



A Familial Cluster of Epidemic Dropsy from Argemone-Adulterated Mustard Oil: A Confirmed Case Report and Review of a Re-emerging, Preventable Food-borne Toxidrome

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Abstract

Background: Epidemic Dropsy is a preventable, food-borne toxidrome caused by consumption of edible mustard oil adulterated with oil of Argemone mexicana (prickly poppy). The toxic benzophenanthridine alkaloids sanguinarine and dihydrosanguinarine produce widespread capillary dilatation and increased permeability, resulting in non-inflammatory bilateral pitting oedema, cutaneous erythema, gastrointestinal disturbance, and, in severe cases, congestive cardiac failure, glaucoma and death. Familial clustering is characteristic because the whole household shares the same adulterated oil.

Case presentation: We report a laboratory-confirmed household cluster of Epidemic Dropsy in a family from Ujhani, Budaun district, Uttar Pradesh, who cooked exclusively with self-harvested, home-pressed mustard oil. A 35-year-old woman (index case) presented to the Department of Nephrology with progressive bilateral lower-limb swelling that ascended to the face over 3-4 days, together with diffuse abdominal pain, anorexia, nausea and hard-stool-related rectal bleeding. Her husband, parents-in-law and two of her three children had similar bilateral pedal oedema ascending upwards; strikingly, the third child - who habitually ate at a relative's house - was spared, providing a natural exposure-response observation. On examination the index case had pallor, facial puffiness and non-tender reddish cutaneous lesions, and per-rectal examination revealed multiple anal fissures accounting for the bleeding. Investigations showed severe microcytic hypochromic anaemia with severe thrombocytopenia (haemoglobin 6.8 g/dL; platelets $0.36 \times 10^5/\text{microL}$), hypoalbuminaemia (2.8 g/dL), prolonged prothrombin time (23.4 s; INR 1.72), Azotemia (creatinine 3.8 mg/dL improving to 2.4) with hypokalaemia (potassium 3.6 falling to 2.1 mmol/L) and a low iron/low-TIBC profile. Ultrasonography showed hepatomegaly, bilateral pleural effusion, mild ascites and abdominal-wall subcutaneous oedema; echocardiography showed a dilated right heart with moderate tricuspid regurgitation and moderate pulmonary arterial hypertension with preserved left-ventricular systolic function. Ophthalmological evaluation, including dilated funduscopy and tonometry (intraocular pressure 13/12 mmHg), showed no evidence of Argemone-related ocular toxicity or glaucoma. Argemone-oil adulteration of the household oil was confirmed by positive bedside nitric-acid and ferric-chloride tests and also by paper chromatography at an independent ISO-accredited laboratory. The patient was managed conservatively with diuretics, supportive care and, critically, withdrawal of the adulterated oil, with improvement in renal function.

Conclusion: A characteristic clinical syndrome, a shared dietary exposure - underscored by the sparing of the one child who ate elsewhere - and confirmatory oil analysis established the diagnosis without invasive testing.

Clinicians in mustard-oil-consuming regions should consider Epidemic Dropsy in any patient or family presenting with acute bilateral non-inflammatory oedema, secure the suspected oil for analysis, screen co-exposed household members, and notify food-safety authorities, because the condition is entirely preventable through enforcement of food-adulteration law.

Keywords: Epidemic Dropsy; Argemone mexicana; mustard oil; sanguinarine; food adulteration; pitting oedema.

Introduction

Epidemic Dropsy is a clinical state resulting from the use of edible oils adulterated with the oil of Argemone mexicana (Mexican prickly poppy). The Argemone seed closely resembles the black mustard seed, and its oil is a cheap adulterant of mustard oil in the Indian subcontinent; contamination may be accidental (the two crops grow together and seeds are harvested and pressed simultaneously) or deliberate. Two benzophenanthridine alkaloids, sanguinarine and dihydrosanguinarine, are chiefly responsible for toxicity: they cause widespread capillary dilatation, proliferation and increased permeability, allowing protein-rich plasma to leak into the interstitium and produce the characteristic non-inflammatory oedema. The resulting relative hypovolaemia stimulates renal salt and water retention, further aggravating fluid overload.

