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CASE REPORT

ISOLATION OF STREPTOCOCCUS PYOGENES FROM PLEURAL FLUID IN A CRITICALLY ILL PATIENT: A CASE REPORT WITH A RARE OCCURRENCE

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ABSTRACT

Transudative or exudative pleural effusion has a diverse etiology including bacteria that differ depending upon hospital or community acquired infections. Most bacterial organisms isolated from pleural fluid include Gram positive bacteria including Staphylococcus aureus, Streptococcus pneumoniae and Streptococcus viridans followed by Gram negative bacteria. The Isolation of Streptococcus pyogenes (Group A Lancefield Group) from pleural fluid is unusual as this bacterium is responsible for causing skin and throat infections which range from asymptomatic cases to full blown symptomatic toxic shock syndrome. Patients with various comorbidities may have the worst prognosis and severe complications from asymptomatic or confirmed infections.

We report a case study of a patient with isolation of Streptococcus pyogenes from pleural fluid, This is a rare finding, but may happen owing to dysbiosis associated with systemic insufficiencies. To our understanding this is the first reported case in Pakistan of Streptococcus pyogenes isolation from pleural fluid.

Keywords: *Antibiotic resistance, Dysbiosis, Emm gene, Pleural effusion, Pleural fluid, Streptococcus pyogenes*

INTRODUCTION

Transudative or exudative pleural effusion, the accumulation of fluid is one of the sequelae of many systemic deficiencies e.g., congestive cardiac failure, chronic renal impairment, chronic liver disease, and malignancy. The key for good prognosis in such patients requires timely antibiotic intervention. This require sound knowledge regarding microbiological isolation and identification from pleural fluid. Isolates may vary depending on hospital or community acquired infection.[1] A recent systemic review by European Respiratory Journal on most common bacterial isolates from pleural fluid reported *Staphylococcus aureus* to be the most common pathogen in pleural fluid globally followed by *Streptococcus pnemoniae* and *Streptococcus viridans* in community acquired infections. Beta hemolytic streptococci were reported as the sixth most common clinical isolate.[2] *Streptococcus pyogenes* is a part of the normal flora of skin and upper respiratory tract. This bacterium, that makes up the local microbiome belongs to Group A of Lancefield grouping among beta hemolytic streptococci and is responsible for various acute skin and soft tissue infections e.g., cellulitis, pharyngitis, impetigo, and other life-threatening sequelae including scarlet fever, glomerulonephritis, pneumonia and toxic shock syndrome owing to its invasive nature, various exotoxins, and multiple mechanisms of drug resistance.[3] There are about 220 different isolates of S. pyogenes based on difference in genetic sequence of its M protein and therefore epidemiologically vast outbreaks of this bacterial infection have been reported in Asia and United Kingdom.

Pathogenesis of streptococcal infection involves a wide range of mechanisms, evading innate and adaptive immunity of the host resulting in invasive infections especially more so in immunocompromised individuals. These mechanisms include attachment and invasion of epithelial barrier and soft tissues via surface M protein, streptolysin O and S, breakdown of complement C5a via C5a peptidase, prevention of virulence recognition by host immune system, decreased neutrophil chemotaxis owing to streptolysin

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S, presence of enzymes that degrade IgG, and superantigens causing cytokine storm.[4] The degree of severity of disease caused by Group A Streptococci (GAS) is directly proportional to immunosuppression and it has been reported that bacteremia caused by streptococci is likely to have worse prognosis in immunocompromised patients resulting in toxic shock syndrome and morbidity.[5] Toxic shock syndrome and invasive streptococcal A infection causing dissemination of bacteria to pleural fluid with symptomatic pneumonia and empyema warrants bacterial isolation from pleural fluid (which is normally a sterile space) for definite diagnosis in elderly or in patients having an underlying systemic deficiency like diabetes or cardiovascular insufficiency.[6] Invasive infections are associated with presence of bacteria in sterile spaces we present a case of a 59-year-old patient, who reported with dyspnea and generalized edema with associated comorbid of diabetes and congestive cardiac failure.

