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Gastric Telangiectasia and Pseudocirrhosis: A Diagnostic Pathway to Hereditary Haemorrhagic Telangiectasia

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ABSTRACT

A 60-year-old man presented with breathlessness, orthopnoea, jaundice and progressive abdominal and lower-limb swelling. Investigations showed atrial fibrillation, iron-deficiency anaemia and a cholestatic liver profile, initially suggesting a diagnosis of decompensated liver disease. A triphasic computed tomography scan of the liver demonstrated an irregular, cirrhosis-like liver with a heterogeneous "nutmeg" pattern of enhancement, alongside incidental bilateral upper-lobe pulmonary arteriovenous malformations, and echocardiography revealed a high-output circulatory state. Oesophagogastroduodenoscopy for anaemia revealed multiple bleeding gastric nodules that required haemostatic clips; biopsies showed reactive changes only, and the lesions were regarded as endoscopically presumed telangiectasias. A spontaneous episode of epistaxis prompted direct questioning, which elicited a lifelong history of recurrent nosebleeds. A nasoendoscopy was performed and revealed nasal mucosal telangiectasia. The picture satisfied three of the four Curaçao criteria, establishing a clinical diagnosis of definite hereditary haemorrhagic telangiectasia; genetic testing was pending at the time of reporting. This case shows how an atypical gastric endoscopic finding led to a diagnosis of an underlying multisystem disease, and how hepatic vascular malformation can mimic cirrhosis radiologically.

Keywords: *Hereditary Haemorrhagic Telangiectasia, Osler–Weber–Rendu, Gastric Telangiectasia, Hepatic Vascular Malformation, Pseudocirrhosis, High-Output Cardiac Failure, Iron-Deficiency Anaemia*

Introduction

Hereditary haemorrhagic telangiectasia (HHT), also known as Osler–Weber–Rendu syndrome, is an autosomal dominant vascular disorder that can present with multisystem manifestations (Shovlin *et al*., 2000; Faughnan *et al*., 2020). The types of vascular anomalies that are present in individuals with HHT can be broadly grouped into two: large arteriovenous malformations (AVMs) affecting internal visceral organs and small mucosal telangiectasias, which are usually found on the lips, tongue or fingertips (Hermann *et al*., 2025).

The diagnosis of HHT is based on the Curaçao criteria, which consist of recurrent epistaxis, mucocutaneous telangiectasia, visceral arteriovenous malformation and an affected first-degree relative (Table 1). Individuals fulfilling three or more of the above criteria have a definite clinical diagnosis of HHT, whereas identification of a pathogenic variant on genetic testing provides molecular confirmation (Faughnan *et al*., 2020). The majority of affected individuals develop epistaxis, the most easily recognised feature of the condition (Hermann *et al*., 2025).

Table 1: The Curaçao criteria for the diagnosis of hereditary haemorrhagic telangiectasia (Shovlin *et al*., 2000; Faughnan *et al*., 2020).

Criterion	Definition
Epistaxis	Spontaneous and recurrent nosebleeds
Telangiectasia	Multiple telangiectasias at characteristic sites — lips, oral cavity, fingers and nose
Visceral lesions	Gastrointestinal telangiectasia, or pulmonary, hepatic, cerebral or spinal arteriovenous malformation
Family history	A first-degree relative with hereditary haemorrhagic telangiectasia

Definite hereditary haemorrhagic telangiectasia: three or more criteria; possible or suspected: two criteria; unlikely: fewer than two criteria.

The presence and severity of visceral involvement are important factors in the overall clinical course of patients with HHT (Bernard *et al*., 1993; Garcia-Tsao *et al*., 2000; Buscarini *et al*., 2004; Garcia-Tsao, 2007). Hepatic vascular malformations, for instance, are detected in 41–78% of patients on systematic screening, but are symptomatic in only a small proportion, most commonly manifesting as high-output cardiac failure secondary to arteriovenous shunting (Garcia-Tsao, 2007; Harwin *et al*., 2020). In some cases, portal hypertension or ischaemic biliary disease may result (Garcia-Tsao *et al*., 2000). On imaging, this hepatic involvement may produce a heterogeneous, nodular enhancement pattern that mimics cirrhosis (Ianora *et al*., 2004; Harwin *et al*., 2020), which may direct clinicians down a different diagnostic pathway.