Because an entire household typically cooks with the same batch of oil, cases characteristically occur in familial and community clusters, and outbreaks have been documented repeatedly across the Indo-Gangetic plain and in Bengal, Bihar, Assam, Delhi, Gujarat and elsewhere, as well as historically in Mauritius, Fiji and South Africa. Illness begins with gastrointestinal symptoms followed by cutaneous erythema and pigmentation and bilateral pitting oedema of the lower limbs; respiratory symptoms and right-sided congestive cardiac failure, glaucoma with visual loss, retinal haemorrhage, anaemia, hypoproteinaemia and renal Azotemia may follow. There is no specific antidote; management rests on immediate withdrawal of the adulterated oil, supportive care and antioxidants.

Despite statutory prohibition under India's food-safety legislation, sporadic outbreaks continue, and the entity is under-recognised by a generation of clinicians who have not encountered it. We describe a laboratory-confirmed familial cluster in which a characteristic clinical picture, a common dietary source and

confirmatory oil analysis (both bedside and by an independent accredited laboratory) secured the diagnosis. The report is presented in accordance with the CARE case-report guideline.

Case Presentation

Index case - history

A 35-year-old woman from Ujhani, Budaun district, Uttar Pradesh, presented to the Department of Nephrology with approximately one month of generalised body swelling, twenty days of diffuse abdominal pain, ten to fifteen days of anorexia and nausea, and ten to fifteen days of blood in the stool.

She had been well until about one month before presentation, when she developed swelling that began in both feet and progressed to involve the face within three to four days. The oedema was pitting in type, associated with reddish discolouration of the overlying skin, without raised local temperature, itching or tenderness; there was no decrease in urine output and no burning micturition. The abdominal pain was diffuse and intermittent (off and on) over twenty days, non-radiating in nature and without any special character associated with reduced appetite and nausea, and was not associated with fever or burning micturition. She also reported blood in the stool for ten to fifteen days, associated with hard-consistency stool and without blackish discolouration. There was no history of hypertension, diabetes mellitus, thyroid disease or tuberculosis, and no history of any drug intake or addiction.

Dietary and family history: The family cooked exclusively with mustard oil that they pressed themselves from self-harvested mustard seeds. Multiple co-resident household members were similarly affected (Table 1): her husband, her parents-in-law and two of her three children had developed bilateral pedal oedema that was progressing upwards.

Notably, the third child was spared; this child habitually took most meals at a relative's (uncle's) house and therefore had little exposure to the household oil. This natural exposure-response pattern - illness confined to those eating food cooked in the shared oil and sparing of the one member who ate elsewhere - was the decisive epidemiological clue pointing to a food-borne toxidrome.

Examination

The index case was conscious and oriented and had pallor. There was facial puffiness and bilateral, symmetrical, non-tender pitting oedema of the lower limbs extending to the dorsa of the feet, with oedema of the dorsa of the hands. The skin showed reddish, non-tender cutaneous lesions with normal local temperature, together with erythematous mottling and scattered telangiectasia over the trunk, neck and upper chest, consistent with the dermal capillary changes of Argemone toxicity. There was no icterus and no lymphadenopathy. The abdomen was soft with hepatomegaly and no localised tenderness, and systemic examination was consistent with a volume-overloaded state with features of right-heart strain. Per-rectal examination revealed multiple anal fissures, accounting for the passage of fresh blood with hard stool. The affected male household member similarly demonstrated facial puffiness and bilateral pitting pedal oedema.

Investigations

Haematological evaluation showed severe anaemia with haemoglobin ranging 6.4-6.8 g/dL and a predominantly microcytic, hypochromic film with moderate anisocytosis and occasional pencil cells; the platelet count was low ($0.30-0.36 \times 10^5/\text{microL}$; approx. 30,000-36,000/microL) with severely reduced platelets and occasional giant forms on smear, and there was a relative lymphopenia. The iron profile (serum iron 22 microg/dL; total iron-binding capacity 198 microg/dL) was in keeping with an anaemia of chronic disease/inflammation superimposed on nutritional deficiency. The prothrombin time was prolonged (23.4 s; INR 1.72). Biochemistry showed hypoalbuminaemia (albumin 2.8 g/dL; total protein 5.9 g/dL) with normal transaminases and bilirubin, mildly elevated C-reactive protein, and borderline-low corrected calcium. Renal function at presentation showed Azotemia (urea 132 mg/dL, creatinine 3.8 mg/dL) that improved over the subsequent days

(creatinine to 2.4 mg/dL); serum sodium was 134 mmol/L and serum potassium fell from 3.6 to 2.1 mmol/L (hypokalaemia). Random plasma glucose was normal. Key results are summarised in Table 2.

