Isoniazid Hypersensitivity Reaction Involving the Eyes
Report of a Case

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INTRODUCTION

Isoniazid is the most widely used drug in the treatment of tuberculous diseases throughout the world. Since its advent in 1952, it has remained the most potent tuberculostatic among the drugs so far discovered in this group. It is a comparatively safe drug and untoward reactions are not frequent. The majority of its untoward effects are due to its toxicity and related to dose, manifesting in peripheral and central nervous system and other organs of the body. The most commonly encountered toxic effect is peripheral neuropathy, as isoniazid competes with pyridoxal phosphate for enzyme apotryptophanase, upsetting tryptophane metabolism leading to pyridoxine deficiency. Isoniazid-induced peripheral neuropathy responding well to withdrawal of the drug and addition of pyridoxine. Among central nervous system reactions, toxic encephalopathy, convulsions, dizziness, ataxia and mental changes are worth mentioning. Administration of pyridoxine for these reactions gives favorable response in cases of peripheral neuropathy, but is of limited value in toxic encephalopathy where residual neurologic lesions tend to remain severe, while it has no beneficial effect regarding ataxia, convulsions and mental changes.1

Untoward effects due to isoniazid hypersensitivity are relatively rare and usually manifested by pyrexia, hepatitis and skin eruptions of wide variety, e.g. erythematous, urticarial, purpural, papular, etc. Untoward effects in this group respond to prompt withdrawal of drug and antihistamines. The writers of this case report were unable to find in the literature a case of isoniazid hypersensitive reaction involving the eyes.

CASE REPORT:

This 36-year-old, light complexioned Negro woman, was admitted in January, 1966 with a confirmed diagnosis of tuberculous pleural effusion on the right side. On admission, her temperature was 98°F, pulse 92 per minute, regular, height 5 feet, 3 inches, weight 114 pounds, blood pressure 140/70. She was two months' pregnant (para six) at the time of admission.

Antituberculosis treatment was started with PAS 12 grams and isoniazid 300 mg daily. She tolerated both of these drugs well until February 12, 1966, when she started having itching, a burning sensation, marked redness and impairment of vision in the right eye. On examination, the right eye revealed a semidilated oval, fixed pupil with two plus cellular reaction in the anterior chamber, without keratitic precipitate formation, marked subconjunctival congestion and multiple hemorrhages. The best visual acuity obtained in the right eye was 20/100, and intra-ocular pressure was eight with Schiotz tonometer, using 5.5 gm weight. Funduscopic examination revealed marked optic neuritis with flame-shaped hemorrhages extending towards the nasal side from disc. The left eye acuity was 20/20 with normal fundus and intraocular pressure of 5 with the Schiotz tonometer using 5.5 gm weight. This picture of acute optic neuritis, iridoplegia, cycloplegia and subconjunctival hemorrhages was suspected to be a hypersensitization reaction to isoniazid.

TREATMENT

Isoniazid was stopped and the patient was given streptomycin 1 gram daily instead. She was given pyridoxine and steroids orally (prednisone 60 mg daily to start with, then tapered gradually) as well as ophthalmic drops. The patient showed progressive improvement on this regimen and by March 22, 1966, her corrected acuity of vision on the right with plus 2.5 sphere was 20/30; intraocular pressure was normal. Only an occasional cell in the anterior chamber was seen, and on funduscopic examination no hemorrhage or edema of the optic nerve head was noted. Subconjunctival hemorrhages completely cleared, and pupillary reaction was beginning to be present. The cycloplegia was not improved.

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Regarding her pleural effusion, she did very well on PAS and streptomycin sulfate.

**DISCUSSION**

This is an unusual case of isoniazid hypersensitivity causing subconjunctival hemorrhages, optic neuritis, iridoplegia and cycloplegia. This was definitely a hypersensitivity and not a toxic reaction. The patient received only 300 mg of isoniazid daily, and she weighed 114 pounds. She had normal kidney function. Withdrawal of isoniazid and a short course of steroids led to satisfactory recovery — although cycloplegia persisted.

Isoniazid hypersensitivity should be suspected in patients receiving this drug and developing such eye complications. Early recognition of the condition, prompt withdrawal of isoniazid and treatment with steroids may reverse much of this untoward reaction.

**REFERENCES**


For reprints, please write: Dr. Ahmad, E. N. C. Sanatorium, Wilson, North Carolina.

**ASPERGILLOMA IN A CAVITY OF EXCAVATING CARCINOMA**

A case of cavitating bronchogenic carcinoma with aspergilloma developing in the cavity is described. In the patient, a woman age 26, initially tuberculosis was diagnosed and she was treated with antituberculosis drugs without any improvement; lung abscess was then suspected and further two-month treatment with various antibiotics was applied. Finally, cavernostomy was done as a preparatory measure for resection; the biopsy specimen showed squamous cell carcinoma coexistent with Aspergillus fumigatus. General condition of the patient was poor; it did not improve after cavernostomy, and soon manifestations of cerebral metastases developed. The patient died at home. The case reported is the third described in the world literature of cavitating carcinoma with the development of aspergilloma within the cavity. Five-month therapy with massive doses of various antibiotics aided the development of aspergilloma.

**ACUTE BENIGN PERICARDITIS**

The author observed two patients with acute benign pericarditis. In one of the cases, pericarditis was combined with an obscure neurologic symptom which was considered diencephalitis. A favorable outcome of the disease was confirmed by three-year observation. The differential diagnostic difficulties are briefly discussed.

**EXPERIMENTAL AUTOTRANSPLANTATION OF PULMONARY TISSUES**

Thirty-one dogs were subjected to reimplantation of the lower lobe of the left lung. Eight of these animals survived for periods ranging from 26 to 263 postoperative days. The major cause of death in these animals was vascular thrombosis, mainly involving the major pulmonary vein. In the eight long-term surviving animals, there was no statistically significant difference in the oxygen uptake and the expired carbon dioxide between the reimplanted and the contralateral normal lower lobe.

Serial biopsies obtained between 23 and 252 days following reimplantation showed patency of the alveoli but some thickening of the alveolar walls, peribronchial fibrosis and vascular congestion. These abnormal findings were not seen in the later period between 142 and 252 days after reimplantation.

**REFERENCES**