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Carbamazepine Induced Pseudo-Lymphoma Syndrome

H. Paramesh
D.A. Satish
Salim A. Khatib
Mukta Jain M.

Pseudolymphoma syndrome, as a hypersensitive reaction to anticonvulsant drugs especially phenytoin, carbamazepine, tridone and phenobarbitone is well known(l-4). Its pathogenesis is uncertain. It consists of a triad of fever, generalized rash and lymphadenopathy. Varying degrees of malaise, hepatosplenomegaly, abnormal liver functions, arthralgia, eosinophilia and, blood dyscrasias have also been reported^). We present a case of carbamazepine induced pseudolymphoma syndrome.

From the Departments of Pediatrics and Dermatology, Lakeside Medical Center and Hospital, Bangalore.

Reprint requests: Dr. H. Paramesh, Director, Pediatrician-in-Chief, Lakeside Medical Center and Hospital, 33/4 Meanee Avenue, Bangalore 560 042.

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Case Report

A 5-year-old male patient was given carbamazepine (Tegretol) in the dose of 200 mg once daily for focal seizures of the right foot for 2 months after evaluation by a pediatric neurologist. He presented with fever and generalized erythematous maculopapular rashes of 15 days duration, which started one-and-half months after initiation of treatment with carbamazepine. He also presented with anasarca and was passing high colored urine.

Examination revealed exfoliative dermatitis, icterus, generalized lymphadenopathy (bilateral cervical, axillary, and inguinal lymphnodes were enlarged) and hepatosplenomegaly. Respiratory system examination revealed bilateral crepitations and rhonchi while the cardiovascular and central nervous systems were normal.

Investigations revealed a total leucocyte counts of 18,000 cells/cu mm, with a differential count of polymorphs 60, lymhocytes 26, monocytes 8 and eosinophils 6. Hemoglobin was 10.6 g/dl, RBC count was 3.5 million/cu mm, PCVwas 33% and ESR 19 mm/ in first hour. Peripheral smear revealed 17% of atypical lymphocytes. Urine and stool examination were normal. Blood urea nitrogen, serum creatinine, ASO titres were all normal. C-reactive protein was 2.4 mg/dl. Liver function tests showed a raised serum bilirubin (3.6 mg/dl), serum alkaline phosphatase of 8.0 KA units, SGOT of 248 units/ml and SGPT of 319 units/ml. Widal test was negative and blood culture yielded no organisms. Paul Bunnel test and mono-spot test were also negative. X-ray chest revealed right hilar lymphadenopathy.

The drug tegretol was discontinued and the patient was treated with antihistaminics, bronchodilators and antibiotics for his reactive airway problem and respiratory infection and local soothening agents.

The fever subsided about 5 days after stopping carbamazepine. The generalized rash and hepatosplenomegaly disappeared within 10 days. The atypical lymphocytes in peripheral blood smear were absent after 10 days. Liver function tests returned to normal in 3 weeks time (SGPT returned to a level of 35 units/ml and. SGOT to 40 units/ml).

Discussion

Pseudolymphoma syndrome was first reported in 1988. The first reported Indian case was in 1991(6). Phenytoin is said to be the commonest drug causing this syndrome. It occurs in about 1% of patients receiving the drug(7). Carbamazepine also does not lag behind. Other drugs known to cause this syndrome are mephytoin, trimethadone, mexiletine (antiarrhythmics), thioridazine and butabarbital.

This condition may also present as a generalized exfoliative dermatitis(8). The minimum clinical criteria for the diagnosis include: (/) Exposure to drugs as stated earlier; *(it)* Triad of fever, generalized rash and lymphadenopathy; and *(iii)* Improvement on stopping the offending drug. Other features seen are malaise, hepatosplenomegaly, arthralgia, congestive cardiac failure, thrombocytopenia, eosinophila, abnormal liver function tests, and blood dyscrasias. Histological investigation of the involved skin has been reported in a few cases. It may reveal a mycosis fungoides like picture or a sezary like syndrome(8). It has been proposed that phenytoin-induced pseudolymphoma syndrome may develop frank malignant lymphoma, because there is an increased chance of a malignant clone developing at a time when the immunosurveillance system is impaired due to lymphadenopathy and a loss of T-cell suppressor function(4).

All cases earlier reported developed the symptoms within 2-8 weeks of starting the offending drug. Our patient also presented with the triad of fever, rash, lymphadenopathy and hepatosplenomegaly, abnormal liver functions and leucocytosis about one-and-half-months after starting the drug.

Due to fever, lymphadenopathy and rash, this condition has to be differentiated from viral infections especially infectious mononucleosis and also bacterial infections. Awareness of this entity helps in an early diagnosis. Treatment consists of omitting the offending drug and systemic steroids, if necessary. Our patient was managed symptomatically.

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Chronic Lung Disease Following Stevens-Johnson Syndrome

Mona Basker
Thomas Cherian
P. Raghupathy

Stevens-Johnson syndrome (SJS) is an acute self-limited eruption of the skin and mucous membranes which represents a hypersensitivity reaction to various etiologic agents(l). Organ involvement, including acute pulmonary lesions, have been described previously(2,3). However, chronic lung disease is an extremely rare complication of SJS. We present here a case of chronic lung disease following SJS in a previously well child.

Case Report

An 8-year-old boy who was well till 2 months prior to admission, received oral

From the Department of Child Health, Christian Medical College and Hospital, Vellore 632 004.

Reprint requests: Dr. Thomas Cherian, Department of Child Health, Christian Medical College and Hospital, Vellore 632 004, Tamilnadu.

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