Urinalysis showed trace proteinuria with a few pus cells and fungal spores/hyphae (attributable to sampling/colonisation), and stool examination showed a negative occult-blood test with no ova, cysts or parasites; the fresh rectal bleeding was clinically attributable to the anal fissures found on per-rectal examination rather than to upper gastrointestinal loss, although the concomitant thrombocytopenia and coagulopathy would aggravate any bleeding. A targeted febrile-illness and infective screen was negative, including *Leptospira* IgM, *Orientia tsutsugamushi* (scrub typhus) IgG, and HIV, hepatitis C and hepatitis B surface antigen serology, making these mimics unlikely.

Ultrasonography of the abdomen revealed hepatomegaly (liver span approx. 18 cm) with an oedematous gallbladder wall, bilateral pleural effusion (left greater than right), mild ascites and subcutaneous oedema of the anterior abdominal wall; the hepatic and splenic veins appeared dilated on review while the inferior vena cava was normal in calibre. Incidental adnexal simple cysts were noted.

Two-dimensional echocardiography demonstrated a dilated right atrium and right ventricle with moderate tricuspid regurgitation and moderate pulmonary arterial hypertension, mild mitral regurgitation, preserved left-ventricular systolic function (ejection fraction approx. 60%) and grade I left-ventricular diastolic dysfunction, without regional wall-motion abnormality or pericardial effusion.

This combination of serosal effusions, hepatic congestion and right-heart pressure/volume loading with preserved left-ventricular function is characteristic of the high-permeability, high-output circulatory state of Epidemic Dropsy rather than of primary cardiac or renal disease.

Ophthalmological evaluation

Because glaucoma and retinal haemorrhage are recognised and sight-threatening complications of Argemone toxicity, a formal ophthalmology consultation was obtained. Anterior-segment examination of both eyes was unremarkable (clear cornea, normal anterior-chamber depth, normally

reacting pupils, clear lens) with full and free extraocular movements. Intraocular pressure was normal (13 mmHg right eye, 12 mmHg left eye). Dilated fundus examination of both eyes showed a healthy, well-defined optic disc with a cup-to-disc ratio of 0.3:1, normal vessels (arteriovenous ratio 2:3), a clear media and no retinal haemorrhage, with only a mildly dull foveal reflex. Autorefractometry revealed a small refractive error only. The documented impression was that the bilateral dilated fundus examination was not suggestive of sanguinarine (Argemone) toxicity and was within normal limits, and the patient was advised review in the eye outpatient department for refraction. This important negative finding indicates that, in this patient, the illness was caught before ocular involvement supervened.

Dermatological evaluation

The cutaneous findings comprised reddish, non-tender skin lesions with erythematous mottling and telangiectasia over the trunk, neck and upper chest, and normal local temperature - changes attributable to the capillary dilatation and increased permeability characteristic of Argemone toxicity, and distinct from an inflammatory or infective dermatosis.

Confirmation of oil adulteration

A sample of the household mustard oil was tested. Qualitative bedside screening was positive: the nitric-acid test produced the characteristic orange-red to brown colour change, and the ferric-chloride test - performed alongside labelled controls - was also positive. Definitive confirmation was obtained from an independent ISO-accredited analytical laboratory (Opal Research & Analytical Services; test report ORAS/CT/452026472), which detected Argemone oil in the mustard-oil sample using a paper-chromatographic method. The convergence of a compatible clinical syndrome, a shared dietary exposure across multiple household members with sparing of the one unexposed child, and positive oil analysis by two independent methods firmly established the diagnosis of Epidemic Dropsy.