CASE REPORT

A 59-year-old patient with past history of diabetes and hypertension presented with aggravated symptoms of generalized body swelling, dyspnea, and orthopnea in emergency room of Fauji Foundation Hospital. She had a history of diabetes for 20 years, was hypertensive, and known case of congestive cardiac failure (CCF) and chronic kidney disease (CKD) in the last 1 year, with acute exacerbation of cardiac dysfunction. Severity of symptoms with ascites, eyelid swelling and pitting edema, had increased over the last one week. Associated complaints of fever, pallor, decreased alertness for the last 2 months were present, which had exacerbated for the last three days since admission. General physical examination revealed bilateral crepts and wheezing on chest auscultation, abdominal distention with shifting dullness, and bilateral pitting edema. The basic vitals had following picture; BP 100/70 mmHg, Pulse 115/min, SpO2 85%, respiratory rate 16/min, blood sugar level random 94mg/dl. Glasgow coma scale was recorded as E4 M6 V3 13/15. Chest X ray showed massive pleural effusion. The baseline laboratory results had following findings; complete blood picture showed hemoglobin count of 11.9 mg/dl (12-14mg/dl), white blood cell count 5.21 ×109/L (4-11 ×109/L), platelets 181 × 109/L (150-400 × 109/L), serum Urea 16.4 mg/dl (5-20mg/dl), creatinine 129 mg/dl (0.6-1.1mg/dl). Liver function tests showed bilirubin 9 (0.2-1.3mg/dl), ALT 23 U/L (4-36 U/L), ALP 157 U/L (44-147 IU/L), albumin 37g/L (34-54 g/L). Cardiac markers showed CK MB 30 (5-25 IU/L) and Troponin-T 0.17 (0 - 0.01 ng/mL). ECG showed T- wave inversion in leads 1 and 2 and aVR. Ejection fraction was reported at 20% (greater than 60%) on echocardiography, global hypokinesia, mild mitral regurgitation and severe left ventricular systolic dysfunction. D-dimers were less than 200. Lipid profile had following results; LDL 108mg/dL (100-129mg/dL), HDL 34.7mg/dL (35-80mg/dL) and serum triglycerides 79.2mg/dL (less than 150mg/dL). Prothrombin time (PT) was 13.5 seconds (11-13.5 seconds), activated partial thromboplastin time APTT was 31.9 seconds (30-40 seconds) and INR was recorded as 1.02 (less or equal to 1.1).

The patient was admitted to the hospital and conservative management was started with decreasing dyspnea with administration of oxygen, and nebulization with Ipratropium bromide 250mcg, Beclomethasone and Salbutamol 800/1600 mcg. Initially oral, prophylactic medicines for cardiac disease were continued and insulin was administered according to sliding scale with regular charting for blood sugar random (BSR). Basic lab tests were repeated and strict vital monitoring was advised. Injectable Ceftriaxone Sodium 2mg once daily was started as empirical antibiotic therapy. The condition of patient did not improve with renal function being deranged. High resolution computed tomography of the chest (HRCT) was advised and diagnostic and therapeutic pleural tap was done for routine examination, cytology, culture sensitivity on 3rd post admission day. The patient's condition continued worsening with not being able to maintain SpO2 at room air. The pleural fluid routine examination showed protein level of 112 mg/dl (15-45mg/dl), microscopy revealed the presence of

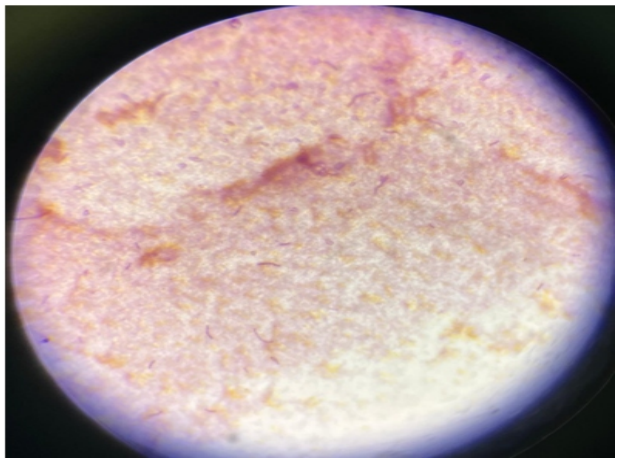

Figure I: Gram positive cocci arranged in chains as shown by arrow head.

Gram-positive cocci arranged in chains as shown in figure I.