In contrast, gastrointestinal telangiectasias have been comparatively under-reported in the literature, in part due to the technical difficulty of examining the gastrointestinal tract in its entirety (Tortora *et al*., 2019). We present a patient in whom an atypical endoscopic finding encountered incidentally during investigation of iron-deficiency anaemia subsequently led to a clinical diagnosis of HHT.

Case Presentation

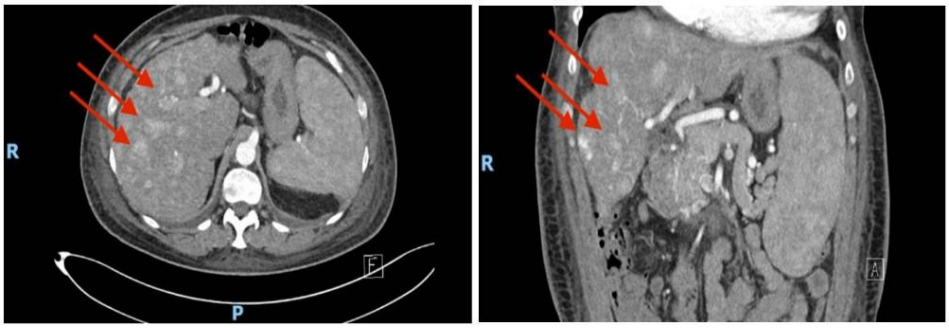
A 60-year-old man with a background of psoriasis presented to the emergency department with a three-day history of worsening breathlessness, orthopnoea and jaundice. This was superimposed on a longstanding history of progressive abdominal distension, bilateral lower-limb and scrotal swelling, alongside a decrease in exercise tolerance.

His past medical history was notable for an episode of unexplained iron-deficiency anaemia and mildly deranged liver function tests several years earlier, for which the patient had defaulted on outpatient follow-up. He was a lifelong non-smoker and non-drinker with no known personal or family history of liver disease.

On physical examination, he was found to have scleral icterus, a soft but mildly distended abdomen, and pitting oedema that involved the lower limbs and scrotum. There was no appreciable hepatosplenomegaly, stigmata of chronic liver disease, or oral or cutaneous telangiectasia on initial physical examination.

An electrocardiogram showed newly diagnosed atrial fibrillation, and initial blood investigations revealed a microcytic, iron-deficiency anaemia together with a cholestatic pattern of raised liver enzymes with conjugated hyperbilirubinaemia. Given the clinical picture of ascites, jaundice and hypoalbuminaemia, decompensated liver disease was initially considered the working diagnosis.

