

Isoniazid Induced Unilateral Painless Gynaecomastia

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Abstract: Gynaecomastia is a rare adverse drug reaction due to isoniazid therapy. Here we report a 21 year old male patient, a case of disseminated tuberculosis treated with ATT for 6 months, developed gynaecomastia two months after completion of ATT with Isoniazid (H), Rifampicin (R), Pyrazinamide (Z), Ethambutol (E).

Keywords: Tuberculosis, Isoniazid, gynecomastia

1. Introduction

Gynaecomastia due to anti-tuberculous drug is a rare side effect. Isoniazid causing breast tissue enlargement has been very rarely reported. Isoniazid is the key antituberculous drug and widely used as an effective drug in all ATT drug regimen. It is bactericidal against metabolically active bacilli and bacteriostatic against resting bacilli. The well known adverse effects of isoniazid are hepatitis, peripheral neuropathy, cutaneous reactions and mental changes. Very rarely it can cause gynaecomastia.

2. Case Report

A 21 year old male patient presented with fever, anorexia and loss of weight of 3 months duration with contact history of Tuberculosis. Clinical examination revealed temperature 101°F, cervical and inguinal lymph node enlargement with hepatosplenomegaly. The patient was investigated as follows:

Investigations	Report
TC	9500 cells
ESR ½ hr	42 mm
1 hr	80 mm
Mantoux test	Negative
Sputum AFB	Negative
X-ray chest PA view	B/L Hilar enlargement Right upper zone small patchy lesion
CT Abdomen	Multiple enlarged necrotic lymph nodes with hepatosplenomegaly with right infra hilar lymph nodes. Suggestive of tuberculous etiology.
FNAC cervical lymph node	Caseating granulomatous lymphadenitis

Patient was diagnosed as Disseminated Tuberculosis and started on Category 1 ATT. After completing ATT course for 6 months, he recovered completely, regained weight and started his routine work. Subsequently after 2 months he developed unilateral painless left sided gynaecomastia.



3. Discussion

Gynaecomastia is a benign enlargement of male breast tissue. It can occur due to numerous causes which include developmental gynaecomastia, congenital causes like klinefelter's syndrome, enzyme defect on testosterone production, acquired causes like chemotherapy, malignancies like bronchogenic carcinoma and alcoholism, systemic causes like cirrhosis of liver and drugs.

Certain drugs, for e.g., spirinolactone, cimetidine causing gynaecomastia is common. However isoniazid causing gynaecomastia is very rare, so far only few reports are published. In case of INH induced gynaecomastia the exact mechanism is not clear. It has been hypothesized that disturbance in vitamin B6 complex activation in liver leading to alteration in oestrogen-androgen metabolism. Another possible mechanism is refeeding gynaecomastia due to recovery from a chronic disease with improved nutrition and restoration of weight, gonadotropin secretion and gonadal functions.

Among antituberculous drugs apart from INH, Ethionamide and Thioacetazone have been incriminated in causing gynaecomastia. First two reports on INH induced gynaecomastia were published in 1953 and 1976. One of these reports described painless gynaecomastia during 4 months of daily therapy with INH 600 mg/day. A recent report published in English literature described bilateral painful gynaecomastia after 4 months of daily therapy with INH 300 mg/day

Mostly patient with gynaecomastia requires no treatment other than removal of any inciting cause if present. Specific treatment is indicated only if it is causing sufficient pain, embarrassment or emotional discomfort to interfere with patient's daily life.

4. Conclusion

Incidence of INH induced gynaecomastia is very rare. And here our patient presented with unilateral painless gynaecomastia 2 months after completion of 6 months daily regimen of INH- dose 200 mg/day and the probable mechanism could be refeeding gynaecomastia

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