Drug Induced Lymphadenopathy

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Introduction: Many a times lymphadenopathy is misdiagnosed as tubercular but there is more into it. We report one such case where a wrongly diagnosed lymphadenopathy revealed itself in a different way.

Case Description: An 18 year old female presented with bilateral swelling in cervical region(fig 1), dry cough, fever and reduced appetite since one and a half years . FNAC of cervical lymph nodes was done which showed necrotising lymphadenitis. Anti tubercular treatment was thus started but patient defaulted after one month due to intolerance. Though Sputum for mycobacterium was negative but somehow ATT was continued. She also had gingival hyperplasia and rashes on face (fig 2). Routine investigations like complete blood counts, liver and renal function test did not reveal any abnormality. Peripheral Smear showed anisopoikilocytes with microcytic hypochromic picture. Chest X-ray(fig 3) revealed mediastinal widening. Subsequently HRCT (fig 4) revealed bilateral hilar and mediastinal adenopathy. Ultrasound Abdomen showed no organomegaly or paraaortic lymph node enlargement. Serum ACE and Urinary Calcium levels were normal. Mantoux test was positive. During hospitalization patient became hypotensive and comatose. Limbs were flaccid and deep tendon reflexes were absent. There was dribbling of urine without bladder distension and passing motions without awareness. Detailed past history of other diseases and drug intake was inquired. Patient’s attendant told she was on phenytoin sodium 300mg since 4 years for generalised tonic clonic seizures. However, she discontinued medical follow up and continued to take the drug. Hence serum Phenytin levels were done which showed increased levels(32.30), In MRI Brain mild diffuse Cerebellar atrophy was present. Thus, Phenytin sodium was replaced by levetiracetam. Gradually patient improved and lymphadenopathy regressed on further follow up.

Discussion: Though we make every effort to obtain detailed treatment history of our patients this patient assumed that the consumption of Phenytin was different from this disease, that’s why patient’s relatives did not disclose phenytin consumption in the beginning. This sort of behaviour is sometimes noted in rural patients who often have prejudices of different kinds. Phenytin use has been associated with various lymph node abnormalities. Lymphadenopathy in association with the use of hydantoin derivatives such as phenytoin was first described in 1940 by Coope and Brown. In 1959, Salzstein and Ackerman reviewed 75 cases and reported a further 7 patients, two of whom subsequently died 5 years later due to lymphoma. In 1966, Hymann and Somers reported 6 patients on anticonvulsant therapy who developed a histologically proven Hodgkin’s lymphoma or lymphosarcoma. More recent publiction divides these occurrences into two, the anticonvulsant hypersensitivity syndrome, and the Phenytoin induced pseudo lymphoma. The anticonvulsant hypersensitivity syndrome develops within 8 weeks after the drug is first prescribed. In contrast to hypersensitivity syndrome, Phenytoin induced pseudo lymphoma is a late effect of phenytoin therapy which can occur years after initiating therapy, as in our case study. In our case study the patient had gingival hyperplasia, gum hypertrophy, rashes on face and cerebellar atrophy as occurs in cases of phenytoin toxicity. Gingival hyperplasia occurs in about 20% of all patients during chronic therapy and is probably the most common manifestation of phenytoin toxicity in children and young adolescent. Also there was generalised atonia of all the limbs along with bowel and bladder atonia which hasn’t been reported till date. After discontinuing the patient’s Phenytoin, atonia resolved over few days and gradually the lymphadenopathy regressed.

On conclusion, pathophysiology of phenytoin-induced lymphadenopathy is incompletely understood. It can require careful pathological scrutiny to differentiate it from monoclonal cell expansion. Given the number of patients regularly using phenytoin, it is an important clinical entity that general physicians need to be cognizant of.

REFERENCES:
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Fig no:1 –Cervical Lymphadenopathy

Fig No:2Hyperpigmented Skin Rashes on face

Fig No: 3 Mediastinal widening and bilateral infiltrates

Fig No: 4 HRCT thorax showing bilateral hilar and medistinal adenopathy.