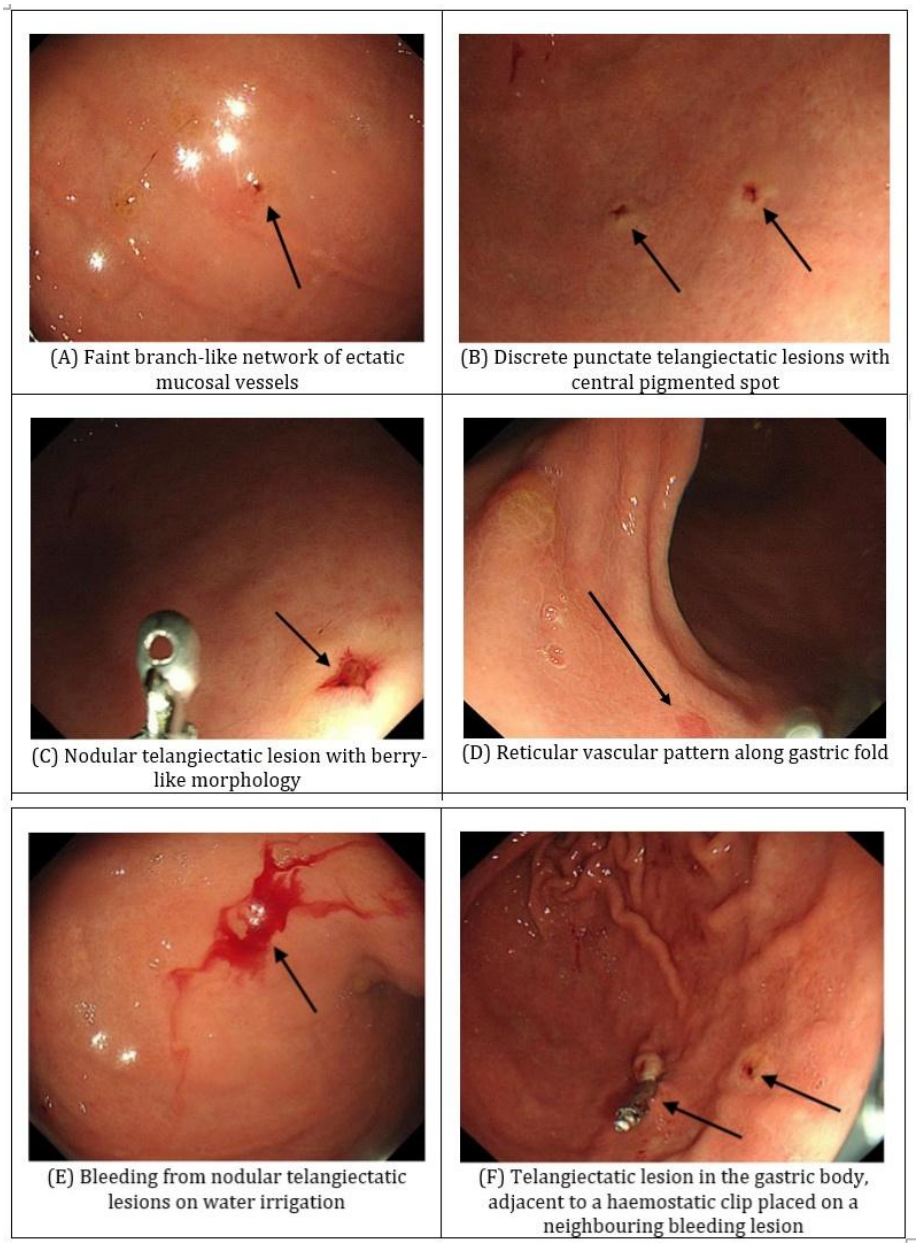
The patient then underwent a triphasic computed tomography (CT) scan of the liver, which demonstrated an irregular, cirrhosis-like contour with a heterogeneous, “nutmeg” pattern of enhancement (Fig. 1). The CT scan also identified bilateral upper-lobe pulmonary lesions in direct continuity with the pulmonary arteries, which were subsequently confirmed to be pulmonary AVMs on a formal CT pulmonary angiography scan. An extensive viral and autoimmune liver screen was unremarkable, and transthoracic echocardiography subsequently reported a hyperdynamic, high-output circulatory state.



(A) Axial arterial phase image showing an irregular, nodular hepatic contour with patchy, heterogeneous parenchymal enhancement ("nutmeg" liver)

(B) Coronal reformat demonstrating the same heterogeneous enhancement in both liver lobes

Figure 1: Triphasic CT of the liver demonstrating irregular contour with heterogeneous enhancement.



