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A Leser–Trélat-Like Eruption in an Elderly Woman With Gastrointestinal Alarm Symptoms and No Identified Malignancy: A Case Report

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Abstract

A Leser-Trélat sign is traditionally characterized by the abrupt appearance or rapid increase of multiple seborrheic keratoses in association with an underlying malignancy, particularly gastrointestinal (GI) adenocarcinoma. However, the specificity of this sign remains controversial because seborrheic keratoses and malignancy are both prevalent in older adults, and Leser-Trélat-like eruptions can appear without identifiable malignancy. We present the case of an 89-year-old woman with hypertension and osteoarthritis who developed diffuse seborrheic keratoses involving the trunk and extremities over approximately two years, accompanied by unintentional 20-pound weight loss, progressive dysphagia, anemia, melena, and cachexia. Her hemoglobin declined from approximately 12.5 g/dL to 9.8 g/dL, prompting investigation for GI malignancy. Anemia was clinically managed as iron deficiency. Esophagogastroduodenoscopy revealed esophagitis without an identified gastric or esophageal mass. Esophageal biopsy documented reactive squamous epithelial changes without malignant cells. Although earlier clinical documentation referenced multifocal esophageal squamous dysplasia, this diagnosis was reconciled with the available biopsy description stating focal low-grade squamous intraepithelial neoplasia. *Helicobacter pylori* testing was negative, CT of the abdomen and pelvis demonstrated no masses or lymphadenopathy, and colonoscopy was unremarkable. Iron supplementation was associated with a reported hematologic response, and subsequent fecal occult blood testing was negative. This case highlights the importance of evaluating GI alarm symptoms when widespread seborrheic keratoses raise suspicion for Leser-Trélat sign. The absence of malignancy during the documented initial evaluation does not establish a benign causal mechanism or permanently exclude occult malignancy. The gradual evolution of the cutaneous findings favors the descriptive term Leser-Trélat-like eruption. Histopathologic correlation, accurate dermatologic documentation, and longitudinal clinical assessment remain essential.

Categories: Oncology, Dermatology, Gastroenterology
Keywords: dysphagia, esophagitis, gastric adenocarcinoma mimic, iron-deficiency anemia, leser-trélat sign, melena, paraneoplastic dermatosis, pseudo-leser-trélat, reactive squamous changes, seborrheic keratoses

Introduction

Leser-Trélat sign describes the abrupt appearance or rapid increase in the number and size of multiple seborrheic keratoses, classically in association with internal malignancy. Gastrointestinal (GI) adenocarcinomas, particularly gastric malignancies, have historically been emphasized in reported cases, although hematologic malignancies and other solid tumors have also been described [1]. Clinically, the eruption may consist of multiple pigmented, waxy, or verrucous papules and plaques involving the trunk and extremities. The characteristic concern is not simply the presence of seborrheic keratoses, which are common in older adults, but a noticeable and relatively rapid change in their number or distribution.

The diagnostic specificity of Leser-Trélat sign remains controversial. Rampen and Schwengle questioned the strength of the proposed causal association between eruptive seborrheic keratoses and internal malignancy [2]. Similarly, Fink et al. conducted a prospective case-control study involving patients with and without malignancy and did not identify a significant association between the number of seborrheic keratoses and internal cancer [3]. These findings suggest that the coexistence of multiple seborrheic keratoses and malignancy may occasionally reflect their shared age-related prevalence rather than a true paraneoplastic mechanism.

Nevertheless, the clinical observation retains potential significance when eruptive skin lesions occur alongside systemic warning signs such as unexplained weight loss, anemia, GI bleeding, or progressive dysphagia. Leser-Trélat-like eruptions have also been described without identifiable malignancy. Safa and Darrieux reported such a presentation in a patient whose extensive evaluation and two-year follow-up revealed no internal cancer [4].

