Case Report

**Purpura Fulminans due to Enterococcus faecalis**

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**Abstract**

We report a case of a 73 year old man who developed purpura fulminans due to *Enterococcus faecalis* sepsis. The patient was treated successfully with oral linezolid. Early diagnosis of the microbial etiology of purpura fulminans is important. If *Enterococcus faecalis* is found as the causal organism, appropriate antimicrobial therapy may be used.

**INTRODUCTION**

Purpura fulminans is characterized by thrombotic occlusion of skin arterioles leading to palpable purpuric lesions with irregular outlines, spreading ecchymosis, hemorrhagic blisters and distal symmetric gangrene, along with hypotension and fever.

Among the infective causes of purpura fulminans are *Neisseria meningitidis*, *Streptococcus pneumoniae*, *Streptococcus suis*, *Haemophilus influenzae*, *Capnocytophaga canimorsus*, *Staphylococcus aureus*, and *Plasmodia*.

We believe that this is the first report of purpura fulminans due to *Enterococcus faecalis* sepsis.

**CASE HISTORY**

SG, 73 year male, non-diabetic, non-smoker, non-alcohol user developed abdominal pain, loose motions and flatulence. Fifteen days later he noticed reddish minute dots on dorsum of hands and feet and presented to another hospital.

He was an epileptic taking valproate 200 three times daily for about 15 years. He had a history of cervical lymph node TB treated fully five years earlier. Laboratory investigations showed Hb 13.8 g/dl, WBC 26.6 x 10⁹ /L, PLT 227 x 10⁹ /L, creatinine, bilirubin, aminotransferases, bleeding time, clotting time, prothrombin time were normal, partial thromboplastin time was 44.5 sec with control 35 sec, d-dimer > 3000 ng/ml.

He was treated with cefpirome for 2 days, followed by oral cefuroxime for 8 days, oral levofloxacin for 5 days and oral artesunate for 7 days. After he was apparently better, he was discharged. He was readmitted 12 days later with an inability to pass urine and was catheterized. Four days later, he developed severe myalgia, arthralgia, marked increase in the rash mainly in the distal portions and large number of reddish black stools, fall in BP. He was treated with coamoxyclov 1.2 g thrice daily for 2 days followed by oral 625 mg thrice daily for 4 days, cefitzoxime 2g twice daily for 4 days and dexamethasone 4mg thrice daily for 4 days followed by oral prednisolone 20mg for 4 days.

During this time his WBC count peaked to 60.8 x 10⁹/L which later declined to 32 x 10⁹/L, the platelet count ranged between 71 x 10⁹/L to 91 x 10⁹/L, creatinine between 1 to 1.9 mg/dL. The ESR was 78 mm/h, Bilirubin and aminotransferases were normal.

He presented to us 4 days later, with fever, pulse110/min, respiratory rate 24/min, blood pressure 100/50mm Hg, he was slightly confused and was hard of hearing. He had pallor, edema, purpura, ecchymoses, distal symmetric gangrene, a few ulcerated spots on the trunk, paraphimosis and penile ulceration.

The laboratory tests revealed Hb 11.7 g/dL, WBC 27.6 x 10⁹/L, PI 98 x 10⁹/L, ESR 78 mm/hr, the peripheral smear was normal, no fragmented RBCs and no malarial parasites were seen. Serum creatinine was 2 mg/dL, electrolytes, anion gap, bilirubin and aminotransferases were normal.

Serum albumin was 1.8g/dL, globulin 3.5 8g/dL, ammonia 16 microgm/dL, LDH 637 u/L, CPK 274 u/L, random serum cortisol was 29.5 microgm/dL, prothrombin time 7 sec with control 8.8, INR 1.42, FDP >160 microgm/ ml and < 320 microgm/ ml.

Serum IgG was normal - 865 mg/dL, IgA was increased to 530 mg/dL, IgE was increased to 270.8 mg/dL, IgM was reduced to 83 mg/dL.

RA, ANA, ANCA were -ve, C3, C4 were normal, cryoglobulins were absent.

Urinalysis showed 10 WBC, 20 RBC per hpf, casts absent, protein 30mg/dL, 24 h protein excretion was 2.26 g.

Chest radiograph and abdominal USG were normal, 2D echocardiography was normal. Blood culture by Bactec resin was negative, buffy coat smear for organisms was negative, serum latex agglutination for *S. pneumoniae*, *N. meningitides*, *H. influenzae*, *C. canimorsus*, *S. aureus*, and *P. falciparum* were negative.

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H. influenzae was -ve.

Antibodies against HIV, HCV, Weil Felix and Paul Bunnel antigens, IgM TORCH, IgM anti-leptospira, IgM and IgG anti-dengue were -ve.

Aspiration of one of the blisters on the fingers was carried out. Microscopic examination of the blister fluid showed sheets of Gram positive cocci in pairs and short chains. Culture showed a non-motile, nonpigmented Gram positive coccus that was able to grow in 6.5% NaCl broth and showed growth on and blackening of bile-aesculin agar. It was tellurite resistant and positive for arginine, mannitol and sorbitol and negative for arabinose and raffinose. The organism was identified as Enterococcus faecalis. The identification was confirmed using the Gram positive ID kit for streptococci from Biomerieux - (Rapid ID 32 strep). It was susceptible in vitro to coamoxyclav, vancomycin, teicoplanin and linezolid. It was resistant to all other antimicrobials. The treatment of the patient had been started with piperacillin-tazobactam and oral linezolid 600 mg twice daily before the identification of the organism. Subsequent to culture identification, only linezolid was continued. There was a rapid improvement in the patient’s clinical condition. He was evaluated for anatomical and functional asplenia. Howell Jolly bodies were absent in the peripheral smear and the liver and spleen radionuclide scan was normal.