(A) Faint branch-like network of ectatic mucosal vessels

(B) Discrete punctate telangiectatic lesions with central pigmented spot

(C) Nodular telangiectatic lesion with berry-like morphology

(D) Reticular vascular pattern along gastric fold

(E) Bleeding from nodular telangiectatic lesions on water irrigation

(F) Telangiectatic lesion in the gastric body, adjacent to a haemostatic clip placed on a neighbouring bleeding lesion

Figure 2: Scattered gastric nodules seen on gastroscopy.

In view of his iron-deficiency anaemia, the patient was offered bidirectional endoscopy. On entry to the stomach, there were multiple scattered gastric nodules, several of which bled profusely on simple water irrigation and required the placement of haemostatic clips to control the bleeding (Fig. 2). Given the non-specific endoscopic appearance of these nodules, targeted biopsies were taken and sent for urgent histopathological analysis. They were subsequently reported as showing reactive changes only, with no histological confirmation of a vascular lesion. Based on their appearance and overall clinical context, the nodules were considered to represent gastric telangiectasias. Colonoscopy was also performed in the same sitting, identifying four adenomatous polyps (largest 20 mm), which were resected and proven to be tubular adenomas with low-grade dysplasia on histopathological analysis.

The following day, the patient experienced a brief, spontaneous episode of epistaxis on the ward. On direct questioning, he disclosed a history of recurrent nosebleeds since childhood. The patient was referred promptly to the otolaryngology service, where a nasoendoscopy was performed and revealed the presence of bilateral nasal mucosal telangiectasia (Fig. 3).



Figure 3: Nasal telangiectasias identified on nasoendoscopy.

Taken together, the clinical picture satisfied three of the four Curaçao criteria (recurrent epistaxis, nasal mucosal telangiectasia and pulmonary AVMs), establishing a clinical diagnosis of definite HHT. The visceral criterion was met by the presence of pulmonary AVMs noted radiologically, independently of the presumed gastric telangiectasias. Molecular confirmation by genetic testing was pending at the time of reporting. A tentative, unconfirmed family history — maternal gum bleeding and a sister with a history of recurrent epistaxis — also emerged on further questioning. A transcranial Doppler bubble study demonstrated a severe right-to-left shunt, and magnetic resonance imaging (MRI) of the brain showed several chronic, clinically silent infarcts, without any features of macroscopic cerebral AVM. His laboratory, imaging and endoscopic findings are summarised in Table 2.

Table 2: Key investigations.

Investigation	Key finding	Interpretation
Full blood count / iron studies	Haemoglobin 9.4 g/dL, microcytic; ferritin 33 µg/L	Iron-deficiency anaemia, consistent with chronic mucosal blood loss
Liver biochemistry	Bilirubin 134 µmol/L (conjugated), ALP 298 U/L, albumin 27 g/L	Cholestatic picture with hyperbilirubinaemia
Viral and autoimmune screen	Unremarkable	Excluded viral and autoimmune liver disease
CT liver (triphasic)	"Nutmeg" hepatic enhancement, irregular contour; bilateral upper-lobe pulmonary lesions	Vascular liver disease mimicking cirrhosis; probable pulmonary AVMs
CT pulmonary angiogram	Bilateral upper-lobe pulmonary AVMs (1.8×0.9 cm; 1.2×0.6 cm)	Confirmed pulmonary AVMs; risk factor for right-to-left shunting
Echocardiogram	Dilated chambers, ejection fraction 55%, elevated cardiac index	High-output cardiac physiology
Oesophagogastroduodenoscopy	Multiple bleeding gastric nodules	Raised suspicion for gastric telangiectasias; biopsies showed reactive changes only
Nasoendoscopy	Bilateral nasal telangiectasia	Confirmed mucocutaneous telangiectasia
Transcranial Doppler bubble study	Severe right-to-left shunt	Supported a systemic right-to-left shunting state

A multidisciplinary approach involving gastroenterology, cardiology, respiratory medicine, haematology, otolaryngology and neurology was adopted in the management of this patient's condition. Fluid overload was managed with intravenous and oral diuretics, whilst digoxin was added for rate control of his newly diagnosed atrial fibrillation and to support the failing high-output circulation. Anticoagulation was withheld, given the competing bleeding risk from the nasal telangiectasias and presumed gastric telangiectasias. In view of his significant hyperbilirubinaemia, referral to a specialist liver transplant centre for consideration of liver transplantation was discussed with the patient. Bevacizumab, a form of systemic antiangiogenic therapy, was also considered as an alternative should he be deemed an unsuitable transplant candidate.