Several mechanisms have been proposed to explain the development of eruptive seborrheic keratoses. Paraneoplastic epidermal proliferation may theoretically result from tumor-derived growth factors or cytokines that stimulate keratinocyte proliferation [5]. Epidermal growth factor, transforming growth factor-alpha, and signaling through the epidermal growth factor receptor have been proposed as potential mediators. However, direct evidence establishing these pathways in individual cases remains limited, and their role should be considered hypothetical rather than a proven explanation [6].

Nonmalignant inflammatory conditions may also be associated with eruptive seborrheic keratoses. Flugman et al. described transient eruptions occurring in association with erythrodermic psoriasis and erythrodermic drug reactions, supporting the possibility that inflammatory signaling may occasionally produce a clinical phenotype resembling Leser-Trélat sign [7].

This case is clinically relevant because it demonstrates the diagnostic challenge posed by extensive seborrheic keratoses occurring alongside multiple GI alarm symptoms despite an evaluation that did not identify malignancy. It also emphasizes the importance of distinguishing a true paraneoplastic Leser-Trélat sign from a Leser-Trélat-like eruption, particularly when the cutaneous findings develop gradually and a causal relationship with an underlying inflammatory condition cannot be established.

Case Presentation

An 89-year-old woman with a medical history of hypertension and osteoarthritis presented with approximately two years of progressive constitutional and GI symptoms. During this period, she experienced an unintentional 20-pound weight loss, progressive dysphagia initially involving solids and subsequently occasional difficulty swallowing liquids, and deterioration in nutritional status. She also reported recent melena.

Serial laboratory assessment demonstrated a decline in hemoglobin from approximately 12.5 g/dL to 9.8 g/dL (reference range: 12.0-16.0 g/dL). Laboratory evaluation confirmed microcytic iron-deficiency anemia, with hemoglobin of 9.8 g/dL, mean corpuscular volume of 76 fL, ferritin of 10 ng/mL, serum iron of 26 µg/dL, total iron-binding capacity of 430 µg/dL, and transferrin saturation of 6% (Table *1*). This was consistent with depleted iron stores and was treated with iron supplementation, with a subsequently reported hematologic response. Given the patient's advanced age, progressive dysphagia, melena, and significant unintentional weight loss, bidirectional endoscopic evaluation was undertaken to investigate possible GI blood loss or malignancy.

Laboratory test	Patient result	Reference range	Interpretation
Hemoglobin	9.8 g/dL	12.0–16.0 g/dL	Anemia
Hematocrit	31.2%	36–46%	Decreased
Mean corpuscular volume	76 fL	80–100 fL	Microcytosis
Serum ferritin	10 ng/mL	15–150 ng/mL	Depleted iron stores
Serum iron	26 µg/dL	50–170 µg/dL	Decreased
Total iron-binding capacity	430 µg/dL	250–450 µg/dL	Upper-normal
Transferrin saturation	6%	20–50%	Markedly decreased
Reticulocyte count	1.1%	0.5–2.5%	Inadequate response relative to anemia

TABLE 1: Iron studies at presentation

The patient was prescribed ferrous sulfate 325 mg orally every other day, providing approximately 65 mg of elemental iron per dose. This regimen is consistent with the American Gastroenterological Association's (AGA) clinical practice update recommending oral iron administration no more than once daily and recognizing that alternate-day administration may improve tolerability while maintaining comparable iron absorption [8].

Physical examination demonstrated cachexia, including prominent temporal and masseter muscle wasting. Dermatologic examination identified numerous seborrheic keratoses distributed over the trunk and extremities as shown in Figure *1*. Dermatology considered the eruption clinically concerning for Leser-Trélat sign. The patient reported that the lesions had progressively appeared or increased over approximately the same two-year period as her constitutional and GI symptoms.



FIGURE 1: Multiple seborrheic keratoses on the posterior trunk

Dermatologic examination showed numerous well-demarcated, brown to dark-brown, waxy, verrucous papules and plaques involving the anterior and posterior trunk and proximal extremities. The lesions varied in size from approximately 0.5 to 2.5 cm and exhibited a characteristic stuck-on appearance consistent with seborrheic keratoses. The patient reported that the lesions had progressively increased in number over approximately two years.

