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CASE REPORT

Plummer–Vinson syndrome complicated by Schatzki ring, non-Hodgkin lymphoma, and myeloproliferative disorder: A rare case report from Pakistan

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Abstract

We report a rare case of Plummer–Vinson syndrome (PVS) in a 55-year-old female with no known premorbid condition who presented to the hospital with a chief complaint of dysphagia for 1 month, weight loss, and a history of fever (high grade up to 40°C) associated with occasional rigors and chills. Laboratory testing showed iron-deficiency anemia, together with dysphagia, raising clinical suspicion for PVS. She was further examined with upper gastrointestinal endoscopy to evaluate the cause of dysphagia, which revealed a Schatzki ring in the esophagus at 38 cm from the incisors, near the gastroesophageal junction. The ring was dilated endoscopically, and the rest of the stomach and duodenum were normal. The computed tomography scan presented hepatosplenomegaly and lymphadenopathy. Findings were consistent with a myeloproliferative disorder and non-Hodgkin lymphoma. Oral iron supplementation (150–200 mg) was recommended, and dysphagia should be evaluated with gastroduodenal endoscopy as indicated.

Keywords: Plummer–Vinson syndrome; Esophageal webs; Iron deficiency; Postcricoid dysphagia

1. Background

Plummer–Vinson syndrome (PVS) or Patterson–Kelly syndrome was first reported by Patterson and Kelly in 1919,[1] a rare disease associated with the classic triad of atrophic oral mucosa leading to dysphagia, glossitis, and an esophageal web, as well as iron deficiency and microcytic hypochromic anemia.[2] It predominantly affects middle-aged women

and is associated with an increased risk of squamous cell carcinoma of the hypopharynx and upper esophagus. The pathogenesis of PVS remains unclear; proposed contributors include genetic susceptibility, nutritional deficiencies, and autoimmune mechanisms. The most common etiologic factor is iron deficiency, which causes mucositis and leads to esophageal web formation. Due to iron deficiency, it is hypothesized that iron-dependent enzyme dysfunction leads to oxidative stress and the death of esophageal mucosal epithelial cells.[3,4]

In approximately 10% of cases, PVS is associated with cancers of the oral cavity, hypopharynx, or esophagus and is characterized by degenerative changes in these regions.[3,5,6] Iron deficiency leads to mucosal atrophy and pharyngeal muscle degradation, which forms esophageal webs.[7] Histological features of PVS are epithelial hyperplasia, fibrosis, basal cell hyperplasia, hyperkeratosis, epithelial atrophy, and chronic inflammation.[8] This syndrome is also linked with autoimmune conditions such as rheumatoid arthritis, Crohn's disease, celiac disease, thyroid disease, and immune dysregulation.[9-11]

Exact data on the incidence and prevalence of the syndrome are unavailable due to disease rarity. However, this disease was prevalent in temperate northern countries over the past century, particularly among middle-aged women. Today, nutritional improvements and iron-deficiency treatment help to control and reduce the prevalence of this disease. In addition, esophageal webs could be diagnosed with barium studies or endoscopy. However, webs could be symptomatic in some cases but not meet the criteria of PVS disease.[12]

Myeloproliferative disorders (MPDs) are a heterogeneous group of hematologic conditions characterized by clonal proliferation of myeloid lineage cells in the bone marrow, often associated with elevated peripheral blood counts and risk of progression to acute leukemia or marrow fibrosis. Commonly driven by somatic mutations, such as *JAK2 V617F*, *CALR*, or *MPL*, MPDs frequently involve systemic symptoms (e.g., fatigue, weight loss, splenomegaly) due to cytokine release and altered hematopoiesis.[13,14] Non-Hodgkin lymphoma (NHL) comprises a diverse set of lymphoid malignancies that arise from B-, T-, or natural killer-cell lineages, exhibiting variable clinical behavior and prognosis depending on the histologic subtype. Risk factors include immune suppression, chronic antigenic stimulation, and certain viral infections. In addition, genetic changes involving *BCL2*, *BCL6*, or *MYC* contribute to the pathogenesis.[15,16]

