Guest Editorial
Novel HIV Prevention Strategies: The Case for Andhra Pradesh
JA Schneider

Review Article
Chikungunya Fever: A Re-emerging Viral Infection
M Chhabra, V Mittal, D Bhattacharya, UVS Rana, S Lal

Special Article
Fabrication and Evaluation of a Sequence-specific Oligonucleotide Miniarray for Molecular Genotyping
J Iqbal, F Hänel, A Ruryk, GV Limmon, A Tretiakov, M Dürst, HP Saluz

Original Articles
A Comparison of PCR Detection of Meca with Oxacillin Disk Susceptibility Testing in Different Media and Sceptor Automated System for both Staphylococcus aureus and Coagulase-negative Staphylococci Isolates
S Ercis, B Sancak, G Hasçelik

Effect of Exposure to Hydrogen Peroxide on the Virulence of Escherichia coli
A Hegde, GK Bhat, S Mallya

A Low Molecular Weight Es-20 Protein Released In Vivo and In Vitro with Diagnostic Potential in Lymph Node Tuberculosis
N Shende, V Upadhye, S Kumar, BC Harinath

Community-based Study on Seroprevalence of Herpes Simplex Virus Type 2 Infection in New Delhi
R Chawla, P Bhalla, K Bhalla, M Meghachandra Singh, S Garg

Changing Patterns of Vibrio cholerae in Sevagram Between 1990 and 2005
P Narang, DK Mendiratta, VS Deotale, R Narang

Rapid Serodiagnosis of Leptospirosis by Latex Agglutination Test and Flow-through Assay
TMA Senthilkumar, M Subathra, M Phil, P Ramadass, V Ramaswamy

High Level Ciprofloxacin Resistance in Salmonella enterica Isolated from Blood
R Raveendran, C Wattal, A Sharma, JK Oberoi, KJ Prasad, S Datta

Role of Enteric Fever in Ileal Perforations: An Overstated Problem in Tropics?
MR Capoor, D Nair, MS Chintamani, J Khanna, P Aggarwal, D Bhatnagar
Brief Communications

Evaluation of a Modified Double-disc Synergy Test for Detection of Extended Spectrum $\beta$-lactamases in Ampc $\beta$-lactamase-producing Proteus mirabilis
MKR Khan, SS Thukral, R Gaind
......58

Antimicrobial Susceptibility Profile of Neisseria gonorrhoeae at STI Clinic
C Shilpee, VG Ramachandran, S Das, SN Bhattacharya
......62

Detection of Extra-cellular Enzymes of Anaerobic Gram-negative Bacteria from Clinically Diseased and Healthy Sites
JM Nagmoti, CS Patil, MB Nagmoti, MB Mutnal
......65

Haemagglutination and Siderophage Production as the Urovirulence Markers of Uropathogenic Escherichia coli
MA Vagarali, SG Karadesai, CS Patil, SC Metgud, MB Mutnal
......68

The use of Dried Blood Spots on Filter Paper for the Diagnosis of HIV-1 in Infants Born to HIV Seropositive Women
S Mini Jacob, D Anitha, R Vishwanath, S Parameshwari, NM Samuel
......71

Evaluation of the Usefulness of Phage Amplification Technology in the Diagnosis of Patients with Paucibacillary Tuberculosis
D Biswas, A Deb, P Gupta, R Prasad, KS Negi
......75

Case Reports

Cytomegalovirus Oesophagitis in a Patient with Non-hodgkin’s Lymphoma
SS Hingmire, G Biswas, A Bakshi, S Desai, S Dighe, R Nair, S Gupta, PM Parikh
......79

Hydatid Cyst of Mediastinum
S Sehgal, B Mishra, A Thakur, V Dogra, PS Loomba, A Banerjee
......80

Ochrobactrum anthropi Septicaemia
U Arora, S Kaur, P Devi
......81

Intestinal Myiasis Caused by Muscina stabulans
S Shivekar, K Senthil, R Srinivasan, L Sureshbabu, P Chand, J Shanmugam, R Gopal
......83

Pyopericardium Due To Group D Streptococcus
K Karthikeyan, KR Rajesh, H Poornima, R Bharathidasan, KN Brahmadathan, R Indra Priyadarsini
......85

Pleural Effusion: A Rare Complication of Hepatitis A
A Bukulmez, R Koken, H Melek, O Dogru, F Ovail
......87

Correspondence

Prevalence of Inducible AmpC $\beta$-lactamase-Producing Pseudomonas aeruginosa in a Tertiary Care Hospital in Northern India
A Bhattacharjee, S Anupurba, A Gaur, MR Sen
......89

Parental History of Ulcer and the Prevalence of Helicobacter pylori Infection in their Offspring
KS Ahmed, AA Khan, JD Ahi, CM Habibullah
......90
Ciprofloxacin Breakpoints in Enteric Fever - Time to Revise our Susceptibility Criteria
C Rodrigues, N Jai Kumar, J Lalwani, A Mehta