Although the cutaneous and systemic abnormalities overlapped chronologically, the documented evolution of the seborrheic keratoses was gradual rather than the abrupt eruption classically associated with Leser-Trélat sign. Accordingly, the clinical presentation is most accurately described as a Leser-Trélat-like eruption rather than definitive evidence of a paraneoplastic dermatosis.

The patient reported no known family history of malignancy. Given the patient's significant unintentional weight loss, progressive dysphagia, melena, iron-deficiency anemia, cachexia, and widespread seborrheic keratoses, evaluation for an underlying GI malignancy was pursued. The patient underwent esophagogastroduodenoscopy (EGD), which demonstrated esophagitis without an endoscopically identifiable gastric or esophageal mass (Figure *2*). *Helicobacter pylori* testing was negative. Additional evaluation with computed tomography of the abdomen and pelvis revealed no masses or lymphadenopathy, and colonoscopy was unremarkable. Collectively, these findings did not identify GI or intra-abdominal malignancy during the initial diagnostic evaluation.

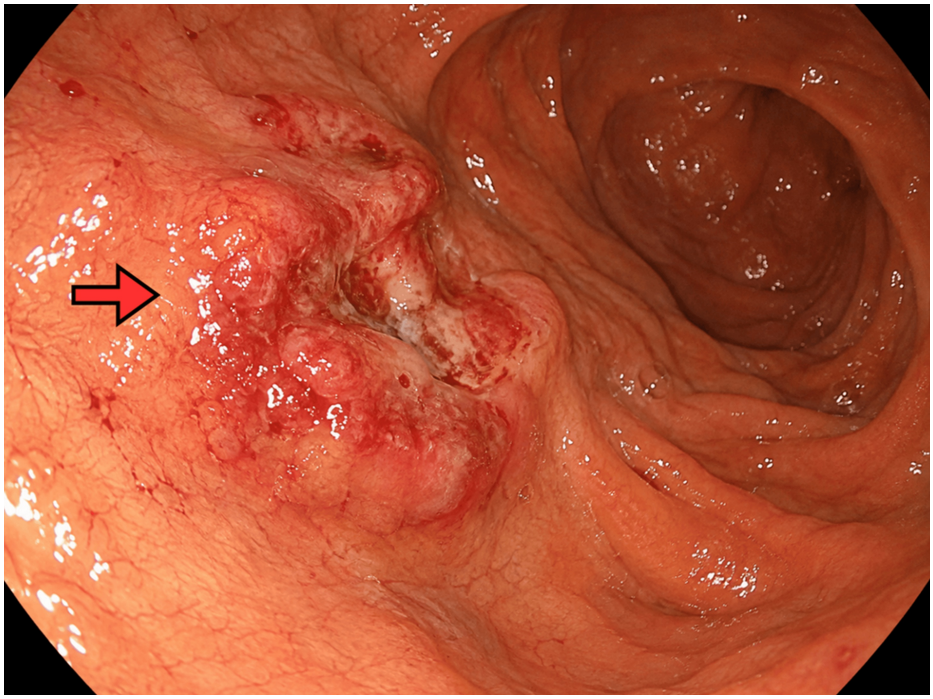


FIGURE 2: Esophagogastroduodenoscopy demonstrating esophagitis without endoscopic evidence of gastric or esophageal malignancy.

Mild esophagitis is seen, demonstrating localized mucosal erythema and superficial inflammatory changes involving the distal esophagus (red arrow). No obvious obstructing mass is depicted.

Histopathologic examination of esophageal biopsies demonstrated distinct findings at separate anatomical sites. A biopsy obtained from the mid-esophagus, approximately 29 cm from the incisors, demonstrated focal low-grade squamous intraepithelial neoplasia, characterized by cytologic atypia and architectural abnormalities predominantly involving the lower portion of the squamous epithelium, with relative preservation of superficial maturation. A separate biopsy from the distal esophagus demonstrated reactive squamous epithelial thickening and chronic inflammatory changes within the underlying lamina propria, without dysplasia or malignant cells. No high-grade dysplasia or invasive carcinoma was identified. These findings support distinguishing the focal low-grade squamous dysplasia from the reactive inflammatory changes observed in the separate biopsy specimen (Figure *3*).