The specimen was inoculated on Blood, MacConkey and Chocolate agar and was incubated at 37 C for 18-24 hours. After 18 hours the agar plates yielded β hemolytic growth on blood and chocolate agar. There was no growth on MacConkey agar plate. β hemolytic growth was Catalase negative. On further investigation, Lancefield grouping was done which showed agglutination with Group A anti sera. It confirmed the organism to be Group A Streptococcus (Streptococcus pyogenes). API -10 Strep (Analytical profile index--bioMerieux) was inoculated at 37 C for 18-24 hrs. It was further confirmed on API to be *Streptococcus pyogenes*. Sensitivity for *Streptococcus pyogenes* was checked on Blood Agar Plate incubated at 37 C for 18-24 hours which showed microorganism to be resistant to Ciprofloxacin (5µg), Ceftriaxone (30 µg). The bacterial isolate was sensitive to Bacitracin (10 µg), Doxycycline (30 µg), Linezolid (30 µg), Vancomycin (30 µg), Penicillin (10 µg) and Clindamycin (2 µg). The patient was continued with IV Ceftriaxone 2g along with supportive parenteral therapy, supplemental oxygen, along with strict monitoring of vitals but unfortunately owing to multiple comorbidities the patient expired third post admission day.

DISCUSSION

Streptococcus pyogenes, group A streptococci (GAS), a β hemolytic Gram-positive bacterium is one of the leading cause of invasive infections worldwide with yearly incidence of 6/100,000 cases. The high degree of virulence related to different serotypes owing to variance in emm gene corresponds to increased disease severity.[7] An epidemiological study on incidence, clinical, and microbiological features of streptococcal infections in Brussels and Hungary has reported that isolation of bacteria from pleural fluid is a rare findings (1% and 3% isolated from pleural fluid) in both probable and confirmed cases of invasive GAS corroborating an unusual finding similar to our case study where bacteria was isolated from pleural fluid.[7,8] Diabetes, cardiovascular disease, and renal insufficiency are one of many risk factors for streptococcal adherence, colonization and infection in adults as reported by a study in USA. All these comorbidities affirmative in our case study.[9]

Regarding antibiotic susceptibility of streptococci, this clinical isolate was sensitive to Penicillin and Chloramphenicol similar to a cohort analysis in Norway except that this study reports bacteria to be resistant to Tetracycline, Clindamycin, and Erythromycin differing from our antibiotic sensitivity tests.[10] This may reflect the evolutionary change of microorganism due to its diverse serotype distributed across different geographical location. The isolated bacteria in our hospital setup were susceptible to clindamycin but there has been reports of emerging increase in resistance to this antibiotic and macrolides in large studies in US which may be a cause for concern regarding antibiotic prescription and administration in streptococcal infections.[11] Analysis of antibiotic sensitivity to *Streptococcus pyogenes* in one study from Pakistan has also shown conferred resistance to this bacterium which differs from our report but is sensitive to Ceftriaxone which was prescribed in this patient although sensitivity tests indicated otherwise.[12] Such variation in sensitivity to one antibiotic has been documented in Egypt where the isolates from one hospital is 100% resistant to Ceftriaxone whereas another study showed 50% resistance to this antibiotic.[13] Critically ill patients with multiple risk factors are likely to have a dysbiosis owing to interplay of lung and oral microflora within the body, its interaction with outside environment including shifting between different nursing setups, invasive treatment measures such as intubation, face masks and therefore ability of microflora to cause disease escalation similar to our case study where unusual isolation of *Streptococcus pyogenes* from pleural fluid was a rare finding therefore it is necessary to take precautionary and prophylactic measures in such medical scenarios where appropriate and timely antibiotic regimes could save lives.[14] Moreover, emerging resistance trends of β hemolytic streptococci requires a collaborative approach between critical care providers, microbiologists, infection control specialists and diagnostic labs to implement antibiotic stewardship policy as required according to local antibiogram which will help curb rising infection outbreaks and will contribute to benefit population in the long run.[15]

Authors' Contribution

Saima Ishtiaq: Conception of study / Designing / Planning, Experimentation / Study Conduction, Analysis / Interpretation / Discussion, Manuscript Writing

Amna Waheed: Analysis / Interpretation / Discussion, Manuscript Writing

Muhammad Moaaz Ali: Facilitated for Reagents / Material Analysis

Uzma Zahid: Facilitated for Reagents / Material Analysis

Hareem Fatima Niazi: Facilitated for Reagents / Material Analysis

Shahid Ahmad Abbasi: Conception of study / Designing / Planning, Critical Review

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