BOHR International Journal of General and Internal Medicine
2025, Vol. 4, No. 1, pp. 31–35
DOI: 10.54646/bijgim.2025.28
www.bohrpub.com

CASE REPORT

A rare case of hematohidrosis, hemolacria and epistaxis in an 11-year-old girl

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Received: 18 February 2025; **Accepted:** 09 March 2025; **Published:** 12 September 2025

Background: Hematohidrosis is an unusual anomaly in which intact skin allows the excretion of blood, with its origin mostly placed on emotional or physical stress. The pathophysiology of hematohidrosis remains mostly unknown. Autonomic dysfunction, microvascular fragility, and capillary rupture are all proposed hypotheses explaining the mechanism of this disorder. This report describes an 11-year-old girl with simultaneous hematohidrosis, hemolacria, and epistaxis, thus contributing to its knowledge and diagnosis together with differential considerations.

Methodology: A retrospective case documentation is done with due permission and informed consent. Clinical details: these were obtained through history-taking and clinical examination. Laboratory workup including complete blood count, coagulation profile, and factor assays. Liver function tests were performed, and a computed tomography (CT) of the brain was done to exclude intracranial pathology. Slit-lamp examination was done for ocular involvement. No therapeutic or follow-up was done—this report serves as a clinical discussion.

Results: The patient having irregular intermittent episodes of hematohidrosis, hemolacria, and epistaxis, all hematological, biochemical, and radiological workups were normal with the only exception of mild ethmoid sinusitis and over-pneumatization of the mastoid air cells on CT scan.

Conclusion: This case shows diagnostic difficulties because of the absence of systemic abnormalities. Further studies should be performed to identify these biomarkers, neurohormonal influences, and potential treatment options, combining expertise from dermatology, neurology, and psychoneuroimmunology to achieve a clinical objective.

Keywords: hematohidrosis, hemolacria, pediatric, case report, diagnosis, management

Introduction

Hematohidrosis, with a synonym of hematidrosis, is an exceptionally rare condition in which blood is sometimes excreted through the skin, in particular, through the eccrine sweat glands (1). It is considered that the pathogenesis of hematohidrosis stems from an increased activity of the sympathetic nervous system towards extreme vasospasm and subsequent rupture of the capillary blood vessels surrounding the sweat glands (2). Its induction is most probably through acute physical or emotional stress, though other possible contributory factors are systemic infections, coagulopathies, and autonomic dysfunction (3). Due to its rare and underreported nature, epidemiological

information concerning hematohidrosis has been sparse (4). Case reports would, however, cite that predominantly children and young adults have been most affected, with females at a higher incidence (5). A review of 42 cases found nearly 85% of the patients reported to be below 20 years, with stress being the primary triggering factor (6). Hematohidrosis can, however, present as isolated episodes of bleeding but has been associated with other such conditions as platelet disorders, systemic vasculitis, or infectious diseases like dengue (7). Hemolacria, or bloody tears, is also rare and may result from trauma, infections, tumors of the lacrimal gland, or systemic bleeding disorders; thus, most often it is idiopathic hemolacria, meaning an underlying further pathology could not be found (8). This becomes more complicated by the presence of epistaxis, as its presence would indicate either primary hematological disorders, vascular anomalies, or inflammatory conditions, such as allergic rhinitis or Wegener's granulomatosis (9).

Hematohidrosis is ill-defined from a pathophysiological standpoint. The most current theorization of this condition holds that excessive sympathetic activation produces instability among the system of microvasculature that leads to the leakage of blood through eccrine sweat glands (10). The histopathological examinations of the skin occasionally demonstrate dilated dermal capillaries with milder perivascular inflammation (11). Yet it occurs that, because of the absence of a systemic coagulopathy in the majority of cases, its actual etiology is equally obscure (12). Some, however, did state that psychological stress incited changes in hormones—especially involving catecholamines—which is believed to cause the weakened structure of the blood vessels and increased permeability (13). Despite its dramatic presentations, hematohidrosis is self-limiting, although the episodes may recur after a few months or years. The management is rather inconsistent, spanning from beta blockers (propranolol) to other alternative therapies, such as homeopathy or Ayurveda. However, there is no one set treatment protocol for the limited reported cases. The coincidence of hematohidrosis, hemolacria, and epistaxis in a child is very rare, and few documented cases can be found in literature. This report aims to enhance the information available on this condition, its diagnosis, laboratory investigations, and treatment approach. In addition, it intends to address poor awareness in its pathophysiology and management strategies.

