BLACK WATER FEVER IN MULTAN

Pages with reference to book, From 194 To 196

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Abstract

Two cases of black water fever are described and in both P. Falciparum rings were present in the peripheral blood. Spleen was palpable and acute renal failure was the presenting feature. Management of black water fever is discussed in detail. Both of these cases recovered completely. None of these cases gave history of intake of quinine. It is suggested that black water fever should be considered as one of the causes of acute renal failure in this part of the world.

INTRODUCTION

Although cerebral malaria, neonatal malaria, malarial complications of lungs, alimentary tract and the heart have been reported from 194.

Peshawar[4], reports about black water fever from this part of the country are missing. We have treated 2 cases of black water fever in Nishtar Hospital, Multan, details of which are as under

CASE NO.1.

M.B., 15 years old school boy from Multan City, was admitted with high grade remittant fever of 17 days duration. He received various types of antibiotics from a local doctor and on the fifth day of ifiness his urine was dark brown resembling pepsi-cola colour and was decreased in quantity. This type of urine continued for 2 days and later on it gradually cleared up. There was no burning micturition, renal or ureteric colic or bowel disturbances. He never took quinine for the treatment. On examination he was severely anaemic, slightly jaundiced, pulse rate 108/minute and temperature 102°F. His spleen was palpable, while liver and lymph nodes were not palpable. Hb was 6.0 g/ul; reticulocyte count 1.9% while TLC, DLC, platelet count were normal. Peripheral blood examination showed occasional rings of P. Falciparum. His blood urea on admission was 350 mg% and serum creatinine 2 mg%, while Coomb's and G6PD deficiency tests was negative. Conservative treatment of acute renal failure, Inj. Chloroquine and Tab. Prednisolone were given to the patient and he gradually recovered in 2 weeks time.

CASE NO.2

S.A. 16 years old farmer, presented with continuous fever since 15 days. He passed dark coloured urine which continued for three days. At the time of admission, though, the colour of urine had become normal, yet urinary output was much reduced. He was markedly pale and slightly jaundiced. His spleen was just palpable and he was drowsy. The reflexes were diminished on both the sides and plantars were down going. Rest of the systems did not reveal any abnormality. His peripheral blood film showed rings of P. falciparum; blood urea was 360 mg%, serum K 6.l mmol/L and sodium 132 mmol/L. He was given chioroquine injection and steroids and recovered with peritoneal dialysis.

DISCUSSION

Two cases of black water fever (B.F.) are described. This condition is brought about by a rapid intravascular haemolysis and is commonly associated with chronic falciparum malaria most commonly in those who have taken antimalarial treatment irregularly or/are deficient in G6PD[3]. The infection of P. falciparum seldom lasts longer than six months[10] and is frequent in indigenous races. The

haemolysis may be quite unexpected and very extensive destroying many uninfected as well as parasitized red cells. The exact cause of this condition is obscure. In many patients parasitism is absent at the time of haemolysis. In the present series the parasites were present in all cases. Because black water fever has usually occurred in patients with chronic falciparum infection who are treated with quinine, it was suggested that the haemolysis results from an autoimmune reaction to red cells that have been altered by the drug, parasite or both[6]. However black water fever has been reported in patients who have not been given any drug. In our cases history of intake of quinine was absent. The most alarming feature of black water fever is the renal failure and in the present series all the cases presented with a renal failure. The renal failure is probably due to renal vasoconstriction reflex. In addition, peripheral circulatory collapse, seen in these cases of shock, makes the condition even worse by reducing GFR. The onset of disease is usually sudden with slight or severe rigors, irregular fever, pain in the loins, over the liver or spleen and urgent desire to pass urine. Epigastric pain with vomiting may also be present. Profuse sweating brings down the temperature. The urine passed is diminished in quantity, dark brown to cherry red in colour and the skin becomes yellow. The patient may gradually recover or may take a serious turn. The urine output in severe cases falls and anuria may ensue. Death occurs in these severe cases in a few hours with hiccough, haemorrhages and heart failure. Black water fever is dangerous for pregnant mothers, during parturition or during puerperium, therefore, these conditions need special care. Another complication noted in black water fever is cholelithiasis. Due to inspissation of bile in the gall blad4er, stone formation occurs, and it may take a few. weeks for the stones to form[6]. Treatment with antimalarial drugs should be given immediately if malarial parasites are present in the blood and the drug of choice is chioroquin; 600 mg. given a single dose in mild attack and in a partially immune person[5,8]. Non-immune adults need 1 .5-2.4g of chioroquin given over 3—5 days period[7,9]. In a patient resistant to chioroquin, quinine sulphate 2.0g daily (in divided doses), for 7-10 days is of value[9]. However a single dose of Sulphadoxine (500 mg) and pyrimethamine (12.5 mg) has also been recominended[9]. Acute renal failure should be treated with haemodialysis, however, if this facility does not exist in the hospital, peritoneal dialysis should be carried out. In renal failure, quinine sulphate should be given in reduced doses (600 mg I/V in normal saline) every day[2]. Severe anaemia needs suspension of red cells especially if TRBC count is less than 1.5 M/ul. Fluids should be given with caution thus intake is 600 mul plus the amount of urine passed over the last 24 hours daily. If the patient is vomiting, sweating or has diarrhoea, additional fluids are given. Corticosteroids have been used with some favourable response[2]. Old practice of giving large doses of alkalies in cases of renal failure has proved ineffective and has been.

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