Following diuresis, the patient improved symptomatically and was discharged with outpatient follow-up arranged across the multiple disciplines. The referral to a liver transplant centre was formalised prior to discharge, given the extent of hepatic and pulmonary shunting alongside his persistent hyperbilirubinaemia. Right heart catheterisation was also offered as an outpatient procedure, to formally quantify the shunt and guide further intervention. Outpatient genetic testing was arranged on discharge. At the time of writing this manuscript, the results of genetic testing, right heart catheterisation and specialist transplant assessment were still pending. Table 3 summarises the sequence of events during the patient's hospital admission, from the time of presentation to the emergency department to discharge.

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Table 3: Timeline of presentation and diagnosis.

Day	Event
Day 1	Presented with breathlessness, orthopnoea and limb swelling; scleral icterus and new atrial fibrillation noted. Admitted with a working diagnosis of decompensated liver disease.
Day 2–3	Triphasic CT liver demonstrated a "nutmeg" liver with a cirrhosis-like contour; bilateral upper-lobe pulmonary lesions were noted incidentally, suspicious for AVMs.
Day 4	Echocardiography demonstrated dilated cardiac chambers with a preserved ejection fraction and an elevated cardiac index, in keeping with a high-output circulatory state.
Day 5	Oesophagogastroduodenoscopy was performed for iron-deficiency anaemia, unexpectedly revealing multiple bleeding gastric nodules; six clips were required for haemostasis. Colonoscopy identified four adenomatous polyps, which were resected.
Day 6	A spontaneous episode of epistaxis occurred on the ward and a lifelong history of nosebleeds was elicited on direct questioning. Nasoendoscopy confirmed nasal telangiectasia, and CT pulmonary angiography confirmed bilateral pulmonary AVMs. A working diagnosis of HHT was made.
Day 9–10	Gastric biopsies were reported as showing reactive changes only. A clinical diagnosis of definite HHT, fulfilling three of the four Curaçao criteria, was established, pending molecular confirmation. Outpatient referral to clinical genetics was made.
Day 11–12	Transcranial Doppler demonstrated a severe right-to-left shunt; MRI brain showed several chronic, clinically silent infarcts but no macroscopic cerebral AVM. Referral to a liver transplant centre was made and the patient was subsequently discharged with multidisciplinary follow-up arranged.

Discussion

In our patient, visceral involvement preceded, and initially overshadowed the more classically recognised mucocutaneous features of HHT. The initial presenting complaint of breathlessness and jaundice did not include a history of recurrent epistaxis, which was only elicited subsequently upon direct, targeted questioning. Whilst we were eventually led to a clinical diagnosis of HHT, our initial differential diagnoses favoured more common presentations, such as new-onset cardiac failure or decompensated liver cirrhosis. This underscores the importance of comprehensive and targeted history-taking and physical examination in guiding appropriate diagnostic evaluation in patients presenting to the emergency department.

Adding further complexity to the diagnostic assessment, gastrointestinal telangiectasias in HHT have been reported to be endoscopically pleomorphic. Lesions vary in size — typically ≤3 mm, although larger lesions may occur in more severely affected individuals (Mora-Luján *et al*., 2019). They may range from flat macular lesions to raised, berry-like lesions as they mature, and their arborisation or branching patterns are variable (Baltes *et al*., 2013; Ortman and Ortolani, 2024). This broad morphological spectrum may also overlap with sporadic angiodysplasia, Dieulafoy lesions and gastric antral vascular ectasia (GAVE) (Regula *et al*., 2008; Iannone *et al*., 2016). Consequently, even structured endoscopic classification systems for vascular lesions may demonstrate interobserver variability (Jang *et al*., 2010; Leenhardt *et al*., 2019; Cortegoso Valdivia *et al*., 2022; Rajabnia *et al*., 2022).

