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Case Report

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PERIPHERAL NEUROPATHY -AS ADVERSE EFFECT OF MULTI DRUG RESISTANT PULMONARY TUBERCULOSIS CHEMOTHERAPY.

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ABSTRACT

Multidrug-resistant tuberculosis is defined as mycobacterium tuberculosis that is resistant to both isoniazid and rifampicin, with or without resistance to other drugs. Emergence and spread of multi drug resistant tuberculosis (MDR TB) can threaten the global Tuberculosis control. The treatment of MDR TB is prolonged, expensive and often unsuccessful. Drug toxicity are important factors leading to defaultation or poor treatment adherence and ultimately low cure rate in MDR TB treatment. Here we report a case of MDR Pulmonary Tuberculosis patient presented with us peripheral neuropathy as a adverse effect of second line drugs.

KEYWORDS: MDR TB, Peripheral Neuropathy.

INTRODUCTION

MDRTB results from either primary infection with resistant bacteria or may develop in the course of a patient's treatment. The emergence of drug resistant mycobacteria has become a significant public health problem world over creating an obstacle to effective TB control. Treatment of MDRTB is challenging due to toxicity of second line drugs. Most common adverse event is gastro intestinal in different studies.[1,2] Though severe adverse reactions were frequent, treatment could be continued in most cases with modifications of the treatment regimen.

CASE REPORT

A 40 years old male suffering from multidrug resistant pulmonary tuberculosis presented with chief complaints of On and off fever for last 10 months, On and off cough for last 10 month, Breathlessness on exertion for last 4 month, Tingling and Numbness in bilateral (B/L) lower limbs for 1 month and generalized weakness for 15 days.

He was non diabetic, non alcoholic and normotensive. There was no history of Chest pain, backache, any bladder and bowel symptoms Respiratory distress, Speech difficulty, Diplopia **,** Vaccination in recent past. Treatment History CAT I DOTS for 5 months (April 2015- August 2015) and CAT IV DOTS (Kanamycin, Cycloserine, Ehionamide, Ehambutol, Levofloxacin, Pyrazinamide, Pyridoxin) Since October 2015.

On examination

Patient was Conscious, oriented to time place and person, Thin built, Pulse rate- 106/min Respiratory rate- 24/min, Blood pressure- 100/76 mm Hg, Temperature- 99.2 ^{0}F, SPO_2- 95% with room air. There was no Pallor, Icterus /cyanosis/clubbing/ lymphadenopathy. In Respiratory System Examination Chest is B/L symmetrical, decreased movement of chest in B/L suprascapular region, decreased intensity of breath sound in B/L suprascapular region. In Central Nervous System examination he was Conscious, well oriented to time place person, Higher Mental Function with in normal limit. Neck Rigidity absent, Kernig's sign absent, Brudzinski sign absent. All cranial nerves examination were within normal limit. Decreased tone in B/L lower limbs and upper limbs. Power of Proximal muscles were of grade 5/5 and distal muscles were Of Grade 4/5 in lower limbs. Cordination intact in upper and lower limbs. Superficial – pain, temperature and touch sensations were lost in the distal part of the limbs and involvement was B/L symmetrical. Vibration sense, Position sense, Joint sense lost distally. Abdominal, cremastric, plantar reflex did not show response. Biceps and Triceps reflex were normal and Supinator and Knee jerk diminished, Ankle jerk absent and Clonus absent B/L. Abdominal and Cardiovascular System examination within normal limit. Lab investigation Hb-11.2 gm/dl), Polymorphonuclear leucocytosis (WBC-14000/cmm with N 85%, L 13%, M 1%, E 1%) Raised ESR (41 mm/1st hour), LFT/KFT/Serum electrolyte/Thyroid function/Lipid Profile were WNL, USG abdomen-

normal. Nerve conduction velocity study (**NCV) was suggestive of axonal poly neuropathy (LL>>UL) (MOTOR >>SENSORY).**

DISCUSSION

Some of the antitubercular agents may cause peripheral or optic neuropathy. Ethambutol is less neurotoxic but may cause optic neuropathy, a mixed sensorimotor neuropathy, or a predominantly sensory neuropathy.[3] Ethambutol is recognized to cause neuropathy in a dose dependent manner. Ethionamide is structurally related to isoniazid, may rarely cause sensory neuropathy which takes several months to resolve upon discontinuation.[4] The drugs most commonly implicated are Isoniazid, Ethionamide, Cycloserine, and Linezolid. Fluoroquinolones and Ethambutol have rarely been associated with the development of neuropathy.[5] The extensive literature survey has revealed that very few previous published case reports of Ethambutol or Ethionamide alone or in combination induced peripheral neuropathy in India.

Co administration of pyridoxine (50-100mg/ day) is protective, although excessive doses >200mg/day can cause peripheral neuropathy especially in individuals with end stage renal disease.[5] We present a case of mild sensory motor neuropathy due to either concurrent administration of ethambutol (E) or/and ethionamide (Eto) or it may be a case of exacerbation of neurotoxic effects of cycloserine due to concomitant administration of E+ Eto+ Lfx.

CONCLUSION

The diagnosis of peripheral neuropathy can be based on clinical presentation alone and effective management of this side effect is possible without sacrificing MDRTB treatment efficacy. Management strategies depended on: Severity of symptoms, Treatment of contributing comorbidities, Medications for neuropathic pain and Adjustment of doses of possible offending agents.

Conflicting Interest: None.

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