International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

*Case Series* *DOI: 10.61336/im/25-11-34*

# Iron Pill–Induced Gastritis: A Case Series.

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**Received:** 13-10-2025
**Revised:** 23-10-2025
**Accepted:** 10-11-2025
**Published:** 22-11-2025

## Abstract

***Background:*** Iron pill–induced gastritis (IPG) is an under-recognized but clinically important complication of solid oral iron supplementation. Though mild gastrointestinal side effects of oral iron are well-documented, severe erosive gastropathy leading to hemorrhage is infrequently reported, and pediatric cases remain exceedingly rare. ***Case Presentations:*** We report three cases of IPG: a 55-year-old woman with hematemesis after two weeks of ferrous sulfate; a 60-year-old woman with erosive gastritis following six months of supplementation; and a 12-year-old boy with progressive epigastric pain after three months of oral iron therapy. All three patients underwent upper gastrointestinal endoscopy demonstrating hemorrhagic erosions with characteristic brownish mucosal deposits concentrated in the gastric antrum. Histopathology confirmed Pattern B gastric siderosis in each case, with iron deposition verified by Perls' Prussian blue staining. ***Conclusion:*** This case series underscores the importance of recognizing IPG across different age groups. Diagnosis requires integration of clinical history, endoscopic findings, and histopathological confirmation by special stains to demonstrate iron. Management involves immediate cessation of solid oral iron, proton pump inhibitor therapy, and transition to liquid or intravenous iron formulations. Early recognition is critical to prevent serious upper gastrointestinal hemorrhage.

**Keywords:** iron pill gastritis; oral iron, erosive gastropathy;gastricsiderosis; ferrous sulfate; pediatric.



## INTRODUCTION

Iron deficiency anemia (IDA) is the most prevalent nutritional deficiency worldwide, affecting approximately one-quarter of the global population according to World Health Organization estimates.(1) Among Oral iron supplementation, most commonly ferrous sulfate tablets remain the first-line treatment due to its low cost and wide accessibility. Although mild gastrointestinal adverse effects such as nausea, constipation, and dark stools are well recognized, the capacity of solid oral iron to induce serious mucosal injury is far less appreciated in everyday clinical practice.(2)

Iron pill–induced gastritis (IPG) represents a distinct clinicopathological entity within the spectrum of drug-induced gastropathy.(3) Unlike the mild discomfort attributable to routine oral iron use, IPG involves direct mucosal damage caused by the concentrated local dissolution of iron tablets, generating reactive oxygen species that disrupt the gastric epithelial barrier and produce erosions, ulcerations in most of the cases and significant upper gastrointestinal hemorrhage in severe cases.

Despite being an established entity in the pathology literature, IPG remains under-recognized clinically because its presenting symptoms such as epigastric pain, nausea, vomiting, and hematemesis that are nonspecific and commonly attributed to other causes such as peptic ulcer disease or Helicobacter pylori infection.(4) Here we present three cases of Iron Pill Gastritis spanning two adult women and one child to illustrate the clinicopathological spectrum of this condition across different age groups, highlight the need for vigilance in patients receiving oral iron therapy, and reinforce the essential role of endoscopy and histopathology in establishing the diagnosis.

## CASE PRESENTATIONS

**Case 1:** Hematemesis and Transaminase Elevation in a 55-Year-Old Woman Clinical Presentation. A 55-year-old woman presented with a 5-day history of multiple episodes of hematemesis and melena, accompanied by nausea and epigastric pain. Lab investigations revelaled microcytic hypochromic anemia – iron defiency anemia with reduced serum ferritin.

She had been prescribed oral ferrous sulfate tablets two weeks prior for recently diagnosed iron deficiency anemia and was taking the medication approximately two hours after meals. She reported no concurrent use of non-steroidal anti-inflammatory drugs (NSAIDs), aspirin, antiplatelet agents, or bismuth-containing preparations.

Examination and Investigations. On admission, the patient appeared pale and fatigued with diffuse abdominal tenderness. Laboratory evaluation revealed microcytic anemia alongside markedly elevated transaminases (Table 1). Serological testing for viral hepatitis (A, B, and C) and autoimmune hepatitis was negative. Celiac disease screening was unremarkable. Abdominal ultrasonography showed no structural hepatobiliary abnormalities.