Management and outcome

There is no specific antidote for Argemone toxicity, and management is supportive with, above all, immediate and complete withdrawal of the adulterated oil from the household. The index case was managed

conservatively. The single most important intervention was cessation of the use of the adulterated self-pressed mustard oil, and the family was counselled to discard the implicated oil entirely. Supportive treatment comprised diuretics for the fluid-overload and congestion, correction of hypokalaemia and other electrolyte disturbances, and general supportive medications, together with monitoring of renal, hepatic and cardiac status. A high-protein diet with multivitamin and antioxidant supplementation is advocated on the rationale that Argemone toxicity is mediated by oxidative stress and depletion of plasma antioxidants. Following withdrawal of the oil and supportive care, the renal dysfunction improved (serum creatinine fell from 3.8 to 2.4 mg/dL), and ophthalmological assessment excluded ocular toxicity.

Discussion

This familial cluster illustrates the classic, and still contemporary, presentation of Epidemic Dropsy and shows how the diagnosis can be reached confidently at the bedside in a resource-limited setting when the syndrome is recognised. The cardinal feature is bilateral, symmetrical, non-tender pitting oedema that is not explained by primary cardiac, renal, hepatic or nutritional disease; its association with cutaneous erythema and telangiectasia, gastrointestinal symptoms and a congestive, high-permeability circulatory picture is highly suggestive. In our index case, the coexistence of hypoalbuminaemia, serosal effusions, hepatic congestion and a dilated right heart with pulmonary arterial hypertension but preserved left-ventricular systolic function reflects the underlying pathophysiology of alkaloid-induced capillary dilatation and leak rather than intrinsic organ failure.

The single most important diagnostic step is a careful dietary history that identifies a shared oil source, because clustering within a household is the epidemiological signature of the disease. In this family, the illness affected every member who ate food cooked in the shared, self-pressed mustard oil - the index case, her husband, her parents-in-law and two of three children - while the one child who habitually ate at a relative's house was entirely spared. This near-experimental exposure-response pattern, with the unexposed child serving as a natural control, strongly implicated the household oil and directed us to secure

and test it; it also illustrates that self-harvested, home-pressed oil is not inherently safe, because Argemone and mustard grow and are harvested together, so accidental contamination at source is common. Bedside qualitative tests (nitric-acid and ferric-chloride tests) provide rapid, inexpensive screening for Argemone adulteration and can be performed in any hospital laboratory; positive bedside tests should nevertheless be confirmed, as here, by an accredited laboratory using chromatography and, where feasible, demonstration of sanguinarine, to satisfy both clinical and medico-legal requirements.

The mechanism of toxicity is well characterised. Sanguinarine and dihydrosanguinarine interfere with membrane $\text{Na}^+/\text{K}^+-\text{ATPase}$, promote oxidative stress with met-haemoglobin formation and red-cell injury, and disturb carbohydrate metabolism with accumulation of pyruvate; the resulting widespread vasodilatation and increased vascular permeability produce oedema, while myocardial involvement can precipitate high-output failure. Anaemia in dropsy is multifactorial, arising from oxidative red-cell damage compounded by nutritional deficiency, as seen in our patient. Beyond the acute illness, chronic exposure to Argemone oil and sanguinarine is genotoxic and has been epidemiologically linked to the high incidence of gallbladder carcinoma in the Indo-Gangetic basin, underscoring that the public-health stakes extend well beyond the outbreak itself.

The differential diagnosis of acute bilateral oedema - Congestive Cardiac Failure, Nephrotic Syndrome, Cirrhosis of liver, Protein-Energy Malnutrition, Hypothyroidism, wet Beriberi, Filariasis and others - was addressed through the clinical pattern, targeted investigations and, decisively, the dietary and family history plus oil analysis. The negative infective serology and the preserved left-ventricular function argued against the principal mimics. It is worth emphasising that the diagnosis is fundamentally clinical and epidemiological: no single laboratory value is pathognomonic, and it is the constellation - syndrome, cluster and confirmed adulterant - that establishes it.