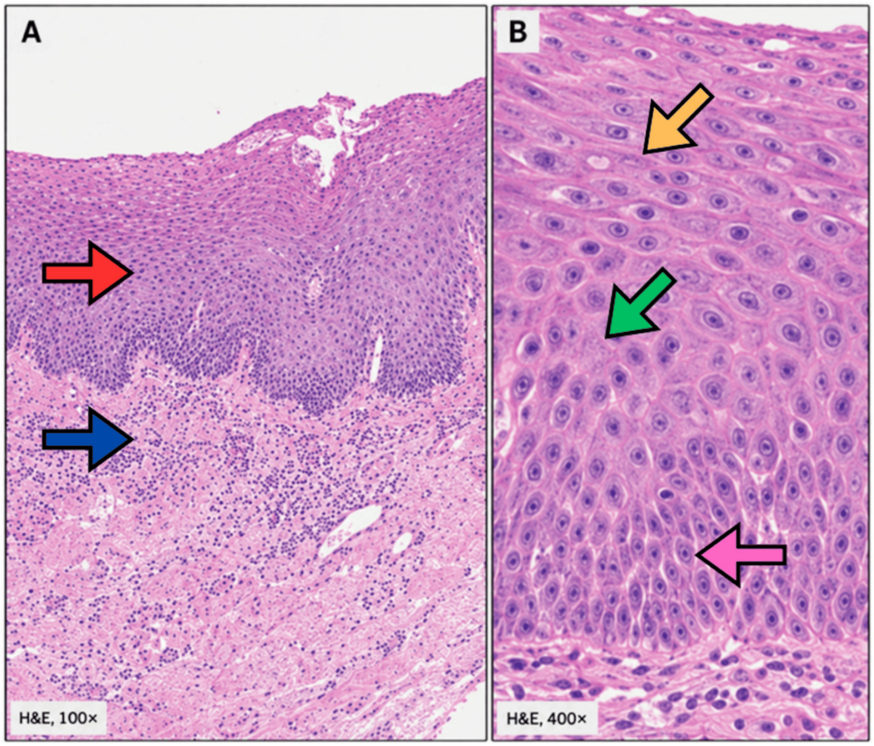


FIGURE 3: Histopathologic features of reactive esophageal squamous epithelial changes

A (H&E, original magnification ×100): Reactive squamous epithelial thickening (red arrow) and underlying chronic inflammatory infiltrates within the lamina propria (blue arrow).

B (H&E, higher-power magnification ×400): Reactive squamous cellular changes (yellow arrow) and preserved epithelial maturation (green arrow). Basal squamous epithelial atypia and disordered maturation compatible with low-grade squamous intraepithelial neoplasia (pink arrow).

H&E: hematoxylin and eosin.

The demographic, clinical, and diagnostic findings are summarized in Table 2.

Parameter	Finding	Clinical significance
Age/sex	89 years/female	Advanced age increases the prevalence of both seborrheic keratoses and malignancy
Constitutional symptoms	Unintentional 20-pound weight loss over approximately two years	Raised concern for systemic disease or occult malignancy
Dysphagia	Progression from solids to occasional difficulty swallowing liquids	Supported upper gastrointestinal evaluation
Gastrointestinal bleeding concern	Recent melena	Suggested possible gastrointestinal blood loss
Anemia	Hemoglobin declined from approximately 12.5-9.8 g/dL	Prompted evaluation for gastrointestinal bleeding and possible iron deficiency
Iron studies	Ferritin, serum iron, TIBC, and transferrin saturation are shown in Table 1	Iron deficiency anemia established
Nutritional status	Cachexia with temporal and masseter muscle wasting	Supported investigation for chronic systemic illness
Dermatologic	Diffuse seborrheic keratoses involving the	Raised suspicion for a Leser–Trélat-like eruption