Methodology

The case report entails retrospective documentation of an 11-year-old female patient presenting with simultaneous hematohidrosis, hemolacria, and epistaxis. The study was deemed ethical according to the Code of Ethics of

FIGURE 1 | The image illustrates blood-stained perspiration droplets on the patient's forehead and around the eyes, as well as blood-tinged tears that were observed when the eyelids were gently moved. This was recorded during an episode of hemolacria and hematohidrosis.

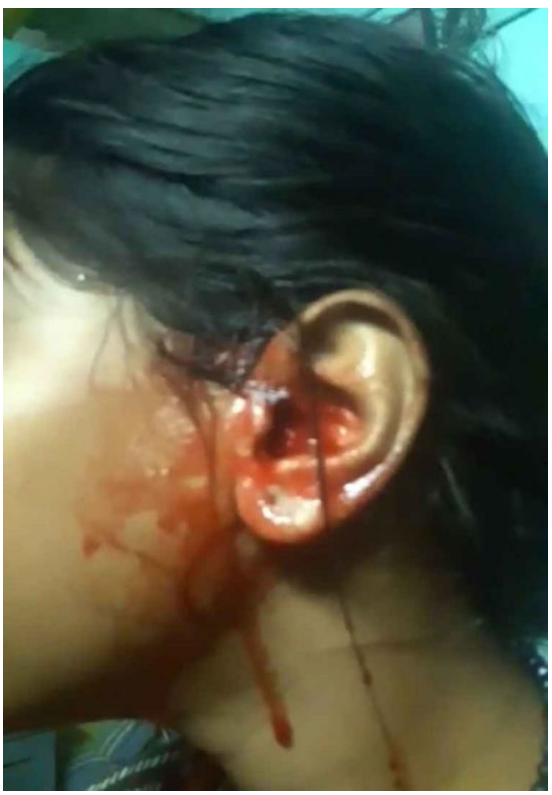

FIGURE 2 | During an episode of hematohidrosis, the picture shows blood-stained sweat droplets coming from the patient's ears.

the World Medical Association (Declaration of Helsinki) and approved by the Institute Ethical Committee (Ethics Approval number). Informed consent for publication was obtained from the guardians of the patient. Case details were collected through a clinical interview and physical examination. Further standard investigations were carried out, which included hematological tests (complete blood count, coagulation profile, and peripheral smear), biochemical analysis (liver function tests), and imaging studies (low-dose brain computed tomography [CT] scan) to rule out systemic, vascular, or hematological causes. Based on the slit-lamp examination, an eye specialist did the evaluation to see if ocular involvement existed. All laboratory and imaging findings returned normal results, which made the case even more interesting. This report reveals the clinical presentation, the diagnostic process, and a differential diagnosis of this rare condition while commenting on the possible etiological mechanisms and the literature on hematohidrosis. No treatment was instituted, and no follow-up was done, since this case report is not

TABLE 1 | Laboratory and diagnostic findings.