Fig. 4 shows endoscopic images from published case reports of gastrointestinal telangiectasias in individuals with HHT. These cases illustrate the endoscopic heterogeneity of gastric telangiectasias in HHT, which may range from solitary, discrete lesions to extensive, near-confluent vascular changes. This morphological variability may complicate recognition and reduce diagnostic confidence. Interestingly, one report (Parva and Tuculanu, 2018) suggested that enhanced imaging modalities such as FICE (Flexible spectral Imaging Color Enhancement) virtual chromoendoscopy could potentially improve visualisation and characterisation of gastric vascular lesions, though further studies are needed to validate this.

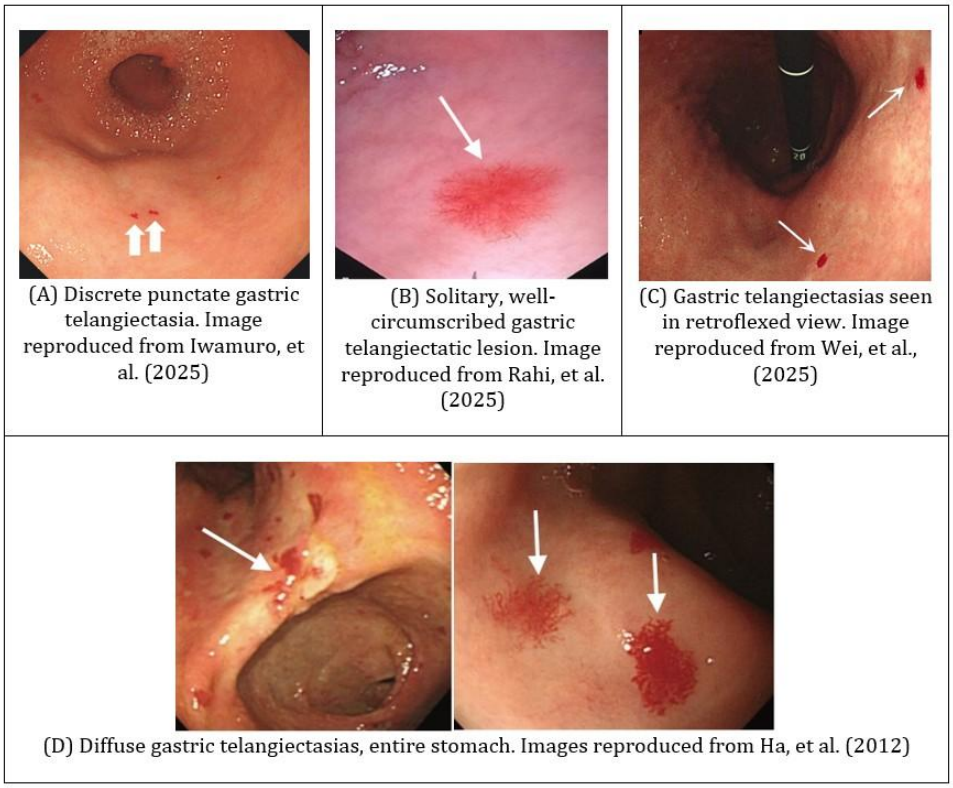


Figure 4: Endoscopic spectrum of gastric telangiectasia in HHT.