findings	trunk and extremities	
Dermatologic chronology	Gradual progression over approximately two years	Less typical of classic abrupt Leser–Trélat sign
Dermatologic photographs	Original clinical photographs shown in Figure 1	Visual confirmation of lesion morphology and distribution
EGD	Esophagitis without identified gastric or esophageal mass	No endoscopically identified mass in the reported examination
Histopathology	Reactive squamous changes and chronic inflammation without identified malignant cells	Supported a reactive or inflammatory process in sampled tissue
Squamous dysplasia	Mentioned in an earlier narrative but not confirmed by the available pathology description	Requires reconciliation with the finalized pathology report
H. pylori testing	Negative	No infection identified
CT abdomen and pelvis	No masses or lymphadenopathy	No intra-abdominal malignancy detected radiographically
Colonoscopy	Unremarkable	No colorectal malignancy identified during examination
Anemia treatment	Reported improvement after iron supplementation	Suggested response to iron therapy
Post-treatment fecal occult blood test	Negative	Reassuring at the time tested but not sufficient to exclude gastrointestinal pathology
Dermatologic reassessment	Remained stable at three- and seven-month follow-up	No significant regression or development of new lesions
Surveillance	Repeat EGD at six months, with subsequent surveillance planned at 12 months from the initial examination	Given the patient's advanced age, comorbidities, and absence of high-grade dysplasia or invasive carcinoma, surveillance was selected following shared decision-making.

TABLE 2: Summary of clinical clinical, hematologic, dermatologic, endoscopic, histopathologic, and radiographic findings.

CT: computed tomography; EGD: esophagogastroduodenoscopy; TIBC: total iron-binding capacity

The clinical chronology is summarized in Table 3.

Clinical period	Event or finding
Approximately two years before presentation	Gradual development or increase of seborrheic keratoses accompanied by progressive systemic and gastrointestinal symptoms
During the two-year course	Approximately 20-pound unintentional weight loss, progressive dysphagia, declining nutritional status, and hemoglobin decrease
Before gastrointestinal investigation	Melena raised further concern for gastrointestinal blood loss or malignancy
Clinical and dermatologic evaluation	Cachexia and numerous seborrheic keratoses documented; Leser–Trélat sign considered
Gastrointestinal evaluation	EGD, biopsies, H. pylori testing, CT abdomen/pelvis, and colonoscopy performed
Diagnostic results	Esophagitis and reactive squamous changes without identified malignancy; negative*Helicobacter pylor*i testing; no abdominal masses or lymphadenopathy; unremarkable colonoscopy
Treatment and reassessment	Iron supplementation associated with reported improvement in anemia; subsequent fecal occult blood test negative
Later clinical course	Repeat EGD at six months, with subsequent surveillance planned at 12 months from the initial examination and stable lesions without occurrence of new ones

TABLE 3: Chronology of clinical symptoms, diagnostic evaluation, and documented outcome

Chronological summary of the approximately two-year symptom progression, initial diagnostic evaluation, and available treatment response. The two-year period describes the evolution of symptoms and should not be interpreted as two years of documented clinical follow-up.

EGD: esophagogastroduodenoscopy

Clinical evaluation therefore demonstrated a combination of widespread seborrheic keratoses and multiple GI alarm symptoms without an identified malignancy at the time of investigation. The inflammatory findings involving the esophageal mucosa offered a possible nonmalignant explanation for some GI symptoms, but the available evidence did not establish that esophagitis caused the cutaneous eruption.

Treatment response and follow-up (dermatologic and esophageal)

The patient received iron supplementation, with a reported improvement in anemia. Subsequent fecal occult blood testing was negative.