**Table 1. Laboratory parameters for Case 1.**

| Parameter | Value | Reference Range |
|---|---|---|
| Hemoglobin | 11.2 g/dL | 12–16 g/dL |
| Mean Corpuscular Volume | 67.4 fL | 80–99 fL |
| Serum Iron | 51.56 µmol/L | 7–25 µmol/L |
| Ferritin | 6.7 ng/mL | 11–307 ng/mL |
| AST | 404.8 U/L | 5–40 U/L |
| ALT | 429.7 U/L | 7–56 U/L |

**Endoscopic and Histopathological Findings.** Upper GI endoscopy revealed edematous gastric folds with multiple hemorrhagic erosions and diffuse brownish deposits most pronounced in the antrum. Gastric biopsies demonstrated erosions with intraepithelial neutrophilic infiltration and coarse brown granular deposits into mucosa and submucosa stained bluish colour on perls prussian blue stain consistent with iron accumulation (Pattern B gastric siderosis). Duodenal biopsies were histologically normal. No Helicobacter pylori organisms were identified. No dysplasia was present.

**Management and Outcome.** Ferrous sulfate was immediately discontinued. Treatment comprised gastric lavage, intravenous proton pump inhibitor (PPI) therapy, hemostatic agents, and hepatic support therapy. The patient demonstrated marked clinical improvement and was discharged on oral PPI and hepatoprotective medication. Follow-up endoscopy at one month showed complete resolution of mucosal lesions with no residual iron deposition on repeat histopathology. Anemia was subsequently managed with a liquid oral iron formulation, resulting in hemoglobin normalization without recurrence.

**Case 2:** Erosive Gastritis after Prolonged Supplementation in a 60-Year-Old Woman Clinical Presentation. A 60-year-old woman presented with progressive epigastric discomfort and nausea. She had been taking ferrous sulfate 325 mg once daily for over six months for iron deficiency anemia. She denied concurrent use of NSAIDs, bismuth, aspirin, or antiplatelet agents and was hemodynamically stable on examination.

**Investigations.** Laboratory parameters are summarized in Table 2. Hemoglobin remained below the normal reference range despite prolonged supplementation, raising concern for ongoing GI blood loss. Renal function and iron saturation indices were within acceptable limits.

**Table 2. Laboratory parameters for Case 2.**

| Parameter | Value | Reference Range |
|---|---|---|
| Hemoglobin | 9.9 g/dL | 12–16 g/dL |
| Mean Corpuscular Volume | 97 fL | 80–99 fL |
| Ferritin | 150 ng/mL | 11–307 ng/mL |
| Iron Saturation | 32% | 11–50% |
| TIBC | 299 µg/dL | 284–507 µg/dL |
| BUN | 19 mg/dL | 9–23 mg/dL |
| Creatinine | 0.75 mg/dL | 0.55–1.02 mg/dL |

**Endoscopic and Histopathological Findings.** Upper GI endoscopy demonstrated edematous gastric folds, multiple hemorrhagic erosions, and diffuse brownish deposits in the antrum. Biopsies showed an eroded epithelial surface with infiltrated and edematous lamina propria. Brownish to golden crystalline material within the ulcer bed was confirmed as iron by Perls' Prussian blue staining, consistent with Pattern B gastric siderosis.

**Management and Outcome.** Oral ferrous sulfate was discontinued and PPI therapy initiated. Despite repeated counseling to switch to a liquid iron formulation, the patient continued tablet use, resulting in persistent mucosal irritation on follow-up endoscopy—underscoring the critical role of patient education and adherence.

**Case 3:** Upper GI Bleeding in a 12-Year-Old Boy Clinical Presentation. A 12-year-old boy presented with a two-week history of progressively worsening epigastric pain and poor oral intake. He had been prescribed oral iron therapy three months earlier for iron deficiency anemia, with no history of NSAID use, toxic ingestion, or prior GI disease.

**Investigations.** Laboratory parameters are summarized in Table 3. Hemoglobin and hematocrit were below age-appropriate reference ranges. Serum iron was subnormal despite three months of supplementation, consistent with ongoing GI blood loss. Elevated ferritin likely reflected acute-phase reactivity.

