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Case Report

EPIDEMIC DROPSY OUTBREAK IN LOCALIZED COMMUNITY OF HARYANA, INDIA

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ABSTRACT

The outbreak of epidemic dropsy in the Indian capital, New Delhi, during the rainy season of 1998 was of one of the most severe forms and had repercussions in both health and political circles. Some 2552 cases were reported and 65 deaths occurred between 5 August and 12 October, causing untold misery and economic loss to the affected families. The actual figures are likely to be much higher due to non-reporting of milder cases to the hospitals. The aim of this article is to consolidate and update the available information on clinical aspects of epidemic dropsy.

KEYWORDS: Epidemic, *Argemone* oil

Epidemic dropsy results from the accidental ingestion of mustard oil adulterated with oil extracted from the seeds of *Argemone Mexicana* (Sarkar, 1926). The main alkaloid of *Argemone* oil is toxic, and belongs to the class of benzophenanthridine group, namely sanguinarine and dihydro-sanguinarine. Lyon reported the first four cases of argemone oil poisoning from Bombay in 1877 (Das and Khanna, 1997) (Sachdev *et al.*, 1989). Various epidemics have been reported, in India from Calcutta (1877), Assam, Bihar, Eastern U.P, Orissa, Madhya Pradesh, Gujrat and Delhi since then (Das and Khanna, 1997) (Smith, 1937). Epidemic Dropsy is an acute non-infectious disease characterized by pitting edema of the extremities, especially of the lower limbs; cutaneous erythema; and local tenderness (Singh *et al.*, 1999) (Kumar *et al.*, 1992). Glaucoma and other visual field defects, leading to blindness, and cardiac and respiratory problems, leading to death are among the most severe manifestations of the condition (Rathore, 1982) (Singh *et al.*, 2006) (Gupta, 2009) (Park, 2009) (Das and Khanna, 1998).

MATERIALS AND METHODS

All members of the concerned family were interviewed and those requiring medical attention were hospitalized. A survey of the surrounding area was conducted to identify similar cases in the vicinity, if any. A thorough personal and medical history, history of present illness, and clinical features of each patient was

noted. Blood was collected for hemoglobin, total leukocyte, and differential leukocyte counts and urine samples were collected for urine examination. Written informed consent before data collection was obtained from all the study participants.

Mustard oil samples were collected from the affected family, six neighboring households, and from the shop where the family got mustard oil extraction done. The oil samples were tested for contamination of argemone oil using the Nitric acid test, Cupric Acid test and Ferric chloride test.

1. Nitric Acid Test: Took 5ml of adulterated mustard oil in a test tube. To this we added 5ml of concentrated nitric acid and the test tube was shaken. Orange yellow colour was developed in the acid layer confirming the presence of argemone oil in the sample.
2. Cupric Acid Test: Took 5ml of adulterated oil in a test tube. To this 1ml of glacial acetic acid and 2 mg of cupric acetate solution was added. Green colour confirmed the presence of argemone oil in the sample.
3. Ferric Chloride test: Took 5ml of adulterated oil sample in a test tube. To this we added 2ml of concentrated HCl of rectified spirit. It was shaken for 1 minute. The acid and oil layers were cleared separately.

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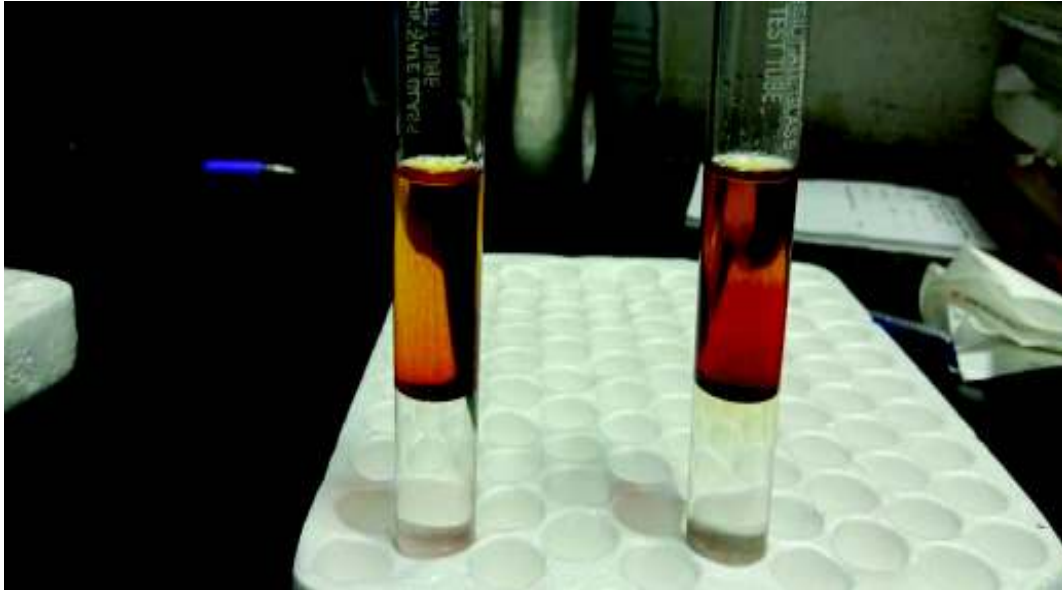