At the one-month follow-up, hemoglobin improved to 10.7 g/dL. Melena resolved, and dysphagia began improving after treatment of esophagitis. At the three-month follow-up, hemoglobin increased to 12.1 g/dL, ferritin to 34 ng/mL, and the seborrheic keratoses remained stable. At the six-month surveillance EGD, repeat examination demonstrated improvement in esophagitis, without residual dysplasia on targeted sampling. Targeted biopsies of the prior low-grade dysplasia site demonstrated no residual dysplasia or invasive malignancy. At the seven-month follow-up, hemoglobin was 12.4 g/dL and ferritin 48 ng/mL. No recurrent melena or progressive dysphagia. Given the patient's advanced age, comorbidities, and absence of high-grade dysplasia or invasive carcinoma, surveillance was selected following shared decision-making. At approximately 12 months from the original examination, repeat EGD, with further management dependent on histopathologic findings and clinical status. Continued surveillance according to biopsy findings, competing health risks, and patient preferences, with instruction to reassess sooner for recurrent dysphagia, melena, weight loss, or worsening anemia.

Seborrheic keratoses involving the trunk and extremities remained stable at three- and seven-month follow-up, without significant regression or development of new lesions. No spontaneous regression was observed following correction of the iron-deficiency anemia and improvement of GI symptoms. The lesions remained asymptomatic and retained their benign clinical appearance. This stability, despite improvement in the patient's systemic symptoms, did not support a clear treatment-responsive paraneoplastic or inflammatory relationship.

Discussion

This case highlights the diagnostic challenge associated with a Leser-Trélat-like eruption in an elderly patient with features strongly suggestive of GI malignancy. The combination of unexplained weight loss,

progressive dysphagia, melena, anemia, cachexia, and multiple seborrheic keratoses appropriately prompted investigation for an occult GI neoplasm.

Historically, Leser-Trélat sign has been regarded as a potential cutaneous manifestation of internal malignancy, particularly GI adenocarcinoma [1]. However, its diagnostic specificity is limited by the prevalence of seborrheic keratoses among older adults and by reports of similar eruptions occurring without malignancy. Rampen and Schwengle questioned the strength of the presumed causal association [2], while Fink et al. found no significant difference in seborrheic keratosis burden between patients with malignancy and matched controls [3].

This distinction is particularly important in the present case because the skin lesions evolved progressively over approximately two years. Classic Leser-Trélat sign typically refers to a relatively abrupt eruption, often developing over weeks or months rather than years [6]. The longer clinical course does not eliminate concern for malignancy, particularly given the GI warning symptoms, but it reduces the specificity of the cutaneous finding as evidence of a paraneoplastic process.

Comparison with previously reported nonmalignant presentations

Safa and Darrieux described a patient with eruptive seborrheic keratoses who underwent extensive malignancy evaluation without identification of cancer and remained without detectable malignancy during two years of follow-up [4]. Their report demonstrates that a clinical eruption resembling Leser-Trélat sign may occur without an identifiable malignant process. The present case is similar in that the documented initial investigation did not reveal internal malignancy. However, the patient's significant constitutional symptoms and absence of a verified long-term surveillance interval prevent equivalent conclusions regarding sustained absence of malignancy.

Flugman et al. described transient eruptive seborrheic keratoses associated with erythrodermic psoriasis and erythrodermic drug eruptions, suggesting that certain nonmalignant inflammatory conditions may accompany epidermal proliferation [7]. In those cases, the underlying inflammatory skin disorders were clinically evident, and the eruption was reported to regress as inflammation resolved. By contrast, the present patient had GI inflammatory findings, but no established inflammatory dermatologic disorder that could confidently explain the cutaneous eruption. Thus, nonmalignant inflammatory mechanisms offer a plausible framework for understanding a Leser-Trélat-like presentation, but a direct causal relationship between the patient's esophagitis and seborrheic keratoses cannot be established.

Proposed pathophysiological mechanisms

The mechanisms underlying the Leser-Trélat sign remain incompletely understood. Proposed pathways involve signaling through epidermal growth factors and their receptors, potentially stimulating keratinocyte proliferation. Epidermal growth factor and transforming growth factor-alpha have been proposed as mediators of epidermal proliferation through activation of epidermal growth factor receptor-related pathways [5,6]. Such mechanisms have been considered in malignancy-associated cutaneous eruptions because tumors may produce growth factors or inflammatory mediators capable of stimulating epidermal tissue. However, similar signaling mechanisms may occur in nonmalignant inflammatory conditions. Neither epidermal growth factor nor transforming growth factor-alpha levels were measured in this patient. Therefore, the proposed inflammatory and growth-factor pathways should be regarded as biologically plausible explanations rather than demonstrated mechanisms. The temporal overlap between GI symptoms and the development of seborrheic keratoses does not prove that the two processes share a common inflammatory or neoplastic origin.

