

Benign Cholestatic Jaundice after Surgical Treatment of Pyonephrosis: A Rare Presentation

Ahmed M. Moeen^{1*}, Seham M. Moeen², Sawsan M. Moeen³, Ahmad F. Thabet³ and Dina A. Mohareb⁴

¹Asyut urology and nephrology hospital, Assiut University, Assiut, Egypt

²Department of anesthesiology and intensive care, Assiut University, Assiut, Egypt

³Department of Internal Medicine and Clinical Hematology, Assiut University, Assiut, Egypt

⁴Department of clinical pathology, Assiut University, Assiut, Egypt

*Corresponding author: Ahmed Mohamed Moeen, MD, Lecturer of Urology, Asyut Urology and Nephrology Hospital, Asyut University, Assiut, Egypt, Tel: +20 01003960931; Fax: 00 088 2333327; Email: moeen3@yahoo.com

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Abstract

Postoperative cholestatic jaundice may occur immediately after surgical treatment of pyonephrosis, but it is a rare condition. Convalescence is the role with no specific treatment. Herein, we report the occurrence of postoperative cholestatic jaundice in a thirty years old female patient after subcapsular nephrectomy for pyonephrosis.

Keywords: Liver Function; Nephrectomy; Pyonephrosis; Sepsis; Subcapsular

Introduction

Association of renal and hepatic dysfunction may occur as a result of primary diseases or systemic disorders involving both organs, primary hepatic disease affecting the renal function and primary renal disease affecting the hepatic function. Abnormal liver function tests (LFTs) may occur postoperatively in patients who already have hepatic pathology, due to certain anesthetic medications or due to postoperative hepatotoxic drugs. Hepatic dysfunction without these predisposing factors may occur also as a paraneoplastic manifestation of malignancy as in case of hypernephroma. But whether the injured or necrotic renal parenchyma may act in a manner like hypernephroma in causing hepatic dysfunction is not uncommon. In this report, we describe the occurrence of postoperative cholestatic hepatic jaundice without theses over-mentioned reasons in a patient treated by subcapsular nephrectomy for right pyonephrosis.

Case Report

In November 2014, 30 years old female patient presented with right loin pain, which was recurrent and progressive and associated with recurrent attacks of high grade fever of 8 years duration. The patient presented to us with fatigue and night sweats. She was on antibiotics and antipyretics for 3 months with no improvement. General examination was unremarkable except pallor with no detectable jaundice. Abdominal examination showed markedly enlarged right renal swelling which was firm in consistency and tender, the liver and spleen were of normal size. Abdominal Ultrasonography showed advanced hydronephrotic RT kidney, thick echogenic cortex with multiple lower calyceal stones and inspissated pus inside. CT Scanning confirmed the previous findings and showed multiple lower ureteric stones on the same side and excludes the presence of a malignant lesion inside that kidney (Figures 1a-1c).



Figure 1a and 1b: CT scanning showing RT Pyonephrosis with multiple stones and inspissated pus around.



Figure 1c: CT KUB showing multiple RT renal stones and RT lower third ureteric stones.

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Page 2 of 2

Complete laboratory workup was normal except microcytic hypochromic anemia with hemoglobin level of 8.4 g/dl and normal reticylocytic count and urine analysis showed pyuria and RBCs >80. RT subcapsular nephrectomy was done through a flank incision due to marked adhesion of the kidney to the surrounding structures. On exploration, about 5 liters of thick pus was aspirated from the kidney. Estimated blood loss was about 200 ccs. Two units of fresh blood were given to the patient inside the operative room to compensate for the low hemoglobin level preoperatively. One day after the operation, the patient developed severe jaundice. This was associated with normal body temperature. Total bilirubin level was 8 mg/dl, direct bilirubin was 7.2 mg/dl and indirect type was 0.8 mg/dl. SGOT and SGPT were normal (35 and 44 U/ml). Reticulocyte count was normal indicating absence of hemolytic reaction. No hepatotoxic anesthetic drugs were used during the operation. The duration of clinical manifestation was 4 days, the condition was gradually regressive with complete resolution of clinical and bilirubin level before hospital discharge. No specific hepatic treatment was advised by the internal medicine specialists but just follow up. The pathology report showed that the removed kidney had extensive chronic and acute on top of chronic inflammatory changes. Follow-up LFTs was performed 2 weeks after the operation showed normal results (total bilirubin 1mg/dl, direct 0.2 mg/dl and indirect 0.8 mg/dl).

Discussion

Pyonephrosis is a suppurative inflammatory condition of the kidney. Clinical presentation includes fever, chills and flank pain. If left untreated it may be lethal. Ideally, it is surgically drained either through percutanous nephrostomy or ureteral catheter insertion, otherwise antibiotics may not be very effective [1-3]. Nephrectomy in prescence of normal contralateral kidney is the preferred treatment. A retrospective study evaluating 25 patients, who underwent nephrectomy for chronic pyonephrosis, showed that those patients may present with hematological and biochemical abnormalities as marked elevation of plasma viscosity (or ESR) and a raised alkaline phosphatase or reduced serum albumin. This suggests extra renal disease and may lead to diagnostic confusion in a number of patients. Although reduced serum albumin is not specific to hepatic diseases, its association with a raised alkaline phosphatase suggests 'abnormal liver function tests' [4].

Postoperative jaundice is often multifactorial [5]. Jaundice developing in critically ill or injured patients should probably be

thought of as a manifestation of severe sepsis until proved otherwise. Patients with generalized peritonitis may develop jaundice due to septicemia in 50-60% of cases. Biochemically, this jaundice is associated with increased bilirubin (particularly the direct fraction) and liver enzymes (particularly the alkaline phosphatase) and a decrease in the serum albumin. Histologically, there is intrahepatic cholestasis. The etiology of these changes is unknown. It may be due to an end organ response to sepsis. Optimal treatment involves control of sepsis and maintenance of a good flow of well-oxygenated blood to the liver [6].

Our studied case was in concordance with Mukamel et al. (1979) reported case. He reported severe benign intrahepatic cholestatic jaundice appeared immediately after nephrectomy in a patient with nephrolithiasis and septicemia. He added that this diagnostic possibility should be considered whenever jaundice appears postoperatively for pyonephrosis and septicemia and no specific treatment was necessary [7].

Postoperative cholestatic hepatic dysfunction may occur after treatment of some benign inflammatory conditions of the kidney, but it is rare. It has a benign course which is self-limiting. Treatment is consisted of supportive liver treatment and follow-up. It has no residual effect on the liver function in those patients.

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