

Epidemic Dropsy in a Family

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Abstract

Argemone poisoning usually occurs in outbreaks. Isolated cases are uncommon. We here report epidemic dropsy in a family long after the major epidemic was supposed to be over in India.

Key Words

Epidemic dropsy, Argemone oil.

Introduction

Epidemic dropsy is a disease characterized by oedema, cardiac insufficiency, gastrointestinal disturbances and vascular changes resulting from inadvertent ingestion of seeds of *Argemone mexicana* (the Mexican poppy). This weed has worldwide distribution and cases of epidemic dropsy have been reported from Mauritius, Fiji, Myanmar, Australia, Madagascar and South Africa, besides India (1). Seeds of this weed resemble mustard seeds and it may be difficult to prevent casual mixing of the two where the crops grow together in spite of the fact that their harvesting is separated by a period of six to eight weeks. A major outbreak of the disease occurred in August - September 1998 in the northern parts of India. We here report occurrence of epidemic dropsy in a family in Jammu in March, 1999 long after the epidemic was supposed to be over.

Case Report

A 28-year old man with average socio-economic status reported to Government Medical College, Jammu in late March 1999 with bilateral pedal oedema of 20 days duration. He had facial puffiness. There was low-grade intermittent fever with rigors and chills right from the beginning and it had subsided on its own 5 days before admission. There was no history of breathlessness or chest pain or palpitation. His urine output was adequate. Bowel habits were normal. On examination, he was afebrile with pulse rate of 102/mt, respiratory rate of 18/mt and his blood pressure was 140/70 mm of Hg. JVP was raised and liver was palpable, non-tender and was 3 cms below the costal margin. There was bilateral pitting pedal oedema, local temperature of both calves and ankles was raised and these were tender to touch. Ankles had erythematous hue. His haemoglobin was 11 gm%. TLC 6000/mm³ and ESR was 35 mm in

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1st hour (Westergran). His urea (32 mg/dl) and creatinine (1.4 mg%) levels were normal and urine examination revealed no abnormality. ECG showed sinus tachycardia. Fundus examination revealed hyperaemic disc with superficial retinal haemorrhages in both the eyes (Fig. 1,2). Intraocular tension was normal. Skin biopsy revealed mild increase in melanin in the basal layer with oedematous superficial dermis.

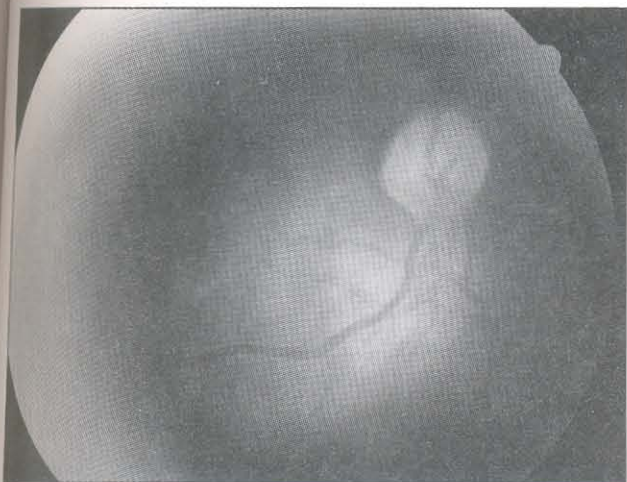


Fig. 1. Shows peripapillary haemorrhage in the retina.

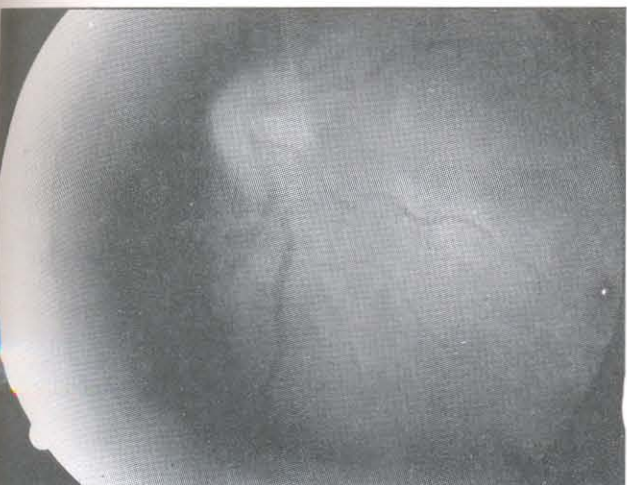


Fig. 2. Shows superficial haemorrhage in the retina.