**Table 3. Laboratory parameters for Case 3.**

| Parameter | Value | Reference Range |
|---|---|---|
| Hemoglobin | 104 g/L | 125–170 g/L |
| Hematocrit | 0.34 L/L | 0.37–0.49 L/L |
| Mean Cell Volume | 100 fL | 78–94 fL |
| Ferritin | 158 µg/L | 20–100 µg/L |
| Serum Iron | 5 µmol/L | 7–25 µmol/L |
| Transferrin | 31 µmol/L | 20–41 µmol/L |

**Endoscopic and Histopathological Findings.** Endoscopic findings mirrored those in Cases 1 and 2: edematous gastric folds, multiple hemorrhagic erosions, and brownish antral deposits. Histopathology revealed an eroded epithelial surface with edematous inflamed lamina propria and brownish-golden crystalline deposits.

Perls' Prussian blue staining confirmed intracytoplasmic brownish black iron granules consistent with Pattern B gastric siderosis. This case represents one of only a few published cases of IPG in a pediatric patient [2,3].

**Management and Outcome.** Oral iron tablets were immediately discontinued. Intravenous PPI therapy and supportive care were initiated. Clinical parameters improved and the patient was transitioned to a liquid iron formulation with close outpatient follow-up.

**Summary of Cases**

**Table 4. Comparative summary of all three cases.**

| Feature | Case 1 | Case 2 | Case 3 |
|---|---|---|---|
| Age / Sex | 55-year-old female | 60-year-old female | 12-year-old male |
| Duration of iron therapy | 2 weeks | 6 months | 3 months |
| Presenting symptoms | Hematemesis, melena, epigastric pain | Epigastric discomfort, nausea | Epigastric pain, poor oral intake |
| Endoscopic findings Site of deposit | Hemorrhagic erosions, brownish deposits (antrum) | Hemorrhagic erosions, brownish deposits (antrum) | Hemorrhagic erosions, brownish deposits (antrum) |
| Histopathology | Coarse brown iron deposits; neutrophilic infiltrate; H. pylori negative | Eroded epithelium; crystalline iron confirmed by Perls' stain | Eroded epithelium; yellow-orange granules confirmed by Perls' stain |
| Siderosis pattern | Pattern B | Pattern B | Pattern B |
| Management | IV PPI, gastric lavage, hemostatic; switched to liquid iron | Oral PPI; counselled to switch formulation | IV PPI, supportive care; switched to liquid iron |
| Outcome | Complete endoscopic and histological resolution at 1 month | Persistent injury due to non-adherence | Clinical improvement; outpatient follow-up ongoing |

## DISCUSSION

This case series illustrates the clinicopathological spectrum of iron pill–induced gastritis across three patients of differing ages and clinical contexts. All three cases share hallmark features: a history of oral ferrous sulfate use, endoscopic evidence of hemorrhagic erosions with brownish mucosal deposits predominantly in the gastric antrum, and histopathological confirmation of iron deposition consistent with Pattern B gastric siderosis. Together, they reinforce several important principles regarding the pathogenesis, diagnosis, and management of this under-recognized condition. Also importance of educating the patient about this entity.

Iron tablet formulations particularly ferrous sulfate causes gastric mucosal injury through both direct and indirect mechanisms.(5)

Directly, as iron tablets dissolve in gastric acid, they release free iron ions that catalyze the Fenton reaction, producing hydroxyl radicals and reactive oxygen species that cause oxidative damage to the mucosal epithelium and disrupt the protective mucosal barrier. Indirectly, the resulting rise in intraluminal iron concentration and acidity may alter the gastric microenvironment, promoting further inflammation.(6) Crucially, this mucosal injury is concentration-dependent that is if tablets dissolve in a localized gastric pool, producing a high-concentration corrosive effect that is not replicable with liquid iron formulations, which distribute more diffusely across the mucosa.(7)

The large-scale systematic review and meta-analysis by Tolkien et al., examined 43 randomized controlled trials comprising 6,831 adult participants, confirmed that ferrous sulfate is associated with a significantly increased risk of gastrointestinal side-effects compared with both placebo (OR 2.32; 95% CI 1.74–3.08) and intravenous iron, and importantly found no relationship between dose and the magnitude of this risk underscoring the inherent mucosal irritant potential of the solid formulation itself.(8)