Investigation	Result	Normal range
Complete Blood Count (CBC)		
Red Blood Cell Count (RBC)	4.31 million/cmm	4.0–5.2 million/cmm
Hemoglobin (Hb)	12.2 g/dL	11.5–15.5 g/dL
Hematocrit (PCV)	35.60%	35–45%
Mean Corpuscular Volume (MCV)	82.6 fL	77–95 fL
Mean Corpuscular Hemoglobin (MCH)	28.2 pg	25–33 pg
Mean Corpuscular Hemoglobin Concentration (MCHC)	34.2 g/dL	31–37 g/dL
Red Cell Distribution Width-CV (RDW-CV)	12%	<12%
Total White Blood Cell (WBC) count		
Total WBC count	8,000 cells/cmm	5,000–13,000 cells/cmm
Neutrophils	47.40%	-
Lymphocytes	43.40%	-
Monocytes	5.30%	-
Eosinophils	3.70%	-
Basophils	0.20%	-
Platelet count		
Platelet count	3.04 lakhs/cmm	1.7–4.5 lakhs/cmm
Mean Platelet Volume (MPV)	10 fL	6.5–12.0 fL
Erythrocyte Sedimentation Rate (ESR)	5 mm/hour	0–20 mm/hour
Coagulation profile		
Prothrombin Time (PT)	14.2 seconds	11.0–15.0 seconds
Activated Partial Thromboplastin Time (APTT)	22 seconds	22–35 seconds
Bleeding Time (BT)	3 minutes 30 seconds	2–7 minutes
Clotting Time (CT)	5 minutes 45 seconds	5–11 minutes
INR	1.21	<1.2
Factor XIII C Assay	Clot stable in 2% acetic acid	-
Factor VIII C Assay	78%	50–150%
Plasma Fibrinogen	298 mg/dL	235–495 mg/dL
Liver Function Tests (LFTs)		
Bilirubin Total	0.5 mg/dL	0.3–1.2 mg/dL
Aspartate Aminotransferase (AST/SGOT)	35.8 U/L	<31 U/L
Alanine Aminotransferase (ALT/SGPT)	19.6 U/L	<34 U/L
Alkaline phosphatase	291 U/L	54–369 U/L
Total protein	6.90 g/dL	6.0-8.0 g/dL
Albumin	3.90 g/dL	3.8-5.4 g/dL
Imaging studies	**Findings**	
Brain CT scan	No infarct, no mass lesion, mild ethmoid sinusitis	-
Peripheral smear study	Normal erythrocytes, slight anisocytosis, lymphocytosis	-
Ophthalmological assessment	Anterior and posterior segments normal, no abnormalities detected	-

a clinical report but rather a description and academic discussion of a rare phenomenon.

Results

The case profile of our patient, an 11-year-old girl, admitted to the hospital in the last 1 year with recurrent episodes of epistaxis, hematohidrosis, and hemolacria. The episodes occurred spontaneously, varying in time interval, and were free of any prior trauma, systemic illness, or hematological disorders. On examination, blood-stained sweat droplets were noted in her ears, and, with gentle manipulations of the eyelids, blood-tinged tears were seen (**Figures 1** and **2**). Nutritionally well and maintaining stable vital signs, her systemic examination was clear; nonetheless, a whole battery of supported laboratory workups was conducted to rule out systemic bleeding disorders. The results (**Table 1**) showed that all of the hematological and coagulation parameters were within the normal range, yielding no evidence of anemia, thrombocytopenia, or coagulopathy. The studies for factor assays and plasma fibrinogen levels were also normal. Liver function tests showed no hepatic dysfunction contributing to bleeding tendencies. A low-dose CT scan of the brain was carried out to rule out any possible sources of intracranial pathology, vascular malformations, and structural malformations. Imaging findings were free of infection, hemorrhage, or mass lesions. Mild ethmoid sinusitis was noted, and the mastoid air cells were hyperpneumatized, as well as had high-riding jugular bulbs. On a peripheral smear, erythrocyte morphology was confirmed as normal with only slight anisocytosis and lymphocytosis. An ophthalmological examination and slit-lamp tests ruled out any ocular pathology responsible for hemolacria (**Table 2**). The absence of systemic abnormalities together with normal laboratory, imaging, and biochemistry results left this case a unique and diagnostic nightmare.

TABLE 2 | Imaging and ophthalmological findings.

Investigation	Findings
Brain CT scan	No infarct, no hemorrhage, no mass lesion detected
	Mild ethmoid sinusitis observed
	Over-pneumatization of mastoid air cells noted
	Widened left jugular foramen suggesting a high-riding jugular bulb
Peripheral smear study	Normal erythrocyte morphology, slight anisocytosis, mild lymphocytosis
Ophthalmological assessment	Anterior and posterior segments normal
	No ocular pathology detected
	Intraocular pressure and lacrimal gland size within normal limits

Despite such dramatic clinical presentation, the underlying cause remains elusive, thus pointing to the need for further research on the etiology and mechanisms of hematohidrosis, hemolacria, and epistaxis.