Wife of this man (23 years old) had bilateral pedal oedema of twenty days duration. She too reported with facial puffiness and low-grade intermittent fever at the time of admission. She had exertional dyspnoea brought

on by ordinary activity. There was no history of chest pain or palpitation. Her urine output was adequate, bowel habits normal and appetite good. She had pallor, pulse rate was 86/min, respiratory rate 24/min and BP 110/60 mm of Hg. JVP was not raised. She had bilateral pedal oedema and the ankles were erythematous and tender. Liver was palpable, non-tender and was 2 cms below costal margin. Haemoglobin was 8.5 g/dl, TLC 7500/mm³ and ESR 45 mm in 1st hour. Serum urea (24 mg/dl) and creatinine (1.2 mg %) levels were within normal range and fundus examination was normal. Skin biopsy report indicated oedema of the superficial dermis with increased pigmentation of the basal layers of epidermis.

The couple had two children, 3½ years (female) and 5 years old (male). Both had normal clinical profile except for mild pedal oedema of two weeks duration. Their fundus examination was normal. Routine haemogram, renal function tests and urine examination were normal.

Mustard oil being consumed by the family was tested and was positive for argemone oil.

Both husband and wife were given diuretics and antioxidants for a period of 2 weeks. The children were given diuretics only. The family remained in our followup for a period of three months although all the patients improved after one month.

Discussion

Argemone poisoning, commonly known as epidemic dropsy occurs in outbreaks. In India, most cases have been reported from Bengal, Bihar, Orissa, Madhya Pradesh and Uttar Pradesh. The disease was first reported in 1877 from Bengal. Since then, numerous epidemics have been documented, worst ones being in 1934 (2) and 1998. In both epidemics, more than two thousand

people were involved. Outbreak of this disease, in J&K State, was first reported from Jammu in September 1998 (3). Retinal haemorrhages were not seen in any of the patients in the series reported from Jammu but has been observed in one of our patients. The outbreak in 1998 was alarming because of the fact that no state of North India remained untouched. Adulteration of edible oils with the toxin is generally unintentional as the disease occurs in outbreaks despite common growth of the Mexican poppy plant and easy availability of its seeds. The argemone oil is pungent just like mustard oil and this makes detection of adulteration impossible without the aid of laboratory support. Contamination of linseed oil, ghee and wheat flour can also cause the disease (1,4). In our cases, contaminated mustard oil was being consumed. Body massage with the adulterated oil can also cause the disease (5). A minimum concentration of 1% of argemone oil in mustard oil is sufficient to produce epidemic dropsy (6). The disease usually occurs in families coming from low socio-economic strata (7). The persons consuming protein-rich diet manifest a mild form of the disease. Incidence of the disease is usually highest in the months of July-August and lowest in April. It has been suggested that the toxicity of the oil is reduced on storage. Breast fed babies and children under two years of age do not suffer from dropsy (8). Sanguinarine and dihydro-sanguinarine, the toxins present in the argemone oil, cause the disease (9). These are thought to affect the carbohydrate metabolism, raising the levels of pyruvic acid (10). This has effects ranging from inhibition of $\text{Na}^+ \text{K}^+$ ATPase, DNA damage, decreased GSH levels to increased lipid peroxidation (10). Free radicals are generated and widespread increased capillary permeability and vasodilatation occurs (10). These changes are responsible for ocular and other clinical manifestations of the disease. Hypoproteinemic states

increase the severity of the disease. Though the disease occurs in epidemics, isolated cases are also reported. Onset of the disease may be acute or subacute. Commonest finding is the bilateral pedal oedema as has been observed in all of our cases. The swollen legs may be erythematous and tender and two of our patients had this finding (11). Pleural and pericardial effusion and ascites may occur. Nausea, vomiting and diarrhoea also occur though none of our cases complained of these symptoms. Low-grade fever as was observed in 2 of our cases is also described (8). Cardiac and renal failure determine the prognosis. Hepatomegaly may occur (11). Mild derangement of liver functions may be there. Two of our cases had hepatomegaly and liver function tests were normal in all the cases. Ocular manifestations include retinal venous dilatation, haemorrhages and papilloedema (12) Five to fifteen percent of cases develop glaucoma and may require urgent management to prevent blindness (2). One of our patients had haemorrhages in the retina. Bleeding tendencies and peripheral neuritis are also known. Cause of death is cardiac failure in most cases.

Diagnosis of the disease requires a high degree of clinical suspicion. The edible oil is tested for the presence of argemone oil. Confirmatory test is detection of sanguinarine in serum or urine. Nephrotic syndrome, congestive cardiac failure, myxoedema, anaemia, hypoproteinemia and beri-beri are conditions considered in differential diagnosis. Treatment is largely symptomatic. Antioxidants and steroids are beneficial (13,14). Aspirin, indomethacin, ephedrine and clonidine are used for glaucoma. Some cases of glaucoma may require surgical management.

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