On the 12th day of treatment with linezolid, anemia, thrombocytopenia and neutropenia developed. Packed red cells were administered and the cytopenias improved in 10 days after withdrawal of linezolid. All the skin lesions of the patient healed and he was discharged after pneumococcal vaccine was administered.

**DISCUSSION**

Enterococci tend to produce infection in elderly, debilitated patients in whom mucosal or epithelial barriers have been disrupted or the normal flora is altered by prior antibiotic treatment. The infections commonly produced are urinary tract infections, intravascular catheter-related bacteremia and endocarditis. Enterococci are frequently cultured from polymicrobial infections like intraabdominal abscesses, biliary sepsis and diabetic foot ulcers but their exact role in these infections is debated. Skin and soft tissue infections are rare.

In this elderly patient, enterococcal sepsis may have followed a GI infection. Low serum IgM and albumin could have contributed to disseminated infection.

Enterococci are resistant to all cephalosporins despite some in vitro reports of susceptibility. They are not reliably killed by penicillin or ampicillin alone at concentrations achieved clinically in the blood or tissues. Hence a combination therapy of penicillin or ampicillin with an aminoglycoside is recommended for organisms that do not demonstrate high level aminoglycoside resistance (HLAR). Vancomycin may be substituted for penicillin in allergic patients. Linezolid is a useful alternative.

The organism in this case was resistant to penicillin due to beta lactamase production which was confirmed by using nitrocefin and was susceptible to coamoxyclav. Before the patient was referred to us he had not received any antimicrobial effective against enterococci with the exception of coamoxyclav for a short period, but it was not combined with an aminoglycoside and the patient had apparently not responded to it.

When streptococci grow to a large number e.g. $10^7$, they reach a stationary phase of growth, certain PBP are not expressed. Hence penicillin does not act so well in this situation due to the physiologic state of the organisms. We suspect that a similar problem occurs in enterococcal sepsis.

The pathogenesis of purpura fulminans is related to bacterial toxin production leading to DIC. In meningococcal sepsis monocytes express large amounts of tissue factor, and acquired deficiency of antithrombin and protein C and S can occur. Antifibrinolytic tendency is favoured by excessive PAI-1. Vascular thrombi occur leading to peripheral necrosis and gangrene. A similar pathogenesis of purpura fulminans is likely in enterococcal infection.

Under these circumstances, antimicrobials like clindamycin and linezolid which act independently of the stage of bacterial growth and inhibit protein and toxin synthesis, would be expected to have a greater clinical benefit.
This case is reported to highlight that Enterococcus faecalis is an additional cause of purpura fulminans. Early diagnosis of the microbial etiology of purpura fulminans is important. If Enterococcus faecalis is found as the causal organism, appropriate antimicrobial therapy may be used.

**REFERENCES**


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**Announcement**

The Department of Immunology, Sanjay Gandhi Post-graduate Institute of Medical Sciences, Lucknow will be conducting the 'Vth National Autoantibody Workshop' from September 13-18, 2004. In addition one day 'Postgraduate clinics in Rheumatology' will be held on September 12.

For information please contact: Dr. Amita Aggarwal, Department of Immunology, SGPGIMS, Lucknow 226 014. Fax: 0522-2668017; E_mail: amita@sgpgi.ac.in

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**Announcement**

**Training in Diabetes Foot Care**

**Project funded by the World Diabetes Foundation (WDF)**

*Academic Support by:*
- Consultative section on the diabetic foot- International Diabetes Federation (IDF)
- International Working Group on the Diabetic Foot (IWGDF)
- Diabetic Foot Society of India (DFSI)
- Muhimbili University and College of Health Sciences (MUCHS), Dar es Salaam, Tanzania

*Project Committee:*
- Sharad Pendsey, India; Karel Bakker, The Netherlands; Ali Foster, U.K.;
- Zulfiqarali Gulam-Abbas, Tanzania, Vijay Vishwanathan, India

*Excellent opportunity for practicing doctors, with a special interest in Diabetes!*

*Project at a glance:* 100 doctors with their paramedics (one doctor with one paramedical staff), to be trained in practical diabetic foot care management.

1. **Basic Course:** 2 days at four centers in India (Kolkata, New-Dehli, Mumbai & Chennai). Each center will have 25 doctors & 25 paramedics. The course is likely to be held between September/October 2004.
2. **Advanced Course:** 2 days (after 1 year) for the same participants is mandatory

*Faculty: Experts in the field of Diabetic Foot Care*

Selected participants will be provided with excellent educational material along with diagnostic/therapeutic instrument kits. Travel to nearest venue, lodging & boarding, access to training and resource materials are covered by a grant from WDF.

Certificate of participation on completion of the advanced course.

Preference to postgraduates, coming from private /public /corporate/govt. medical institutions.

Opportunity to start Preventive Diabetes Foot Care Clinic.

Selection committee’s decision will be binding on all applicants.

*The last date of receipt of application is 30th June 2004.*

Write for application form to

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“Shreeniwas”, Opp.Dhantoli Park, Nagpur 440 012 (India)