Two management principles deserve emphasis. First, removal of the offending oil is both the most effective treatment and the pivot of prevention; continued exposure worsens outcome, and the illness is entirely preventable. Second, because glaucoma and cardiac

decompensation are the feared complications, every patient warrants ophthalmological assessment with intraocular-pressure measurement and cardiac evaluation; in our index case the dilated fundus examination and tonometry were normal and specifically not suggestive of sanguinarine toxicity, documenting that the disease had been recognised before ocular involvement and providing a reassuring, objectively recorded baseline. It is also worth noting that the rectal bleeding in our patient arose from anal fissures related to hard stool rather than from a primary toxic enteropathy, a reminder that not every symptom in a dropsy patient is directly alkaloid-mediated and that alternative, treatable explanations should still be sought. At the community level, a single confirmed case should trigger active case-finding among household and neighbourhood contacts sharing the oil, seizure and testing of the implicated oil, and prompt notification of food-safety authorities under the Food Safety and Standards framework, so that the source can be traced and further cases averted. Reported case-fatality in severe hospitalised series has been of the order of 10%, chiefly from cardiogenic and septic shock, which reinforces the value of early recognition.

Strengths

The strengths of this report are the documented familial clustering, the completeness of multisystem characterisation, and - unusually - dual confirmation of adulteration by bedside tests and an independent accredited laboratory.

Limitations

Limitations include the descriptive nature of a single-family report, incomplete individual work-up of the co-affected members (who were identified and screened clinically), and the absence of a quantitative sanguinarine assay; formal ophthalmological and longer-term follow-up data should be added to complete the natural-history picture.

Conclusion

Epidemic Dropsy remains a real, preventable and re-emerging hazard wherever mustard oil is consumed. A family presenting together with acute bilateral non-inflammatory pitting oedema, cutaneous erythema and a congestive circulatory picture should prompt a focused dietary history, examination of co-exposed household members, and immediate testing of the suspected cooking oil. Bedside nitric-acid and ferric-

chloride tests, confirmed by an accredited laboratory, allow rapid and definitive diagnosis without invasive investigation. Withdrawal of the adulterated oil, supportive care, screening for glaucoma and cardiac complications, and mandatory notification to food-safety authorities are the essentials of care. Sustained enforcement of food-adulteration law and public education remain the only durable means of prevention.

Declarations

Patient consent: Written informed consent for the use and publication of clinical photographs for educational and research purposes was obtained from the index patient and her next of kin (husband), and from the affected family members (with parental consent for minors). Consent was documented on the institution's standard consent form (Rohilkhand Medical College & Hospital, consent form no. RMCH-378/06/2026/50), signed by the patient's relative with a thumb impression and countersigned by the treating physicians; the signed form is retained on file and is available to the editor on request. Because the consent form contains patient identifiers, it is not reproduced here; all facial images are presented with identity-masking (eye bars) in accordance with journal policy.

Ethics: Institutional ethics-committee approval/exemption for this single-family case report was obtained as per institutional policy

Authors' contributions:

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Figure legends

Figure 1. Facial puffiness / periorbital and facial oedema in affected household members.

Figure 2. Bilateral, symmetrical non-tender pitting oedema of the lower limbs extending to the dorsa of the feet.

Figure 3. Cutaneous changes over the trunk: erythematous mottling with scattered telangiectasia and small vascular lesions.

Figure 4. Bedside qualitative tests on the household mustard-oil sample: (a) nitric-acid test showing the

characteristic orange-red/brown colour change; (b) ferric-chloride test with labelled controls.

Figure 5. Independent ISO-accredited laboratory report (Opal Research & Analytical Services, report no. ORAS/CT/452026472) confirming detection of Argemone oil in the household mustard-oil sample by the paper-chromatographic method.

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Tables

Table 1. The household cluster: exposure to the shared self-pressed mustard oil and clinical status (a natural exposure-response pattern).

Household member	Ate food cooked in the shared oil	Clinical status
Index case (35 y, Female)	Yes	Affected - generalised pitting oedema (feet ascending to face); reddish cutaneous lesions; pallor; anaemia and thrombocytopenia; anal fissures
Husband (39 y, Male)	Yes	Affected - bilateral pedal oedema, progressing upwards
Parents-in-law	Yes	Affected - bilateral pedal oedema
Two of three children	Yes	Affected - bilateral pedal oedema, progressing upwards
Third child	No (ate mainly at a relative's house)	Spared - asymptomatic

All affected members shared food cooked in the household's self-harvested, home-pressed mustard oil; the single unexposed child was spared, supporting the dietary aetiology.