Figure 1: Orange yellow colour was developed in the acid layer confirming the presence of argemone oil in the sample

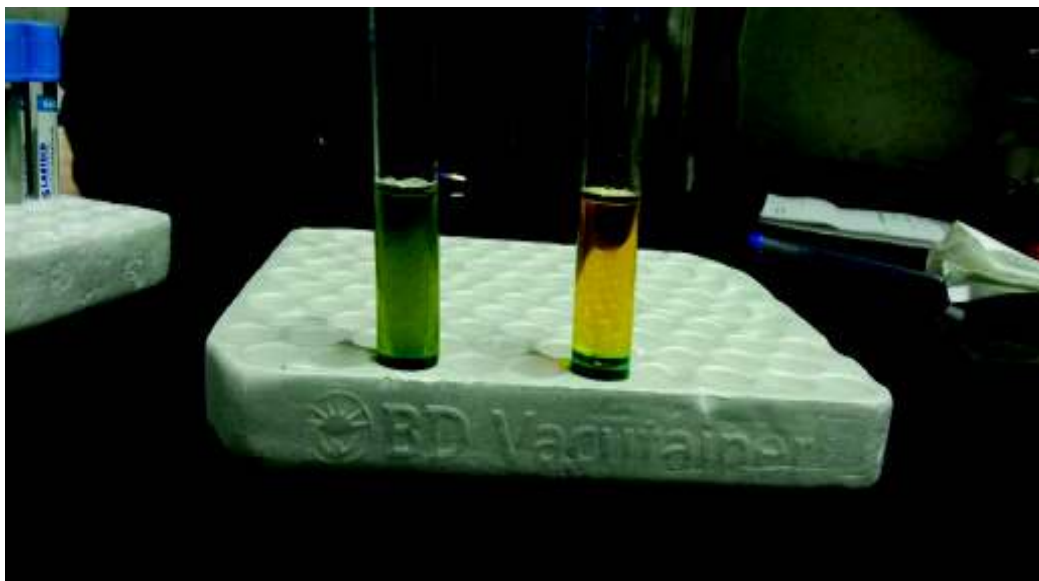


Figure 2: Green colour confirmed the presence of argemone oil in the sample

CASE REPORT

A 41-year old man presented to the emergency services with a month history of a burning sensation, swelling and redness over the feet. He also complained of a recent-onset diarrhea which was not associated with fever, tenesmus, abdominal pain, distension or vomiting.

The patient was cooperative and well oriented to time, place and person. Vital signs were abnormal, with the pulse rate at 118 beats per minute and respiratory rate

at 26 breaths per minute, and a blood pressure of 141/98 mmHg. The patient had no jaundice, cyanosis, clubbing or lymphadenopathy was detected. Examination of the lower limbs revealed pitting edema up to the lower abdominal wall, and tenderness in the same distribution. Abdominal and neurological examinations were normal and on auscultation of chest normal S_1 and S_2 were heard with no added sounds. The results of his laboratory investigations are summarized in the table.

Table 1: Results of laboratory investigations of the index case

Investigation	Results	Units	Bio. Ref. Level
Hemoglobin	12.90	g/dl	13.00-17.00
Total leukocyte count	8.00	Thou/mm ³	4.00-10.00
Platelet count	264.0	Thou/mm ³	150.00-450.00
MCV	71.00	fL	80.00-100.00
MCH	22.40	Pg	27.00-32.00
MCHC	31.60	g/dl	32.00-35.00
Red Cell Distribution Width	14.80	%	11.50-14.50
HbA1c	5.4	4.0-5.6	%
Blood sugar	74.0	mg/dl	Up to 160 mg/dl
Blood urea	25.0	mg/dl	15-45
Serum creatinine	0.55	mg/dl	0.4-1.3 (male) 0.4-1.2 (female)
Total Cholesterol	192.00	mg/dl	<200.00
Triglycerides	143.00	mg/dl	<150.00
HDL Cholesterol	44.00	mg/dl	>40.00
LDL Cholesterol	119.40	mg/dl	<100.00
VLDL Cholesterol	28.60	mg/dl	<130
T ₃ Total	0.82	ng/ml	0.70-2.04
T ₄ Total	8.58	ug/dl	5.74-13.03
TSH	1.70	uIU/ml	0.34-5.60
24 hour Urine Protein	262.00	mg/24 hr	<140
Albumin	1.7	g/dl	6.40-8.30
A:G ratio	4.50	g/dl	3.50-5.20
Total Protein	6.5		0.90-2.00
AST	20	U/L	<50
ALT	24	U/L	<50
Sodium	142.0	mEq/dl	136.00-146.00
Potassium	4.20	mEq/dl	3.50-5.10
Calcium	9.60	mg/dl	8.80-10.60
Phosphorus	3.00	mg/dl	2.40-4.40
Bilirubin total	1.00	mg/dl	0.30-1.20
Bilirubin Direct	0.20	mg/dl	<0.20
Bilirubin Indirect	0.80	mg/dl	<1.10

