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Case Report

Infections and reactions in leprosy: a diagnostic dilemma

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ABSTRACT

Leprosy (Hansen's disease) is a chronic infectious disease caused by *Mycobacterium leprae*. It continues to be a public health problem in India, which contributes about 60% to the world leprosy burden. Leprosy patients when on treatment can develop either lepra reactions or reactions due to antileprosy drugs, also they can develop other infections endemic in their areas during the course of their disease. We are presenting such two cases where in one case patient on treatment with multibacillary multidrug therapy (MBMDT) developed fever, lympadenopathy and other systemic features during the course of therapy and was mistakenly diagnosed as type 2 lepra reaction but turned out to be a case of dapsone hypersensitivity. Similarly another case developed fever and other systemic features after 6 weeks of MBMDT, thinking it to dapsone syndrome his MBMDT pack was stopped but later it turned out to be a case of dengue.

Keywords: Dapsone syndrome, Dengue, Leprosy, Lepra reaction

INTRODUCTION

Leprosy (Hansen's disease) is a chronic infectious disease caused by *Mycobacterium leprae*. It continues to be a public health problem in India, which contributes about 60% to the world leprosy burden.[1]

Leprosy patients when on treatment can develop either lepra reactions or reactions due to antileprosy drugs, also they can develop other infections endemic in their areas during the course of their disease.

We are presenting such two cases where in one case patient on treatment with multibacillary multidrug therapy (MBMDT) developed fever, lympadenopathy and other systemic features during the course of therapy and was mistakenly diagnosed as type 2 lepra reaction but turned out to be a case of dapsone hypersensitivity.

Similarly another case developed fever and other systemic features after 6 weeks of MBMDT, thinking it to dapsone syndrome his MBMDT pack was stopped but later it turned out to be a case of Dengue.

CASE REPORT 1

48 years old otherwise healthy male came in the leprosy clinic of tertiary care centre (medical college) presented with multiple hypopigmented patches over buttocks and thighs with decreased sensations and loss of sweating over the patches. On examination there was thickening of bilateral ulnar nerves. Punch biopsy was sent for histopathological confirmation which revealed epitheloid

cells and lymphocytic infiltration around nerve terminals. He was then diagnosed as a case of borderline leprosy and was put on MBMDT.

After completing two packs he developed sudden onset of high grade fever, pain abdomen, lymphadenopathy, flaring up of old lesions as well as eruption of new painful erythematous plaques over the legs. These features were clinically suggestive of type 2 lepra reaction. On further laboratory investigations- complete blood count, renal function tests were within normal limits with markedly elevated liver enzymes. His MBMDT was continued along with the course of oral corticosteroids and non-steroidal anti-inflammatory drugs were given. Despite treatment, his clinical condition deteriorated, patient developed frank jaundice. Second possibility of dapsone syndrome was then considered, his multidrug therapy was modified with elimination of dapsone. After stopping dapsone there was marked improvement clinically with resolution of erythema and corresponding fall in liver enzymes.

CASE REPORT 2

26 years old male presented in urban leprosy centre with multiple hypopigmented hypoaesthetic patches of varying sizes over trunk and limbs for the last one year. Thorough clinical and laboratory investigations e.g. complete blood count, fasting blood sugar levels, liver function test, kidney function test, G-6PD deficiency were done. Everything was normal and he was classified as a case of borderline tuberculoid leprosy and was put on MBMDT. He was advised monthly follow up. After six weeks he came with high grade fever, sore throat, nausea, deranged liver function tests and thrombocytopenia. Before coming to leprosy centre he went to some private practitioner for treatment of fever who stopped his MBMDT thinking it to be a reaction to the drugs but no improvement was seen.