Table 2. Key investigations in the index case.

Parameter	Value	Reference range
Haemoglobin	6.8 g/dL	12.0-15.0
Total leucocyte count	6,650 /microL	4,000-11,000
Platelet count	0.36 x10 ⁵ /microL (~36,000/microL)	1.5-4.1
Peripheral smear	Microcytic hypochromic; platelets severely	-

Parameter	Value	Reference range
	reduced, occasional giant forms; lymphopenia	
Serum iron / TIBC	22 / 198 microg/dL	35-145 / 250-400
Prothrombin time / INR	23.4 s / 1.72	12.6-16.2 s
Total protein / albumin	5.9 / 2.8 g/dL	6.4-8.3 / 3.5-5.2
Bilirubin (T/D); AST/ALT	0.51/0.15; 20/18	within normal
Blood urea	132 mg/dL	15-40
Serum creatinine (presentation to later)	3.8 improving to 2.4 mg/dL	0.6-1.3
Serum sodium	134 mmol/L	135-145
Serum potassium (trend)	3.6 falling to 2.1 mmol/L	3.5-5.0
C-reactive protein	Mildly elevated	0-0.5 mg/dL
Random plasma glucose	110 mg/dL	70-150
24 Hour urinary Protein	540 mg/dl	< 150 mg/dl
Stool for Ova/Cysts/Eggs/ RBC/ Occult Blood	Negative	Negative
Infective serology - Leptospira IgM, scrub-typhus IgG, HIV, HCV, HBsAg	all negative	Negative

TIBC, total iron-binding capacity; T/D, total/direct; AST/ALT, aspartate/alanine aminotransferase.

Table 3. Imaging findings in the index case.

Modality	Findings
Ultrasound abdomen	Hepatomegaly (approx. 18 cm); oedematous gallbladder wall; bilateral pleural effusion (left>right); mild ascites; anterior abdominal-wall subcutaneous oedema; dilated hepatic and splenic veins with normal IVC; incidental adnexal simple cysts
2D echocardiography	Dilated RA/RV; moderate tricuspid regurgitation with moderate pulmonary arterial hypertension; mild mitral regurgitation; preserved LV systolic function (EF approx. 60%); grade I LV diastolic dysfunction; no RWMA; no pericardial effusion

IVC, inferior vena cava; RA/RV, right atrium/ventricle; LV, left ventricle; EF, ejection fraction; RWMA, regional wall-motion abnormality

Figures

Identifiable faces have been masked; written consent for publication was obtained

Figure 1. Facial and periorbital puffiness in affected household members: (a) adult male; (b) the index case. Identifying features are masked.



Figure 2. Bilateral, symmetrical, non-tender pitting oedema of the lower limbs extending to the dorsa of the feet: (a) adult male; (b) the index case.



Figure 3. Cutaneous changes: (a) erythematous/haemorrhagic mottling over the trunk; (b) telangiectatic and vascular changes over the neck and upper chest.