On further medical history, patient admitted that several other members in his neighborhood have developed similar painful leg edema recently. During the epidemiological investigation, it was found that all these families were using mustard oil for cooking, which had been extracted from same local oil extraction machine. The mustard oil samples that were being used for cooking by the affected families tested positive for *Argemone* oil contamination. The presence of *Argemone* oil contamination was confirmed using nitric acid, cupric acid and ferric chloride test.

Our index case was managed with bed rest with leg elevation and a protein rich diet. Supplements of

calcium, antioxidants like vitamin C and E, thiamine and other B vitamins were given. To relieve oedema patient was started on 20 mg furosemide OD. The oedema resolved in a week and the patient was discharged and is doing fine now.

DISCUSSION

Epidemic dropsy results from intoxication with argemone oil resulting from its willful adulteration with mustard oil, which is possible by virtue of their similar color (Mukerjee *et al.*, 1941). In the most recent Indian epidemic in 1998, the first case was reported on Aug. 5, 1998 and the epidemic lasted for seven weeks. 2178 cases

were reported out of which 66 (3.2%) died. This was possibly the largest epidemic in India. The 'Food Safety and Standards of India' (FSSI) have laid down norms for marketing and sale of refined edible vegetable oils in India. According to FSSI, mustard oil for sale must be free of *Argemone* oil and selling mustard oil contaminated with *Argemone* oil is liable to be punished by law. These legislative measures have resulted in a change in the epidemiology of the disease. Epidemic dropsy now affects families involved in cultivating, harvesting, and consuming mustard oil that possibly get contaminated with *Argemone* oil during the harvesting of mustard.

Mukerjee *et al.* (1941) had isolated a toxic substance from argemone seeds which was later identified as sanguinarine by Sarkar and Rahman, 1945. Argemone seeds contain 2 physiologically active alkaloids; dihydrosanguinarine (~ 87%) and sanguinarine (~ 5%) Sarkar (1948). The fact that argemone oil is responsible for epidemic dropsy has been established by several human feeding trials with argemone oil and argemone adulterated mustard oil (Chopra *et al.*, 1939). These alkaloids have a direct toxic effect on cells. Possible mechanisms are inhibition of Na-K ATPase activity, reduction of ATP production due to reduced glycogen levels, raised Interleukins 1 and 6, elevated TNF- α level, and an increased production of reactive oxygen and nitrogen species which lead to DNA damage and apoptosis (Das *et al.*, 2005). All these mechanisms cause endothelial damage, increased capillary permeability, leakage of protein rich fluid into the interstitial spaces, and vascular damage (Babu *et al.*, 2010).

Review of literature suggests that pitting edema is the main clinical feature in patients with epidemic dropsy, and this finding was corroborated by the results of our study. Other cutaneous findings such as erythema and skin tenderness are the features which were variably reported in previous studies (35-82% and 22-88%, respectively) (Tandon *et al.*, 1975) (Gomber *et al.*, 1994) (Singh *et al.*, 2000) (Singh *et al.*, 1998). Other skin changes such as widespread telangiectasis, erythrocyanosis, mottling and blanching were not seen in our case. Our patient didn't developed any cardiological or neurological complication. Anemia is a common feature of epidemic dropsy was seen in our case too (Rangan and Barceloux, 2009).

Withdrawal of the offending agent - the contaminated mustard oil - is the cornerstone of management, being vital to limit the progression of symptoms. A review of the literature based on animal

experiments in rats has elaborated upon the use of a high protein diet and vitamin supplementation to replenish the depleted antioxidant levels, which are mainly riboflavin, tocopherol and retinol. We followed the aforementioned recommendations for our patient along with diuretic to relieve edema.

CONCLUSION

Although epidemic dropsy still exists in India, its mode of presentation has changed from an epidemic to a sporadic pattern. This poisoning should be suspected in patients presenting with edema, erythema, tenderness of the limbs, and a collection of these symptoms within farmer families. Epidemic dropsy can be confirmed by a laboratory exam of contamination of mustard oil with *Argemone* oil. Withdrawal of the contaminated cooking oil is the most important initial step. Bed rest with leg elevation and a protein-rich diet are useful. Supplements of calcium, antioxidants (vitamin C and E), and thiamine and other B vitamins are commonly used. Diuretics are used universally but caution must be exercised not to deplete the intravascular volume unless features of frank congestive cardiac failure are present, as oedema is mainly due to increased capillary permeability. Effective strategies to eradicate this poisoning involve public education, mandatory quality inspections, and detection of possible contaminations.

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