The degree of crystalline iron accumulation and its correlation with the extent of mucosal injury have been further substantiated by Onorati et al., who highlighted in their retrospective series that iron overload within the gastric mucosa represents an underdiagnosed and underreported entity in routine pathology practice, with recognition requiring active histochemical evaluation rather than passive identification on hematoxylin and eosin staining alone.(9)

Three distinct histopathological patterns of gastric iron deposition have been characterized and validated in subsequent multi-cohort analyses. Kothadia et al., in a comprehensive review of gastric siderosis incorporating clinical and histopathological data, described Pattern A (nonspecific gastric siderosis) as iron deposits predominantly within stromal macrophages and the surface epithelium, typically associated with pre-existing inflammation, prior mucosal hemorrhage, or underlying systemic conditions.

Pattern B observed in all three patients reported here is defined by predominantly extracellular crystalline iron accumulation in the lamina propria and is most specifically linked to solid oral iron supplementation. Pattern C (gastric glandular siderosis) involves iron accumulation in the glandular epithelium of the antrum and fundus and is characteristically associated with systemic iron overload disorders such as hereditary hemochromatosis or transfusion-related siderosis.(10)

The clinicopathological significance of this three-pattern classification has been most rigorously validated by Sarwate et al. in a large multi-institutional study of 76 gastric siderosis cases drawn from three independent cohorts. They demonstrated that the degree of glandular iron deposition (Pattern C/gastric glandular siderosis) was positively and significantly associated with serum ferritin levels ($p = 0.002$), transferrin saturation ($p = 0.003$), and a history of blood transfusion ($p = 0.009$), thereby providing objective clinicopathological anchoring for distinguishing Pattern C from Pattern B.(11) Correct identification of Pattern B on biopsy ideally confirmed with Perls' Prussian blue stain and thus this is highly discriminating and should prompt immediate reassessment of the patient's oral iron regimen.

The prevalence of crystalline iron deposition in upper GI endoscopic biopsies is relatively low but not negligible. Kothadia et al. confirmed that gastric iron deposition has been documented in association with oral iron medications, hemochromatosis, alcohol abuse, blood transfusions, and hepatic cirrhosis, and that recognition of the precise pattern carries direct therapeutic implications.(10) In the largest published case series to date incorporating both prospective and retrospective elements, gastric iron deposition was associated with erosion in the majority of cases and with reactive gastritis in the overwhelming majority, with nearly all patients having a history of oral iron supplementation.(12)

These figures suggest that IPG may be substantially underdiagnosed in routine clinical practice , a conclusion reinforced by Onorati et al., whose retrospective series found that iron overload in the gastric mucosa is rarely documented in clinical and pathology reports even when present,(9) and by Parajuli et al., who documented iron-pill–induced gastric ulceration after three months of ferrous sulfate therapy in a patient with pre-existing gastroesophageal reflux disease, underscoring that the burden of solid iron–related GI injury is likely underestimated.(5)

The clinical presentation of IPG is heterogeneous and non-specific, ranging from mild dyspepsia to life-threatening hemorrhage. Patients may complain of epigastric pain, nausea, vomiting, and early satiety,(13) or as in Case 1 present acutely with hematemesis and melena. The absence of pathognomonic symptoms and the common occurrence of polypharmacy in patients with IDA may delay recognition of the iron–mucosal injury relationship. Risk appears elevated in patients with pre-existing upper GI disease, including chronic GERD, peptic ulcer disease, and gastric dysmotility.

The markedly elevated transaminases in Case 1 (AST 404.8 U/L; ALT 429.7 U/L) alongside supranormal serum iron levels (51.56 µmol/L) warrant specific comment. While thorough serological exclusion of viral and autoimmune hepatitis was performed, the concurrent systemic iron overload evidenced by markedly elevated serum iron despite low ferritin, suggesting acute iron loading exceeding storage capacity usually raises the possibility of direct iron-mediated hepatotoxicity, as has been described in the context of acute and subacute iron excess states.