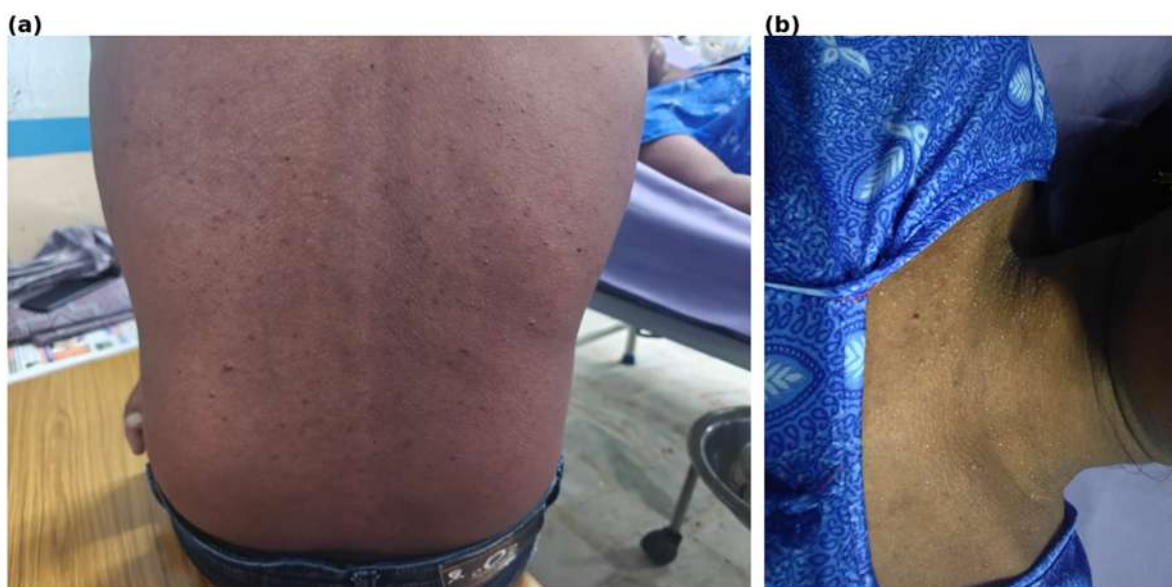


Figure 4. Bedside qualitative tests on the household mustard-oil sample: (a) nitric-acid test showing the characteristic orange-red/brown colour change; (b) ferric-chloride test.

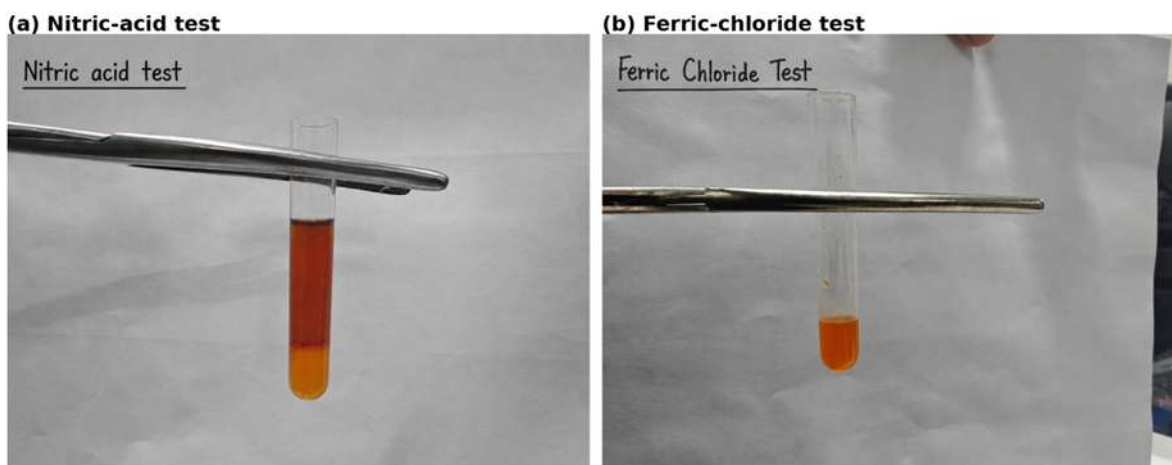


Figure 5. Independent ISO-accredited laboratory report (Opal Research & Analytical Services, report no. ORAS/CT/452026472) confirming detection of Argemone oil in the household mustard-oil sample by the paper-chromatographic method.



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Test Report

Report No.: ORAS/CT/452026472	Date : 02/07/2026
Issued to: NANHI DEVI BAREILLY	Your Reference : E - Mail
	Date of Receipt : 01/07/2026
	Our Reference : opalresearch123@gmail.com
	Test Started on : 01/07/2026
	Test Completed on : 02/07/2026
Sample Description : One Sample Received As " Mustard Oil"	

Adulteration Analysis of " Mustard Oil"

S. No.	Parameter	Test Result	Protocol
1	Argemone Oil	Detected	Paper Chromatographic Method



Auth Signatory
Gautam Budh Nagar

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