Case Report

Postmortem Findings in a 10-Year Old Girl Dying from Yellow Fever: Clinical Autopsy as a First Point of Disease Surveillance

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ABSTRACT

Yellow fever is an emergency notifiable disease of public health significance with epidemics seen in many tropical countries. We report a case of yellow fever infection in a 10-year-old girl diagnosed at post-mortem examination. She presented with a 10-day history of high-grade fever, jaundice and abdominal pains, and died after a month of hospitalization. Post-mortem examination revealed massive hepatomegaly with histologic sections showing extensive mid-zonal necrosis, microvesicular steatosis and Councilman bodies. The findings emphasize the relevance of autopsy in clinical diagnosis; medical education and audit; and the need to institute postmortem examination as a point of disease surveillance.

Key words: Yellow fever, post-mortem, councilman bodies, mid-zonal necrosis, disease surveillance

INTRODUCTION

Communicable diseases constitute enormous public health problems in many tropical countries. Worldwide outbreaks of communicable diseases have increased in recent times for a number of reasons [1]. Yellow fever is one of the vaccine-preventable emergency notifiable diseases covered by International Health Regulations[2]. In recent years, epidemics of yellow fever have been reported in various locations, including in Nigeria. Yellow fever is one of the viral hemorrhagic fevers, caused by a *Flavivirus*, one of the ecologic groupings of viruses transmitted by different genera of mosquitoes[3].

The recent pattern of yellow fever epidemics in Africa shows that children have been predominantly infected[4]. Severe cases are characterized by hepatic and renal dysfunction, hemorrhagic tendencies and a high mortality. The lesions of yellow fever are due to localization and propagation of the virus in particular organs. Diagnosis is by isolation of the virus, serology, or histopathology [5]. There is no specific antiviral therapy. Vaccination is however, a most effective preventive measure.

We present a case of yellow fever infection in a 10-year-old school girl, which was diagnosed at post-mortem examination. This case was reported before any other case in the recent local outbreak. We hope to make the argument that, as a focal point for disease surveillance, the autopsy pathology practice is a critical one. We observed universal precaution at every stage of the post-mortem examination. A report was immediately filed with the local authorities.

CASE REPORT

A 10-year-old schoolgirl was admitted with a 10-day history of fever, abdominal pain and yellowness of the eyes. A diagnosis of suspected typhoid perforation was made and she had an exploratory laparotomy which showed no bowel perforation or any other specific abnormal intraoperative finding, except for dark bilious fluid in the peritoneal cavity.

She was treated with antibiotics and other supportive measures . However , there was no clinical improvement and the fever continued with derangement of hepatic and renal functions as evidenced by rising liver enzymes, deranged clotting profile and elevation of serum urea and creatinine. She had an episode of convulsions few hours before she died, a month after admission.

At post-mortem , she looked older than her age, the body weighed 36 kg and measured 145 cm in length. She was dressed in her personal clothing with a bandage-covered 13 cm long healing surgical scar on the right hypochondrial region . She was deeply jaundiced as demonstrated by yellowness of the sclera and the internal organs including the pachy - meninges. No other significant findings were noted on the body externally.

There was an excess of dark bilious fluid in all the serous cavities. The lungs, heart, liver, spleen and kidneys were all enlarged for her age. The markedly enlarged liver had fibrinous adhesion bands on its peritoneal surface. Cut sections showed mild fatty changes . The remarkable histologic findings were in the liver which showed some thickening of the capsule with preservation of reticular architecture , few surviving hepatocytes around the portal tracts and the central vein with extensive mid-zonal necrosis , numerous scattered Councilman bodies, microvesicular fatty changes and extensive bile stasis as shown in Figure 1 a-c

The gall bladder contained free -flowing dark bile. The enlarged spleen had diffluent cut-surfaces and sections showed atrophic follicles with splenic congestions whereas, the sampled generalized lymph nodes essentially showed reactive hyperplasia . The kidneys showed blurring of the cortico - medullary differentiation with unremarkable pelvi -calyceal systems and the ureters . The urinary bladder contained 20 mls of purulent material , and had punctate mucosal hemorrhages . Sections from the kidneys (Figure 1d) showed capsular thickening and widespread extensive acute tubular necrosis and sprinkles of lymphocytic infiltration . The urinary bladder showed ulcerated urothelium with submucosal fibrosis. Culture of the purulent material in the urinary bladder yielded *Staphylococcus aureus*.