Interpretation of the esophageal findings

The esophageal findings require particular attention because the initial clinical narrative referred to multifocal squamous dysplasia, while the available histopathologic description documented reactive squamous epithelial thickening and chronic inflammation without identified malignant cells. Reactive epithelial atypia and true squamous dysplasia are distinct pathologic entities. Esophageal squamous dysplasia is a recognized precursor lesion for squamous cell carcinoma and requires appropriate histopathologic classification [9]. The WHO classification distinguishes low-grade and high-grade squamous intraepithelial neoplasia. However, assigning a patient-specific diagnosis or grade requires the finalized pathology findings, including the presence and extent of epithelial atypia. In the present case, the available biopsy description established focal low-grade squamous intraepithelial neoplasia, classified using the WHO two-tiered system [10]. Importantly, neither reactive esophagitis nor squamous dysplasia establishes a causal explanation for the seborrheic keratoses.

Evaluation of anemia and GI alarm symptoms

The patient's anemia, melena, progressive dysphagia, unintentional weight loss, and cachexia were clinically significant regardless of the dermatologic findings. In older adults, particularly postmenopausal women, anemia accompanied by GI alarm symptoms warrants evaluation for GI blood loss and potential malignancy. The AGA recommends GI investigation in appropriately selected patients with confirmed iron-deficiency

anemia [11].

The patient's EGD, colonoscopy, *H. pylori* testing, and abdominal imaging were clinically appropriate given the documented symptom profile. However, confirmation of iron-deficiency anemia requires biochemical assessment. The documented hemoglobin decline and response to iron supplementation are compatible with iron deficiency, along with the ferritin, serum iron, total iron-binding capacity, and transferrin saturation measurements shown in Table *1*. The absence of identified malignancy during the initial evaluation is reassuring but does not permanently exclude occult neoplasia. Similarly, a negative fecal occult blood test following treatment does not definitively exclude GI disease in a patient with previously reported melena and anemia.

Limitations

This educational case illustrates several challenges in the interpretation of Leser-Trélat-like eruptions. Although the clinical evaluation included extensive GI investigation, iron studies, esophageal histopathology, and longitudinal follow-up, the mechanism underlying the cutaneous findings remains uncertain. The gradual two-year evolution of the seborrheic keratoses is less typical of the classic abrupt Leser-Trélat sign, and their persistence despite improvement in GI symptoms and correction of anemia does not establish a direct causal relationship between the skin eruption and the underlying GI findings.

The absence of an identified malignancy following initial and subsequent investigations is reassuring but cannot permanently exclude occult or subsequently developing neoplasia. Similarly, the absence of residual dysplasia in targeted surveillance biopsies does not establish permanent eradication of esophageal premalignant disease. The source of the patient's original GI blood loss was not definitively established despite the diagnostic evaluation.

Finalized histopathologic review confirmed two distinct esophageal findings: focal low-grade squamous intraepithelial neoplasia in the mid-esophagus and reactive inflammatory changes in the distal esophagus. The low-grade dysplasia was classified using the WHO two-tiered grading system and confirmed on specialist GI pathology review. No high-grade dysplasia or invasive carcinoma was identified. These findings reconciled the earlier discrepancy between the reported multifocal dysplasia and the representative biopsy description showing reactive squamous epithelial changes.

Longitudinal GI and hematologic follow-up is important because the initial absence of malignancy does not guarantee that occult disease will never become detectable. Surveillance should be individualized according to persistent symptoms, nutritional status, anemia trends, evolving dermatologic findings, and any confirmed esophageal pathology.

