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Dapsone induced hypoalbuminemia and ascites in lepromatous leprosy

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Abstract
A 66 old year female under treatment for lepromatous leprosy – multidrug therapy with grade 1 deformities of extremities presented with chief complaints of shortness of breath on exertion. Biochemistry revealed anaemia with a haemoglobin of 4.4 gm % with red cell indices suggestive of microcytic hypochromic anaemia (MCV of 81/ MCH of 21.1 /MCHC of 25.9) ,TLC and platelet count within normal limit, vitamin b12 levels >2000 pg/ml. Liver function test revealed total bilirubin 1.01 with sgot/ sgpt 26/13 with alkaline phosphatase 97 with total protein 4.4 with hypoalbuminemia 2.3, Prothrombin time 12.9 seconds / INR.95,serum Creatinine and electrolytes within normal limit serum calcium 8.2 mg% and serum phosphorous 2.7 mg%. Chest x-ray and ecg were normal. Urine routine microscopy revealed no proteinuria.
Ultrasound abdomen revealed liver 12,3 cm in span , spleen 9.2 centimetre in span , common bile duct dilated with no dilated intrahepatic biliary radicles with free fluid in abdomen and pelvis suggestive of ascites. During hospital stay patient was managed with three units of blood transfusion and diuretics. Subsequently the oedema settled and dyspnoea improved. Patient was discharged in stable condition.

Keywords: ascites, leprosy

Introduction
Dapsone, or 4, 4′-diaminodiphenylsulfone, is commonly used in several dermatologic indications such as neutrophilic dermatoses, vasculitis, and autoimmune bullous diseases. Axonal neuropathy is a rare cumulative dose-related adverse effect attributed to direct drug toxicity, occurring after prolonged exposure [1]. Idiosyncratic drug reactions have also been described such as dapsone hypersensitivity syndrome that appears in the few weeks after dapsone introduction, or hepatitis, which generally occurs within a few days after the start of treatment [2, 3, 4, 5].
We report a case of severe dapsone-induced hypoalbuminemia leading to ascites.

Case Report
A 66 old year female under treatment for lepromatous leprosy – multidrug therapy with grade 1 deformities of extremities presented with chief complaints of shortness of breath on exertion. No associated history of fever, cough,sputum or palpitation ,chest pain or orthopnoea. Physical examination was suggestive of anaemia with dependent oedema. No sign of icterus, clubbing, lymphadenopathy and jugular venous pressure was not raised. Respiratory system cardiovascular system examinations were normal. However, examination of abdomen revealed ascites with flanks dull to percuss. Blood pressure 92/60 mm of mercury with pulse rate 92 per minute regular rhythm Biochemistry revealed anaemia with a haemoglobin of 4.4 gm % with red cell indices suggestive of microcytic hypochromic anaemia (MCV of 81/ MCH of 21.1 /MCHC of 25.9) ,TLC and platelet count within normal limit, vitamin b12 levels >2000 pg/ml. Liver function test revealed total bilirubin 1.01 with sgot/ sgpt 26/13 with alkaline phosphatase 97 with total protein 4.4 with hypoalbuminimia 2.3, Prothrombin time 12.9 seconds / INR.95,serum Creatinine and electrolytes within normal limit serum calcium 8.2 mg% and serum phosphorous 2.7 mg%. Chest x-ray and ecg were normal. Urine routine microscopy revealed no proteinuria.
Ultrasound abdomen revealed liver 12,3 cm in span , spleen 9.2 centimetre in span , common bile duct dilated with no dilated intrahepatic biliary radicles with free fluid in abdomen and pelvis suggestive of ascites. Hepatitis B and Hepatitis C serology was negative. Immune status was tested and patient was found to be immune-competent.
5 ml of Ascitic fluid was aspirated which was turbid in appearance, tapped fluid revealed protein content of 1.9 gm/dl with albumin 1.2 gm/dl with a total leukocyte content of 8320 /mm3 with 96% neutrophils with concomitant serum albumin 2.3 gm /dl concluding ascites as high SAAG low protein with ADA18.7u/l. In view of high SAAG, upper gastrointestinal endoscopy was performed which was non-contributory for Varices. During hospital stay patient was managed with three units of blood transfusion and diuretics. Subsequently the oedema settled and dyspnoea improved. Patient was discharged in stable condition.

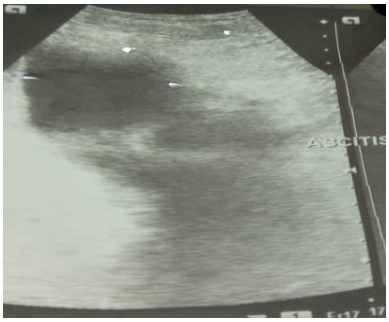

Fig 1: Image: Ultrasound abdomen showing gross ascites

Discussion

It has also been hypothesized that a metabolite of dapsone, an albumin-bound molecule, might act as haptens, leading to the formation of antibodies directed against dapsone and the destruction of both dapsone and albumin [2].

Hypoalbuminemia has also been reported in patients with "dapsone hypersensitivity syndrome," which is a severe multiorgan drug reaction, close to or identical to a drug reaction with eosinophilia and systemic symptoms [2, 4, 9, 10]. However, the "dapsone syndrome" occurred within the first weeks after initiation of dapsone, whereas the 4 cases of dapsone-induced hypoalbuminemia, including the present one, occurred after a much longer delay. Additionally, these 4 patients did not have a rash, fever, lymphadenopathy, blood eosinophilia, and various organ involvement, which are characteristic of dapsone syndrome [6, 7, 8].

Although rare, hypoalbuminemia is a potentially serious adverse effect of dapsone that can be observed several years after its introduction

Conclusion

Dapsone use can lead to hypoalbuminemia and ascites after long term use.

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