West Nile Virus in the Blood Donors in UAE
M Alfaresi, A Elkoush

Estimation of Antibodies To HBsAg in Vaccinated Health Care Workers
TV Rao, IJ Suseela, KA Sathiavathy

Seroprevalence of Rubella Among Urban and Rural Bangladeshi Women Emphasises the Need for
Rubella Vaccination of Pre-pubertal Girls
A Nessa, MN Islam, S Tabassum, SU Munshi, M Ahmed, R Karim

Novel Digestion Patterns with Hepatitis B Virus Strains from the Indian Subcontinent Detected using
Restriction Fragment Length Polymorphism
P Vivekanandan, HDJ Daniel, S Raghuraman, D Daniel, RV Shaji, G Sridharan, G Chandy, P Abraham

Acute Urticaria Associated with Dicrocoelium dendriticum Infestation
A Sing, K Tybus, I Fackler

Book Reviews

Guidelines to Authors
proper diagnosis is essential to avoid unnecessary treatment. Intestinal myiasis can easily be prevented by protecting the foodstuff from contamination with the eggs or larvae of dipterous flies. Thorough washing of fruits and vegetables and adequate cooking of food before consumption offer better protection. The need for correct diagnosis of myiasis, which is potentially destructive, needs to be emphasized.

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References


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PYOPERICARDIUM DUE TO GROUP D STREPTOCOCCUS

Beta-hemolytic Enterococcus faecalis was isolated from the pericardial fluid obtained from a patient with pyopericardium. The patient was immunocompetent and had mild pleural effusion. He was treated with parenteral co-amoxiclav and amikacin, underwent pericardiectomy with repeated pericardial aspiration, and recovered completely. To our knowledge, this is the first report of pyopericardium due to E. faecalis.

Key words: Enterococcus faecalis, pleural effusion, pyopericardium

In the antibiotic era, purulent pericarditis, an infection associated with high mortality, is uncommon. Several bacterial agents have been reported to cause purulent pericarditis. These include Staphylococcus, Streptococcus, Haemophilus influenzae, Pseudomonas spp., Salmonella spp., Nocardia spp., coliforms and anaerobic bacteria. Bacterial pericarditis is usually characterized by purulent pericardial effusion (pyopericardium). Direct extension from pneumonia or empyema accounts for a majority of the cases. Haematogenous spread during bacteraemia and contiguous spread after thoracic surgery or trauma are also important mechanisms.[1] Pyopericardium can also result from the rupture of perivalvular abscesses into the pericardial space in patients with endocarditis. Rarely, pericardial invasion spreads along fascial planes from the oral cavity, particularly periodontal and peritonsillar abscesses. There is increasing evidence of anaerobic organisms being grown from pericardial fluid. The pericardium can become infected during meningococcal sepsis producing primary meningococcal pericarditis.[1-3]

To the best of our knowledge, the present report is the first case of pyopericardium due to E. faecalis.[4]

Case Report

A 60-year-old patient was admitted to the Cardiology Department, Vinayaka Mission Medical College Hospital, Salem, with complaints of breathlessness and mild retrosternal chest pain. On examination, the patient had engorged neck veins, muffled heart sound and presence of left basal crepts and rhonchi. Haemogram showed a total WBC count of 14,200 cells/cu mm with neutrophils 80%, lymphocytes 16% and eosinophils 4%. Haemoglobin was 11.2 gm/dL, and random blood sugar was 114 mg/dL. The other biochemical parameters including liver function tests and the serological tests like HIV and HBsAg were normal.

Echocardiography showed pyopericardium and pleural
effusion. CT scan of the chest showed exudative pericardial effusion with thickened pericardium and mild bilateral pleural effusion. He was taken up for pericardiectomy and drainage and had undergone repeated pericardial aspiration using pigtail catheter, which was left in situ.

The purulent pericardial fluid was processed microbiologically. Gram stain of the direct sample showed plenty of gram-positive cocci in pairs, short chains and polymorphonuclear leucocytes. The ZN stain was negative for acid fast bacilli. The fluid culture yielded heavy growth of \( \beta \)-haemolytic colonies on sheep blood agar (Figure) after 48 hours of incubation in candle jar and tiny lactose-fermenting colonies on MacConkey agar. The organism was catalase and oxidase negative, resistant to bacitracin, and failed to ferment mannitol. It was also positive to heat test and sodium chloride tolerance. The organism was tentatively identified as *Enterococcus* spp., which was resistant to penicillin but susceptible to co-amoxiclav, amikacin, linezolid and vancomycin. The strain was confirmed as *E. faecalis* at the Department of Microbiology, Christian Medical College, Vellore, by tests standardized in the laboratory.[5] Blood culture was found to be negative.

