S
Ciprofloxacin	≥ 8	R
Erythromycin	≥ 8	R
Linezolid	2	S
Rifampin	≤ 0.5	S
Trimetho/sulfa	≤ 10	S
Cefazolin	≥ 2	R
Vancomycin* (Vitek2)	≥ 8	R

R-resistant, S-sensitive

the hepatic capsule, representing an abscess with thrombotic occlusion of the left portal vein. Low molecular weight (LMW) heparin was started.

He was investigated for suspected congenital immunodeficiency. Lymphocyte subset analysis (LSSA), nitroblue tetrazolium test (NBT) and flow cytometry for leucocyte adhesion test were normal. HIV antibody testing of his mother was non-reactive. He received antibiotics for six weeks and LMW heparin for 3 months. The baby recovered with treatment and follow up Doppler at the end of 3 months showed complete resolution of PVT.

Discussion:

Pylephlebitis is a condition with significant morbidity and mortality. Etiologies of pylephlebitis include diverticulitis, appendicitis, cholecystitis, pancreatitis, and intra-abdominal infections. Abdominal surgery can also predispose to pylephlebitis.^{2,3,4} Imaging techniques like Doppler ultrasound and contrast-enhanced CT facilitate early diagnosis. USG may show portal vein

thrombosis and signs of the primary abdominal inflammatory process, but its accuracy is limited by the interference of bowel gas and experience of an operator.⁵ Contrast-enhanced CT scan can display intraabdominal processes such as appendicitis and diverticulitis as well as mesenteric and portal vein thrombosis, liver abscesses, and bowel ischemia. In our patient it showed liver abscesses.

In a review of 100 cases by Kanellopoulou et al⁴, 81 patients were reported with acute pylephlebitis, while the remaining patients had chronic pylephlebitis. Cultures from blood or other tissues were positive in 77%. The infection was polymicrobial in half the patients and the most common isolates were *Bacteroides* sp., *Escherichia coli* and *Streptococcus* sp. Antibiotics were administered in all and anticoagulation in 35 cases. Patients who received anticoagulation had a favourable outcome compared to those who received antibiotics alone (complete recanalization 25.7% vs 14.8% : $p>0.05$, no recanalization 5.7% vs 22.2%: $p<0.05$, and death 5.7% vs 22.2%: $p<0.01$).

In a study performed by Nayman et al, they concluded that a primary source of infection could not be identified in 70% of patients.⁶ There appear to be no previous reports of VRSA isolated from patients with pylephlebitis. Vancomycin was considered to be the best alternative for the treatment of multi drug resistant methicillin resistant *S.aureus* (MRSA). However, there are increasing numbers of reports indicating the emergence of VRSA strains.¹ Infections with VRSA strains are treated with bacteriostatic drugs such as linezolid and clindamycin. Our patient was treated with co-trimoxazole, rifampicin and clindamycin. Treatment of pylephlebitis consists of treating the primary septic process by broad-spectrum antibiotics and appropriate surgical interventions^{2,3,4}. The use of anticoagulation has been controversial. It has been proposed that the purpose of anticoagulation in pylephlebitis is to prevent bowel ischemia and infarction secondary to extension of the thrombus. In the retrospective case series study done by Kanellopoulou et al, it was found that patients who received both antibiotics and heparin had a better outcome than those who received antibiotics alone.⁴ Our patient improved with both antibiotics and heparin. Though there has not been an established consensus on the duration of anticoagulation therapy in pylephlebitis, our patient received it for 3 months.

Conclusion: VRSA is an uncommon aetiological agent causing pylephlebitis, which responds to aggressive antibiotic therapy. The use of anticoagulants continues to be debated.

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