In an elderly patient with GI alarm symptoms and a Leser-Trélat-like eruption, the appropriate diagnostic approach is to investigate malignancy while avoiding premature assumptions that the skin findings are necessarily paraneoplastic. Confirmed low-grade esophageal dysplasia warrants specialist-directed evaluation and surveillance, whereas improvement in anemia and GI symptoms does not necessarily establish a causal relationship with persistent seborrheic keratoses.

Conclusions

Leser-Trélat-like eruptions may occur in patients with findings strongly suggestive of internal malignancy, but the presence of multiple seborrheic keratoses does not independently establish a paraneoplastic diagnosis. In this case, an elderly woman presented with progressive seborrheic keratoses, unintentional weight loss, dysphagia, melena, anemia, and cachexia, prompting extensive GI evaluation. No malignancy was identified during the initial endoscopic, histopathologic, radiographic, and colonoscopic investigations. Iron deficiency anemia was confirmed by the available iron studies. The gradual two-year progression of the skin lesions and absence of detected malignancy support describing the eruption as Leser-Trélat-like rather than definitively paraneoplastic. The relationship between esophageal inflammation and the skin findings remains hypothetical. Careful evaluation of GI alarm symptoms, accurate pathology interpretation, and individualized longitudinal assessment remain essential to avoid both premature attribution of cutaneous findings to malignancy and false reassurance after an initially negative evaluation.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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References

1. Schwartz RA: Sign of Leser-Trélat. J Am Acad Dermatol. 1996, 35:88-95. 10.1016/S0190-9622(96)90502-2
2. Rampen FH, Schwengle LE: The sign of Leser-Trélat: does it exist?. J Am Acad Dermatol. 1989, 21:50-5. 10.1016/S0190-9622(89)70147-X
3. Fink AM, Filz D, Krajnik G, Jurecka W, Ludwig H, Steiner A: Seborrhoeic keratoses in patients with internal malignancies: a case-control study with prospective accrual of patients. J Eur Acad Dermatol Venereol. 2009, 23:1316-9. 10.1111/j.1468-3083.2009.03163.x
4. Safa G, Darrieux L: Leser-Trélat sign without internal malignancy. Case Rep Oncol. 2011, 4:175-7. 10.1159/000327363
5. Ramos-E-Silva M, Carvalho JC, Carneiro SC: Cutaneous paraneoplasia. Clin Dermatol. 2011, 29:541-7. 10.1016/j.clindermatol.2010.09.022
6. Yamamoto T: Leser-Trélat sign: current observations. Expert Rev Dermatol. 2013, 8:541-6. 10.1586/17469872.2013.835926
7. Flugman SL, McClain SA, Clark RA: Transient eruptive seborrheic keratoses associated with erythrodermic psoriasis and erythrodermic drug eruption: report of two cases. J Am Acad Dermatol. 2001, 45:S212-4. 10.1067/mjd.2001.103641
8. DeLoughery TG, Jackson CS, Ko CW, Rockey DC: AGA clinical practice update on management of iron deficiency anemia: expert review. Clin Gastroenterol Hepatol. 2024, 22:1575-83. 10.1016/j.cgh.2024.03.046
9. Taylor PR, Abnet CC, Dawsey SM: Squamous dysplasia--the precursor lesion for esophageal squamous cell carcinoma. Cancer Epidemiol Biomarkers Prev. 2013, 22:540-52. 10.1158/1055-9965.EPI-12-1347
10. WHO Classification of Tumours, 5th Edition, Volume 1. Digestive System Tumours . International Agency for Research on Cancer, Lyon, France; 2019. https://publications.iarc.who.int/Book-And-Report-Series/Who-Classification-Of-Tumours/Digestive-System-Tumours-2019.
11. Ko CW, Siddique SM, Patel A, Harris A, Sultan S, Altayar O, Falck-Ytter Y: AGA clinical practice guidelines on the gastrointestinal evaluation of iron deficiency anemia. Gastroenterology. 2020, 159:1085-94. 10.1053/j.gastro.2020.06.046