**Discussion**

*E. faecalis* is an organism of the normal commensal flora of the gastrointestinal and female genitourinary tracts. Endogenous strains gain access to the sterile sites either directly or by contaminated medical equipment, resulting in nosocomial spread and colonization with multidrug-resistant strains. Once colonized, the compromised patients are at risk of developing infections with resistant strains. Most infections are nosocomial in nature and include urinary tract infections, bacteremia, endocarditis, mixed infections of abdomen and pelvis, wounds and occasionally ocular infections.[6]

Life-threatening infections like pyopericardium are thought to occur due to complex interplay between the magnitude of virulence of the strain and the host response. *E. faecalis* has virulence factors like adhesins, cytolsins and other metabolic capabilities, which may allow the organism to proliferate extensively. In addition to the above mechanisms, the infection may also be due to the multidrug resistance of the organism.[6,7]

Pericarditis is usually secondary to the extension of an underlying condition such as infection from the pleural cavity.[8,9] For this patient, the infection would have spread from the pleural effusion that already existed.[10]

As this organism was resistant to penicillin, there was a possibility of developing resistance to other antibiotics also. Hence, a combination therapy (co-amoxiclav and amikacin) was administered to the patient. We have also administered anti-tuberculous drugs empirically although acid-fast staining was negative and the culture reports were awaited.

To the best of our knowledge, this is the first case of pyopericardium due to *E. faecalis*. Combined antibiotic therapy and daily drainage from pericardium after several days of pericardiectomy was done, which helped the recovery of the patient. The rarity of *E. faecalis* and its ability to develop antibiotic resistance makes the identification of the isolate and determination of its antibiotic susceptibility pattern essential for instituting proper therapy.

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**References**


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**Figure:** Colonies of *E. faecalis* grown on sheep blood agar showing beta haemolysis after 48 hours of incubation
Hepatitis A (HAV) infection, which is the most common form of hepatitis in the paediatric age group and which sometimes has a fulminant course, is endemic in Turkey, constituting one of the country’s important health problems. Pleural effusion also represents a rare benign complication of acute HAV infections. We describe here a case of Hepatitis A who developed pleural effusion.

Key words: Hepatitis A, pleural effusion

Hepatitis A (HAV) is a common illness, with prevalence rates highest in areas with limited hygiene and sanitation practices. In developing countries, where infection is endemic, most people are infected during the first decade of life. The illness is self-limited, and severity is age dependent. One of the rare extrahepatic complications of hepatitis A is pleural effusion. [1-4]. The pleural effusion is a rare and benign complication of hepatitis A (HAV), and its appearance doesn’t seem to correlate with seriousness of illness in children. To the best of our knowledge, only ten cases associated with HAV have been previously reported (2-8) We present a case of HAV complicated by pleural effusion.

Case Report

A 7-year-old girl was referred to the Department of Pediatrics of the Faculty of Medicine of Afyon Kocatepe University for abdominal distension, fever, vomiting and jaundice of the skin and sclera. Six days earlier, she complained of headaches, vomiting, anorexia, abdominal pain and mild fever. She had been previously well. On physical examination, the body temperature was 37.5 °C, heart rate was 88 /minute, respiratory rate 26/minute and moderate jaundice of the skin and sclera was present. The abdomen was mildly distended. The liver was palpable 3 cm below the costal margin in midclavicular line and tenderness was present. The spleen was not palpable. Breath sounds had decreased prominently at the base of right lung. All other physical examination findings were normal.

On admission, the laboratory studies showed WBC count 6550/µL, with 60% lymphocytes, haemoglobin 12.8 g/dL and platelets 199 000/µL. The blood urea nitrogen, electrolytes, glucose levels were normal. Aspartate aminotransferase was 120 U/L, alanine aminotranferase 434 U/L, gamma-glutamyl-transferase 115 U/L, total bilirubin 8.2 mg/dL and conjugated bilirubin 6.7 mg/L. Total protein was 7 g/dL and albumin was 3.3 g/dL. Prothrombin time was 14 seconds and activated partial prothrombin time 28 seconds. The results of urine analysis showed 2+ value of bilirubin, but no protein. Sediment was normal. Anti-HAV IgM antibodies and anti-hepatitis B surface antigen antibody were detected. HAV IgG, hepatitis B surface antigen, hepatitis B-e antigen, anti-hepatitis B-e antigen antibody, anti-hepatitis B core antigen antibody IgM, anti-hepatitis C virus, anti hepatitis E virus were negative. Serological analysis for cytomegalovirus and Epstein Barr virus were negative. Chest radiograph showed consolidation of the right lower lobe, and thorax ultrasound revealed a pleural effusion in the right chest (Fig. 1). Abdominal ultrasound revealed mild hepatomegaly with normal echogenicity and normal echotexture, right-sided pleural effusion. Thorax CT showed pleural effusion at the posterobasal segment of right lung (Fig. 2). Twelve days