This finding suggests that the consequences of ferrous sulfate–induced mucosal injury may extend beyond the gastric mucosa, and that liver function monitoring is appropriate in patients presenting with severe IPG, particularly those with supranormal serum iron concentrations.(14)

Case 3 is particularly noteworthy as one of only a few documented instances of IPG in a pediatric patient. Although iron deficiency is common in children and ferrous sulfate is among the most frequently prescribed iron preparations in pediatric practice, the potential for mucosal injury is not routinely communicated to caregivers. Published pediatric cases remain rare; Meliţ et al. described IPG in a 14-year-old girl receiving oral iron for only two weeks who presented with upper GI hemorrhage, (15) elevated transaminases, and elevated serum iron, a presentation strikingly similar to our Case 1. YOur pediatric case confirms that the mucosal injury potential of solid iron formulations is not limited to adults and that clinicians managing pediatric patients on prolonged oral iron therapy should maintain a low threshold for endoscopic investigation when GI symptoms develop or fail to resolve.

Prompt discontinuation of oral iron tablets is the most important initial step, as this alone often leads to symptomatic improvement and mucosal healing, as demonstrated in Case 1. Proton pump inhibitors are the pharmacological agents of choice to accelerate mucosal recovery by reducing acid-mediated injury. H2 receptor antagonists may provide adjunctive benefit, and sucralfate has been used to form a protective coating over eroded surfaces.

For patients with significant hemorrhage as in Case 1, intravenous PPI therapy and targeted hemostatic measures are indicated. Lichtenstern and Akhtar highlighted the persistent iron tablet use despite medical advice,(16) as observed in our Case 2, can lead to prolonged and recurrent mucosal injury even when endoscopic healing has partially occurred, emphasizing that patient education and close follow-up are non-negotiable components of management.

For patients requiring ongoing iron replacement, intravenous iron preparations entirely bypass the GI tract and are preferred in those with significant mucosal injury or persistent bleeding. Among oral formulations, liquid iron preparations are considerably less injurious than solid tablets owing to their inability to concentrate within the stomach to the same extent.(17) As Bloor et al. demonstrated in a detailed review of the GI consequences of oral iron supplementation, luminal iron causes intestinal inflammation through reactive oxygen species generation and promotes dysbiosis by increasing pathogenic species while reducing protective microbiota effects that are substantially mitigated when iron bypasses the gut lumen entirely.(18) Of the solid oral formulations, ferrous sulfate carries the greatest mucosal irritant potential, followed by ferrous gluconate, succinate, and carbonate.

Current American Gastroenterological Association guidelines do not specifically endorse any single oral formulation over another, and drug selection should be guided by individual patient risk factors, comorbidities, adherence, and tolerability.(19) Preventive strategies include administering iron with food to reduce direct mucosal contact time, using enteric-coated or modified-release preparations where appropriate, and selecting the lowest effective dose.

**Limitations**

This report carries limitations inherent to its case series design. The absence of a standardized follow-up protocol across all three patients precludes formal conclusions regarding long-term outcomes. Photographic endoscopic and histopathological images, which would substantially strengthen the evidence, could not be included in this manuscript. Minor informational gaps particularly regarding the complete medication history and comorbidity profile in Cases 2 and 3 may limit analytical depth. Nonetheless, the consistency of endoscopic and histopathological findings across all three cases, and their concordance with the established published literature, supports the validity of the diagnosis and the broader clinical conclusions drawn.

## CONCLUSION

Iron pill–induced gastritis is a clinically meaningful but under-recognized complication of oral iron supplementation. This case series demonstrates that IPG can affect patients across a broad age spectrum from children to older adults with presentations ranging from chronic erosive gastropathy to acute upper GI hemorrhage.

The diagnosis requires integration of clinical history, endoscopic findings, and histopathological evidence of Pattern B gastric siderosis. Clinicians should suspect IPG in any patient receiving solid oral iron preparations who develops upper GI symptoms or unexplained hemorrhage, and should not assume that therapeutic doses are inherently safe.

Early recognition, prompt cessation of iron tablets, PPI therapy, and transition to liquid or intravenous iron formulations are the cornerstones of management.

**Declarations**

**Ethics Statement:** Written informed consent was obtained from all adult patients and from the guardian of the pediatric patient for publication of this case series. Patient identifying information has been anonymized in accordance with institutional guidelines.

**Conflicts of Interest:** The authors declare no conflicts of interest.

**Funding:** This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

**Author Contributions:** All authors contributed to the conception, data collection, drafting, and critical revision of this manuscript. All authors approved the final version for submission.

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