The endoscopic appearance encountered in our patient — multiple discrete nodules with disproportionately brisk bleeding on simple water irrigation — was atypical for routine peptic or erosive disease. Such endoscopic findings should prompt consideration of an underlying vascular or telangiectatic aetiology. A further diagnostic pitfall, as encountered in this case, is that forceps biopsies of such lesions may yield non-specific "reactive" histology, possibly because the ectatic vessels lie largely within the submucosa, beyond the reach of standard forceps (Sami *et al*., 2014). To our knowledge, published data on the diagnostic yield of gastric biopsy in HHT are limited, and endoscopic diagnosis is generally made on morphological grounds (Longacre *et al*., 2003; Faughnan *et al*., 2020). In our patient, given the absence of

histological confirmation, we have regarded the gastric lesions as presumed telangiectasias based on endoscopic appearance and the broader clinical context.

Although our patient presented with overt jaundice, hepatic HHT may instead initially manifest solely as heart failure, often high-output cardiac failure resulting from intrahepatic arteriovenous shunting (Bernard *et al*., 1993; Garcia-Tsao *et al*., 2000; Buscarini *et al*., 2004; Garcia-Tsao, 2007; Cusumano *et al*., 2021). Radiologically, hepatic vascular malformations may closely resemble cirrhosis (Ianora *et al*., 2004; Harwin *et al*., 2020), a phenomenon known as "pseudocirrhosis". In general, the term pseudocirrhosis refers to morphological changes in the liver that resemble cirrhosis, but lack the classical histopathological features seen in true cirrhosis (Adike *et al*., 2016).

In HHT, pseudocirrhosis describes a nodular, cirrhosis-like liver in which diffuse intrahepatic arteriovenous, arterioportal or portovenous shunting causes heterogeneous parenchymal perfusion and regenerative nodular transformation (nodular regenerative hyperplasia or focal nodular hyperplasia), rather than the bridging fibrosis and architectural distortion that are seen in true cirrhosis (Wanless and Gryfe, 1986; Garcia-Tsao, 2007; Sadlik *et al*., 2024). Pseudocirrhosis should also be distinguished from congestive hepatopathy, in which hepatic injury and fibrosis result from venous congestion due to raised right-sided filling pressures, without a primary hepatic vascular abnormality (Fortea *et al*., 2020). In our patient, the high-output state, pulmonary AVMs and an unremarkable chronic liver disease screen favoured HHT-related hepatic vascular involvement, although liver histology was not obtained and a superimposed congestive component secondary to high-output cardiac failure cannot be excluded.

Limitations

This report has the inherent limitations of a single retrospective case, and its findings cannot be generalised. Molecular confirmation of HHT, haemodynamic assessment with right heart catheterisation and liver transplant assessment were pending at the time of writing this manuscript, and neither the gastric lesions nor the liver lesions were characterised histologically. Nonetheless, we believe this case adds a descriptive example of the gastric endoscopic phenotype of HHT, and illustrates a diagnostic pathway that may be of practical use to gastroenterologists and general physicians encountering similar multisystem presentations.

Conclusion

Hereditary haemorrhagic telangiectasia may first present in adulthood with visceral complications, such as high-output cardiac failure, hepatic vascular malformations and pulmonary arteriovenous malformations, rather than the classical mucocutaneous features. Multiple gastric nodules that bleed disproportionately on simple water irrigation should prompt consideration of an underlying vascular malformation syndrome, and non-specific "reactive" histology on forceps biopsy does not exclude telangiectasia. A cirrhosis-like ("nutmeg") liver on imaging, alongside the presence of arteriovenous malformations and no evidence of primary liver disease, should raise the possibility of pseudocirrhosis from intrahepatic shunting rather than true cirrhosis.

Declarations

Patient consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the consent form is available for review by the Editor-in-Chief upon request.

Reporting guideline compliance: This case report was prepared in accordance with the CARE (CAse REport) guidelines. Permission to reproduce the endoscopic images was obtained from the relevant copyright holder(s).

Contributors: TWD and DP conceived and designed the case report. SL and TWD collected the relevant clinical data. SL drafted the manuscript. TWD and DP interpreted the clinical findings and critically revised the manuscript. All authors reviewed and approved the final manuscript and accept responsibility for the integrity and accuracy of its content.

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