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Tuberculosis Associated Septic Shock

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Abstract
Diagnosing and identifying the underlying disease of septic shock is so difficult, cases of tuberculosis-associated septic shock are extremely rare. A 55-year-old Woman with weakness, nausea, low intake, fiveepisodes of diarrhea, three weeks of coughing, and a positive rapid molecular test result for tuberculosis.The patient had a history of AIDS and was receiving ARV treatment. There had also been a lump in theright neck for a month. A fine needle aspiration biopsy (FNAB) was performed, and the results showed afocus of atypical cells with a background of suppurative chronic inflammation. A physical examinationrevealed a fever (38^0C), blood pressure of 78/46 mmHg, and a pulse rate of 110 beats per minute. WBC:2.60, HB: 7.5, HCT: 23.3, Plt: 353, SGOT/SGPT: 68/8, GDA: 142, BUN/SC: 108/2.6, Na: 141, K: 2.7, Cl:107 were the results of the laboratory tests. The thorax x-ray revealed a pattern of miliary tuberculosis with adifferential diagnosis of the metastatic.

Keywords: Tuberculosis, Miliary Tuberculosis, Septic Shock, Metastatic, Sepsis

1. Introduction

The body dysregulated reaction to an infection is known as sepsis. Sepsis continues to be the world's leading cause of death. Sepsis was linked to 48.9 million cases, 11 million deaths, and about 20% of all deaths worldwide[17]. Sepsis is most frequently reported to be caused by bacterial infections, with respiratory infections accounting for the largest percentage of cases[6, 9] . The leading cause of death globally is still tuberculosis (TB), with an estimated 2.64 million cases and 1.5 million fatalities annually[5], 3.4% of TB patients need critical care.

2. Case Illustration

A 55-year-old Woman with weakness, nausea, vomiting, shortness of breath and weight loss over the past two months. She had also noted three weeks of productive cough, hemoptysis, night sweat. The patient had a history of AIDS and was receiving ARV treatment. There had also been a lump in the right neck for a month. A fine needle aspiration biopsy (FNAB) was performed, and the results showed a focus of atypical cells with a background of suppurative chronic inflammation. Anemia (Hb: 7.5 g/dL), decreased kidney function (Ketone: 108 g/dL and Creatinine: 2.6 g/dL, Glomerular Filtration Rate: 23 mL/min/1.73 m2), blood sugar (142), SGPT/SGOT (8/68), sodium (141), potassium (2.7), chloride (107), leukocytes (11.60 x 10^3/uL), hemoglobin (7.5 g/dL), and thrombocytes (261 x 10^3/dL), Albumin (2 g/dL), were among the laboratory results (table 1).

In the emergency department, She was tachycardia 130bpm, hyoptensive 80/50 mmHg, tachypneu 38 breaths per minute, and hypoxic 82% on room air requiring a Non-rebreathing face mask. An ECG showed sinus tachycardia, haemoglobin of 7.5 g/dL. A chest X-ray was significant for miliary tuberculosis (Figure 1).

Table 1. Laboratory Result

Examinations	Result
Complete Blood Count	
Leucocytes	11,60 x 10^3 u/l
Hemoglobin	7.5 g/dL
Hematocrit	30.0%
Platelets	261 x 10^3 dL
Diff Count	
Neutrophils	73.9%
Lymphocytes	19.2%
Monocytes	6.9%
Mean Corpuscular Volume	87.9
Clinical Chemistry	
Ureum	108mg/dL
Creatinine Serum	2.6 mg/dL
SGOT	68 U/L
SGPT	8U/L
Electroliyte	
Sodium	141 mmol/L
Potassium	2.7 mmol/L
Chloride	107 mmol/L
Albumin	2 g/dL

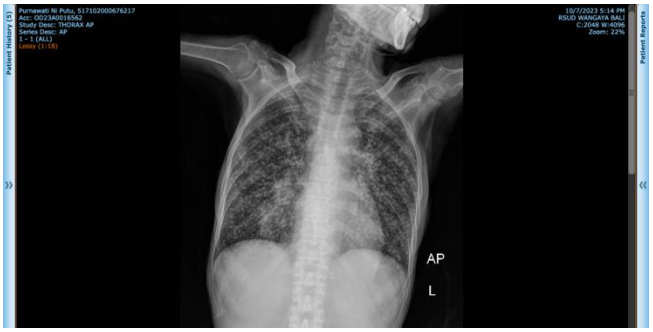

Figure 1: Chest X-ray showed of miliary tuberculosis with metastatic disease being the differential diagnosis.

3. Discussion

A type of extra-pulmonary tuberculosis known as "miliary tuberculosis" is caused by the hematogenous spread of the Mycobacterium tuberculosis germ. HIV infection is a risk factor for extra-pulmonary tuberculosis (TB); extra-pulmonary TB typically follows HIV infection[24]. Adults are more likely than children to experience TB with septic shock[10]. Immunocompromised cells from tuberculosis can lead to SIRS (Systemic Inflammatory Response Syndrome), multi-organ failure, and sepsis or septic shock[8]. TB contineu to spread both lymphatically and hematogenously causing a systemic response. Proinflammatory mediators promote leukocytosis and stimulate the release of other cytokines, all of which act as pyrogens, active WBCs and macrophages, promote chemotaxis, cause immunosuppression, and stimulate both coagulation and fibrinolytic activation[3]. Arachidonic acid and adhesion molecules further cause vascular permeability and migration of aforementioned leukocytes. there is a link between tumor necrosis factor-alpha (TNF-alpha) and activation of the complement pathway, which further enhances neutrophil trafficking and inflamation[11]. This leads to tissue ischemia, cytopathic injury, and increased programmed cell death, eventually resulting in widespread multi-organ damage[4]. The rearrangement and distribution of intravascular fluid, inhibition of vasopressin release, and upgregulation of vasoactive mediators such as nitric oxide result in hypotension or shock. At the level of the lung, endothelial damage in the respiratory vasculature disrupts blood flow and increasesmicrovascular penetrability, leading to interstitial and alveolar pulmonary edema. The odematous fluid protein to plasma protein ratio is nearly 0.95, which is significantly higher than those with cardiogenic pulmonary edema. This proteinaceous fluid destroys pneumocytes which increase surface tension, traps leukocytes, and further damage lung vasculature. Diffuse alveolar damage (DAD) ensues, a significant ventilation/perfusion mismatch occurs, and ARDS develops. when widespread TB-related lung damage occurs, ARDS is hardly reversible, especially when complicated by multi-organ failure.

Conclusion

In areas where tuberculosis is endemic, in particular, it is still impossible to connect the illness with septic shock. The primary therapeutic management for tuberculosis with septic shock still involves timely diagnosis, fluid resuscitation, administration of inotropes, and appropriate administration of antibiotics, just like it does for any other septic shock. A high degree of suspicion for septic shock and prompt management can save many lives. Physicians should be especially aware of tuberculosis that presents as septic shock in endemic areas. To maximize clinical care, multicentric observational studies are required to determine the prevalence and risk factors for tuberculosis septic shock.

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