Discussions

The present case fits into the general pattern; thus, the patient was an 11-year-old girl free of hereditary history or any medical history of bleeding disorders. Similar to previous reports, hematological, coagulation, and imaging studies were within normal limits, adding to the mystery of this condition. The absence of defined systemic abnormalities indicates the need to search for additional research to find reliable diagnostic markers (14). Whereas there does not exist a standard treatment protocol, many management strategies have been explored. In some reported cases, beta-blockers like propranolol have reduced sympathetic nervous system hyperactivity, though treatment responses to these agents remain inconsistent (15). In other reports, integrative approaches combining vitamin C supplementation with Ayurvedic formulations and homeopathic remedies purportedly reduced bleeding episodes in some patients (16). One case report noted a 4-year period free of symptoms in a 21-year-old male treated with Ayurvedic medicine and counseling, suggesting a role for stress management methods in controlling symptoms (17). Another case provided evidence of an effective homeopathic treatment of a 9-year-old boy with a constitutional remedy that gave lasting symptomatic relief for a 3-year follow-up period (18). Nevertheless, these observations are only anecdotal and have received very scant clinical confirmation; indeed, they require that controlled studies be conducted to ascertain their therapeutic influence.

Hematohidrosis tends to get more attention with varying degrees of dramatic presentation. However, in the absence of standardized diagnostic criteria and guidelines, treatment poses considerable stiffness to the reluctance of clinicians (16). The extensive workup and misdiagnosis could impose psychological strain on the patient and a financial toll on patients and their families. In this light, there is a need for a very strong campaign directed toward the underpinnings of timely diagnosis and appropriate treatment. The strength of the case report is found in the focused clinical keying into identifying these contributions to the ever-increasing catalog of literature on hematohidrosis, hemolacria, and epistaxis. Findings are further supportive of the theory that these conditions occur in the absence of systemic abnormalities, thus stressing the importance of non-invasive methods for diagnostics. Suffice it to say, the case report is limited, as it fails to include any longitudinal follow-up period aimed at charting symptom patterns over time, nor were psychological or any tests on autonomic functions conducted.

Conclusion

Upon clarifying the mechanisms behind hematohidrosis and identifying biomarkers for early diagnosis, there will be **a** need to evaluate the role of stress-related neurohormonal pathways and conduct randomized trials to assess treatment efficacy. Finally, it may help open pathways for interdisciplinary research between dermatology, neurology, and psychoneuroimmunology to help delineate the mechanism(s) involved in hematohidrosis and improve clinical outcomes for affected patients. This case highlights the diagnostic complexity of hematohidrosis, hemolacria, and epistaxis and demonstrates the need for further research and awareness in the medical community.

Abbreviations

APTT, Activated Partial Thromboplastin Time; BT, Bleeding Time; CBC, Complete Blood Count; CT, Clotting Time/Computed Tomography (context-specific); INR, International Normalized Ratio; LFT, Liver Function Test; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; MCV, Mean Corpuscular Volume; MPV, Mean Platelet Volume; PT, Prothrombin Time; RDW-CV, Red Cell Distribution Width-Coefficient of Variation; SGOT (AST), Serum Glutamic Oxaloacetic Transaminase (Aspartate Aminotransferase); SGPT (ALT), Serum Glutamic Pyruvic Transaminase (Alanine Aminotransferase)

Author contributions

V.A.: Conceptualization, Data collection, Clinical assessment, Literature review, Writing – original draft. G.S.: Data analysis, Interpretation of laboratory and imaging findings, Literature review, Writing – review and editing. G.G.: Data interpretation, Literature review, Writing – review and editing.

Funding

No external funding was received for the preparation of this case report.

Acknowledgments

The authors thank Vinayaka Mission's Homeopathic Medical College & Hospital, the patient and her family, and the clinical, radiology, and laboratory teams for their support and cooperation in this case report.

Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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