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AN UNUSUAL DRUG REACTION TO DAPSONE

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A case of acute haemolytic crisis following dapsone treatment in a patient of Hansen's disease is reported.

Key Words : Haemolytic crisis, G6 PD deficiency, Dapsone therapy

Introduction

Dapsone is a commonly used sulphone being prescribed extensively for treatment of Hansen's disease. We herewith report a newly diagnosed case of Hansen's disease in which dapsone was given and patient presented with unusual drug reaction.

Case Report

A 47 years old Hindu male was admitted with history of high coloured urine and pain in abdomen for the last 7 days. The pain was localised to paraumbilical region and used to increase after meals. Patient gave a history of hypopigmented patches over his skin for last 6 months which were gradually increasing in the size. He also noticed that there was diminished sensation on these patches for which he was investigated. Skin biopsy was done and a diagnosis of borderline Hansen's disease was made. He was prescribed dapsone 100 mg/day, rifampicin 450 mg once a month and clofazimine 100 mg A/D.

He took treatment for 2 days only following which he had above symptoms and he stopped treatment immediately. High coloured urine and pain in abdomen however persisted inspite of stopping treatment. Physical examination revealed marked pallor, patient was mildly jaundiced, pulse 100/minute, BP 130/60, heart soft systolic murmur in apical area, lungs-NAD, liver was 1 cm enlarged below costal margin, spleen was also palpable 2 cm below subcostal border. There were 5-6 hypopigmented patches over forearm and other parts of body. These patches were hypoanaesthetic and left ulnar nerve was thickened.

Investigations done on day of admission revealed haemoglobin 5.7 gm%, TLC 48,750 cells/cu mm, DLC polymorph 50%, lymphocytes 17%, eosinophil 02%, monocytes 06%, myeloblast 03%, promyelocyte 04%, myelocyte 07%, metamyelocyte 11%, RBCs were normo-chromic but moderate degree of anisocytosis, poikilocytosis, early and immediate normoblasts were present. Platelets were adequate. Other investigations were, S.creatinine 4.98 mg%, S.uric acid 7.8 mg%, S. bilirubin 2.3 mg% and Wanderberg test- indirect positive. Urine showed presence of albumin and 15-20 pus cells. Ultrasonography examination did not reveal any evidence of obstructive uropathy. A hyperechoic lesion was present in lower pole of right kidney measuring 4x3.6 cm in size. Kidney size was within normal limits.

Patient was put on symptomatic treatment. After 5 days of admission repeat blood examination revealed that

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haemoglobin was now 9 gm%, total count was 12,500 cells per cu mm with normal differential count. Platelet count was 16% and RBC showed hypochromic picture with anisocytosis, macrocytosis and polychromasia. S. creatinine level also came down to 2 mg%, glucose-6 phosphate dehydrogenase estimation revealed values to be 15.3 mu/10^9 RBC (N 131 mu/10^9 RBC).

Discussion

Glucose-6 phosphate dehydrogenase (G-6PD) deficiency is the commonest hereditary enzyme defect seen in clinical practice. It is an X-linked inherited disorder with incomplete dominant expression.

G-6PD deficiency is observed frequently in North West India, Sri Lanka, countries of South East Asia and the littoral states of Mediterranean sea and black races but very few case of this disorder has been reported from this part of India.[1] Affected persons are apparently normal. On exposure to certain drugs with oxidant properties such as antimalarial, nitrofuran, sulfones, sulphonamide, naphthalene (Moth balls), probenecid, PAS, analgesic etc. patient develops severe intravascular haemolysis.

Individuals with an inherited defect in the hexose monophosphate shunt are unable to maintain an adequate level of reduced glutathione in their red blood cells, as a result sulfhydryl group in haemoglobin becomes oxidised and haemoglobin tends to precipitate within RBC and in severe reactions haemolysis takes place. Acute severe haemolysis may precipitate acute tubular necrosis leading to acute renal failure in these cases.

As there is no specific therapy a due caution should be taken in prescribing known oxidant drugs in geographical areas known to have high prevalence of G-6PD enzyme deficiency. Patient should also be forewarned to report back if he develops any above symptoms.

References

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