Tuberculous Miliary Presenting with Fulminant Myocarditis

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Summary:

Tuberculosis miliaria is a disseminated form of infection by Mycobacterium tuberculosis, most often via the hematogenous or lymphatic route. Depending on its degree of extension, it can lead to severe organ failure, which may explain its high mortality rate. Cardiac involvement during tuberculosis miliaria worsens the prognosis of these patients. This localization remains rare, and most often results in pericarditis with pericardial effusion, while fulminant myocarditis is extremely rare. Anti-bacillary treatment is a therapeutic emergency. Cardiocirculatory resuscitation of the associated hemodynamic instability is similar to that of any state of cardiogenic shock, and calls for vasoactive and positive inotropic amines after adequate filling. The role of corticosteroid therapy is still under discussion. Prognosis depends on the degree of organ failure, the presence of severe malnutrition, tolerance of antibacillary treatment and the existence of resistant or non-resistant strains of Mycobacterium tuberculosis.

Introduction:

Tuberculosis is a common disease, and a major cause of mortality worldwide. Pulmonary localization remains by far the most common, but extra-pulmonary localization is not uncommon. They often reflect underlying immunodepression. We report here on a case of tuberculous miliaria with pulmonary, neuromeningeal, cardiac, peritoneal and spinal cord involvement. We will focus on isolated myocardial involvement without pericardial effusion in the form of fulminant myocarditis, which remains extremely rare and has been described mainly in post-mortem autopsy series. Its prognosis remains grim, given the severity of the localization, the importance of this noble organ and the organ failure that follows its dysfunction.

Observation:

Mrs. E. K., 27 years old, presented to the emergency department with atypical diffuse abdominal pain associated with diffuse abdominal distension with alternating diarrhea and constipation, all evolving in a context of altered general condition due to significant weight loss. General examination revealed a conscious patient 15/15 BP= 10/5 HR= 111 SpO2= 94% on fresh air, respiratory rate 23 cycles per minute, apyretic at 37°C. Abdominal examination revealed copious ascites. Pleuropulmonary examination revealed crepitating rales at the apexes, with bilateral fluid effusion at the bases. The rest of the clinical examination was unremarkable. Biological workup was disturbed, with hemoglobin 8.9, white blood cell count 10,358, predominantly lymphocytes, platelets 189,000, hypokalemia 3.3, CRP 45, albumin 25, renal and hepatic function normal. Correct haemostasis. GeneXpert screening of ascites puncture fluid confirms tuberculosis infection of ascites fluid. Chest X-ray shows almost innumerable tiny interstitial nodules distributed in both lungs. BK sputum positive. The patient was admitted to the Pneumology-Physiology Department, where anti-bacillary treatment was started. On day 2 of hospitalization, she presented with a generalized tonic-clonic convulsive seizure and post-critical coma. The patient was transferred to the intensive care unit on August 20. On admission, the patient was unconscious Glasgow 8/15 BP= 70/33 mmHg HR = 140 SpO2= 74% on free air T= 36.8 Capillary blood glucose 1.2g/l. Cold extremities. The patient was intubated, ventilated, sedated and stabilized on 2 mg/h of norepinephrine. Cerebral CT: normal, thoracic CT: appearance of tuberculous miliary. Lumbar puncture: hyperproteinorachia, hypoglycorachia with a ratio of less than 0.5 and predominantly lymphocytic cellularity. Trans-thoracic echocardiography showed dilated cardiomyopathy, LVEF 25-35%, dilated right ventricle, IVC 20, no pericardial effusion. Biological workup on day 4 of hospitalization in favor of bone marrow aplasia with inflammatory myelogram.

Discussion:

Tuberculosis is not an exceptional pathology in the ICU. Acute respiratory failure is the main reason for admission. Tuberculosis miliaria is a disseminated form of tuberculosis resulting from hematogenous dissemination of Mycobacterium tuberculosis[3]. It

occurs most frequently in immunocompromised patients, usually due to HIV infection, cancer or severe malnutrition [1]. We report here a case of tuberculous miliaria with multifocal involvement: pulmonary, neuro-meningeal, cardiac and medullary. We will discuss the cardiac involvement, as it is the rarest and most serious, and classically gives rise more to pericarditis with pericardial effusion than to isolated myocarditis with hemodynamic instability. In fulminant myocarditis, cardiac pump dysfunction results from invasion of the myocardium by Mycobacterium tuberculosis, which can lead to ventricular arrhythmias, prolonged QT, atrioventricular block, coronary artery damage and sometimes sudden death. The clinical picture is that of cardiogenic shock in the context of tuberculosis infection. However, the clinical signs of cardiac involvement by tuberculosis may be latent, which is why systematic screening for the presence of tuberculosis is discussed, especially in view of the seriousness and life-threatening complications of such localization. Another explanation for cardiac failure in tuberculosis miliaria is the systemic inflammatory response syndrome (SIRS), which can be triggered either directly by Mycobacterium tuberculosis or by bacterial co-infection, since tuberculosis occurs in an immunosuppressed environment. Resuscitation measures (invasive devices, mechanical ventilation) are also factors favoring super/co-infection, which can progress to septic shock with septic cardiomyopathy through mitochondrial dysfunction due to the secretion of inflammatory cytokines such as interleukin 6, TNF [2] [5]. Treatment is based on supplementation and optimization of hemodynamics by introduction of catecholamines; dobutamine, noradrenaline after restoration of euvolemia. It is also clear that anti-bacillary treatment must be started as a matter of urgency. It must be discussed as a matter of urgency, without waiting for microbiological confirmation, because the speed with which it is instituted has a significant impact on the patient's functional and vital prognosis. The usual anti-tuberculosis treatment for a patient infected with a susceptible strain of M. tuberculosis is based on dual therapy combining rifampicin and isoniazid for six months, in combination with pyrazinamide and ethambutol for the first two months of treatment. The total duration of treatment with rifampin and isoniazid should be extended to 9-12 months in the case of extra-pulmonary localization. The hepatotoxicity of rifampicin, isoniazid and pyrazinamide can also complicate the management of such patients, with elevated transaminases being quite common. The impact of corticosteroid therapy on improving survival in tuberculosis patients with meningeal and pericardial localization, but has never been clearly demonstrated in patients with pure myocardial involvement, This is due firstly to the rarity of this form of tuberculosis, and secondly to the lack of any specific means of monitoring the effects of corticosteroid therapy on myocardium invaded by Mycobacterium tuberculosis. However, it seems logical that corticosteroid therapy, through its anti-inflammatory effect, can have beneficial effects on myocardium in a state of inflammation. Differentiation between fulminant myocarditis and septic cardiomyopathy can be difficult on the basis of clinical findings, electrocardiogram and trans-thoracic echocardiography; the only definitive test is endomyocardial biopsy.

Conclusion:

Tuberculosis is not an infectious pathology with which the intensive care physician often comes into contact. However, the severity of certain pulmonary, neuromeningeal and cardiac localizations makes it a cause for admission to the intensive care unit. We report here a case of fulminant myocarditis in the setting of tuberculous miliaria, with pseudo-refractory cardiogenic shock. All cases of extra-pulmonary tuberculosis, and in particular HIV infection, should be investigated for immunodepression. Endomyocardial biopsy remains the gold standard for confirming tuberculous myocarditis. Mortality remains high, especially with the emergence of new multi-resistant or even ultra-resistant strains, which poses a real problem for therapeutic management.

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