He was then further investigated under medical supervision for fever along with dapsone hypersensitivity and rifampicin toxicity as the additional differentials. This was the time when already more than 600 cases with dengue fever had been reported officially in the district. His blood reports showed haemoglobin 11 g%, TLC 4500, PCV 39, Platelet count 80,000, SGOT/PT 112/184 and dengue NSI positive so he was labeled as a case of dengue infection and was put on symptomatic treatment and with strict follow up. Complete blood count was done on a daily basis. His recovery started after about a week and MBMDT was once again restarted after complete clinical and haematological cure. Even now he is coming for regular follow ups and is healthy.

DISCUSSION

Leprosy is a chronic disease affecting peripheral nervous system, skin and certain other tissues after an incubation period of 5 to 7 years. Disease currently affects approximately a quarter of a million people throughout the world, with majority of these cases being reported from India.[2,3] Borderline leprosy and the subtypes are characterized by more extensive disease than polar tuberculoid, with more numerous skin lesions and more nerve involvement, but not as widespread disease as in lepromatous leprosy.[4] Tuberculoid type induces a cell-mediated response that limits its growth whereas there is an immunological instability in the borderline type with a tendency to move on either side of the spectrum. It is the deficient cell mediated immunity and delayed hypersensitivity response to *M. leprae* that are responsible for an individual developing leprosy as a result of contact with the bacterium, and its degree of deficiency which determines the type of leprosy.[5]

Dengue is an acute viral infection transmitted by *Aedes aegypti* mosquito common in tropical climate. The incidence of dengue has grown dramatically around the world and over 40% of world's population is now at risk of contracting this disease.[6] Symptoms typically begin three to fourteen days after infection showing high grade fever, headache, vomiting, muscle, joint pains and skin rash with recovery generally taking two to seven days.[7] Some people never have significant symptoms but can still infect mosquitoes. Dengue has become a global problem since the second world war and each year between 50 and 528 million people are infected and approximately 10,000 to 20,000 die.[8,9]

When a mosquito carrying dengue virus bites a person, the virus enters the skin together with the mosquito's saliva. It binds to and enters white blood cells, and reproduces inside the cells while they move throughout the body.[10] The initial reaction of infected cells is to produce interferon, a cytokine that raises a number of defenses against viral infection through the innate and adaptive immune system, which leads to the generation of antibodies against the virus as well as T cells that directly attack any cell infected with the virus.[11]

The possible explanation to this co infection can be the persistence of *M. leprae* within the already immune-tolerated liver and immunologic instability in the borderline spectrum of the disease which led to decreased defenses against dengue virus infection which was already multiplying unchecked in the district during that time.

Dapsone (DDS) syndrome is also called the "five week dermatitis", because it suddenly occurs five to six weeks after the administration of dapsone. It usually subsides on cessation of dapsone therapy. This reaction is characterized by sudden onset of papular or exfoliative rash, accompanied by fever, malaise and weakness, followed by jaundice and tenderness of liver, lymphadenopathy and mononucleosis (lymphocytes and monocytes 70%). However, all symptoms need not necessarily be present.[12]

The pathogenesis of the dapsone syndrome is as yet unknown. However, it is reported to have been probably due to hypersensitivity, because there was an interval of 5 to 6 weeks from the beginning of therapy in every case. Dapsone is metabolized in two pathways, N-acetylation and N-hydroxylation (oxidation). The formation of toxic intermediate metabolites such as nitrosamines and possibly other compounds through N-hydroxylation pathway are thought to be responsible for the haemolytic anemia, methemoglobinemia and dapsone syndrome.[13] However, the production and detoxification of toxic metabolites of dapsone is influenced by a number of genetic and environmental factors.[14]

CONCLUSION

These cases were underscored to highlight the fact that various clinical manifestations in leprosy patients can masquerade as lepra reaction. Vigilant clinical examination and laboratory investigations should be undertaken as it could prove fatal to stop or continue MDT in haste. Various adverse drug reactions for example dapsone syndrome can occur invariably in predisposed patients with grievous outcomes if physicians and field workers are not familiar with it. Similarly epidemic viral infections in developing countries can simulate lepra reactions. Thus these patients should be carefully evaluated during follow up for any co infection or side effects of drugs.

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