The heart weighed 200g. The valves, chordae tendinae and papillary muscles , coronary vessels and coronary sinus were unremarkable . The upper respiratory tract was unremarkable ; the lungs were dark and subcrepitant , sections revealed dark solid appearances with oozed dark frothy fluid in about 80% of the lung substances. Similar fluid was seen oozing out of trachea and main bronchi . Sections of the lungs showed pleural thickening , features of interstitial pneumonia , hemorrhage , congestion and alveolar edema.

The skull and scalp were unremarkable . The brain was moderately edematous with marked surface congestion. There were bilateral subarachnoid hemorrhages at the convexities of the cerebral hemispheres . The cerebral hemispheres , cerebellum and brainstem showed good grey-white matter differentiation . Sections of the brain showed subarachnoid hemorrhage , subarachnoid and intraparenchymal congestion,intravascularmicrothrombi and hyperplasia of the ependymal cells lining the ventricles (Figure 1e).

On the basis of the final anatomic summary , the cause of death was ascribed to respiratory failure secondary to pulmonary edema with multiple organ failure on a background of septicemia with severe yellow fever infection.

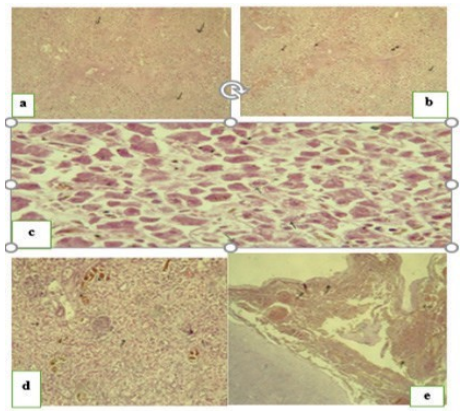

Fig. 1. (a) Photomicrograph of the liver (H&E x40 magnification). Section of the liver showing preservation of some residual hepatocytes around portal triad with mid-zonal necrosis [arrows] (b). Liver (H&E stain x40 magnification). Section of liver showing few surviving hepatocytes around portal triad (double stars) and central vein (single star). Note the mid-zonal necrosis [arrows]. (c). Liver (H&E stain) x40 magnification). Section of the liver showing microvacuolar microvesicular fatty changes (long arrows) and Councilman bodies. (d). Photomicrograph of the kidney (H&E stain x100 magnification). Section of the kidney showing acute tubular necrosis (arrows) and tubular cast [Stars]. (e). Photomicrograph of the brain (H& E stain x 40 magnification). Section of the brain showing subarachnoid haemorrhage [arrows]

DISCUSSIONS

The clinical course and natural history of yellow fever infection is variable ; ranging from asymptomatic infection to fatal disease accompanied by high-grade fever , jaundice , impaired hepatic functions with deranged liver enzymes . Other features include abnormal clotting profile with bleeding tendencies , acute tubular necrosis and complications such as pneumonia and suppurative parotitis [6]. Confusion , seizures and coma before death are known to characterize the late stages of this illness[7].

Post-mortem findings in fatal yellow fever viral infections are rather uncommon , perhaps due to paucity of post-mortem examination conducted in epidemic situations as it is highly infectious.

Diagnosis of yellow fever infection is made in the laboratory either by isolation of the virus early in the disease , detection of specific antibodies to the virus or by demonstration of characteristic histologic findings in liver biopsy or post-mortem specimens [8,9]. Histologic sections of the liver showed preservation of the reticular framework with few surviving hepatocytes around the central veins and peripherally around the portal triads with extensive midzonal necrosis , microvesicular steatosis and Councilman bodies . The observed histologic triads were characteristic and adequate for diagnosis of yellow fever infection [10,11].

There was no significant inflammation . The extent of the hepatic damage also explained the elevated liver enzymes - AST , ALT ; hyperbilirubinaemia and the abnormal clotting profile[12].

The proteinuria and azotaemia were consistent with extensive acute tubular injury seen histologically . Other important findings were interstitial pneumonia , extensive pulmonary edema and congestion. Though there was no bleeding externally , subarachnoid hemorrhage was seen at the postmortem examination , Bleeding tendencies are a common presentation of severe yellow fever. Other findings in the brain were cerebral and cerebellar congestion with microthrombi formation ; in keeping too with the postmortem diagnosis . Also , in agreement with the findings at autopsy , and the eventual diagnosis , secondary bacterial infection resulting in pneumonia or sepsis is a recognized complication of yellow fever infection[7].

In conclusion , the findings in this case underscore the need for post-mortem examination in routine clinical practice , not only in establishing the cause of death or the advancement of the frontiers of medical knowledge , in confirming, or indeed denying, clinical diagnosis ; and clinical audit ; and the role of laboratory medicine in general and autopsy pathology in particular, in disease surveillance.

DECLARATIONS

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