While the co-occurrence of PVS with either MPDs or NHL is rare, possible links may involve chronic inflammation, nutritional deficiencies, immune dysregulation, and iron metabolism disturbances—factors that can overlap in the pathophysiology of these conditions.[17]

First, chronic inflammation and altered cytokine signaling in MPDs or NHL may exacerbate iron sequestration and functional iron deficiency through hepcidin-mediated pathways. Second, nutritional depletion due to malignancy-associated cachexia or gastrointestinal involvement may contribute to PVS development. Third, bone marrow infiltration or suppression by neoplastic cells may synergistically worsen anemia in susceptible individuals. Moreover, immune dysregulation in lymphoproliferative and MPD may predispose mucosal epithelial changes similar to those seen in PVS.

Here, we report a rare case of a female patient having anemia and dysphagia. The dilation of the esophageal web was carried out using a gastroduodenal endoscopic procedure. The endoscopic evaluation, blood tests, diagnosis, and radiological assessment revealed that the patient has MPD and NHL.

2. Case presentation

A 55-year-old menopausal female presented to the medical emergency department of Holy Family Hospital, Rawalpindi, Pakistan, with epigastric pain and anemia with a month of dysphagia (unable to tolerate solids and liquids), weight loss of 38.1 kg, and high-grade fever (up to 40°C) with intermittent rigors and chills. The patient was treated with antipyretics, including Mucaine syrup (two teaspoons 3 times a day) and Bisleri syrup (two teaspoons twice a day), and folic acid, Qbal, and Brufen (twice a day) to relieve symptoms. She reported no associated cough, headache, or burning micturition.

On physical examination, she was significantly anemic, and her vitals were notable for a pulse rate of 93 beats/min, oxygen saturation of 98% on room air, a blood pressure of 130/90 mmHg, and a temperature of 37.8°C. Initial laboratory investigation showed microcytic anemia. She was admitted for further evaluation; the upper gastroduodenal endoscopy was performed to evaluate the cause of dysphagia, and it revealed a Schatzki ring in the esophagus at 38 cm from the incisors near the gastroesophageal junction that was auto-dilated with the endoscope, as shown in Figure 1.

Endoscopic ultrasound-guided fine-needle aspiration cytology of the large lymph nodes was performed, suggesting a monotonous population of small, atypical lymphocytes with mild nuclear abnormalities, consistent with NHL. In addition, bone marrow analysis revealed hypercellular marrow with expansion of myeloid lineages

Table 1. Different laboratory parameters with reference ranges

Blood tests	Patient value	Reference values
Complete blood picture		
Total leukocyte count	3100/mm^3	4000–10,000/mm^3
Hemoglobin	5.2 g/dL	11.5–16.5 g/dL
Platelets count	237,000/mm^3	150,000–450,000/mm^3
Total red blood cell count	3.01×10^{12}	3.8–5.8×10^{12}
Hematocrit	16.5 L/L	41–45 L/L
Mean corpuscular volume	71.1 fL	82–98 fL
Mean corpuscular hemoglobin	22.3 pg	27–31 pg
Mean corpuscular hemoglobin concentration	31.5 g/dL	32–36 g/dL
Red cell distribution width-coefficient of variation	16.4%	11–16%
Neutrophil	76%	40–75%
Lymphocyte	15%	15–45%
Monocyte	6%	2–12%
Eosinophils	3%	2–6%
Erythrocyte sedimentation rate	68 mm/hour	10–15 mm/hour
Coagulation profile		
Prothrombin time	15 s	10–16 s
Activated partial thromboplastin time	37 s	4–39 s
Renal function test		
Serum urea	27.1 mg/dL	10–52 mg/dL
Serum creatinine	0.9 mg/dL	Up to 1.2 mg/dL
Liver function test		
Total bilirubin	1.0 mg/dL	0.2–1.0 mg/dL
ALT	35 U/L	5–50 U/L
ALP	236 U/L	35–104 U/L
Serum albumin	2.4 g/dL	3.4–5.5 g/dL
Serum electrolytes		
Serum sodium	141 mmol/L	136–145 mmol/L
Potassium	4.8 mmol/L	3.5–5.1 mmol/L
Chloride	95 mmol/L	98–107 mmol/L
Calcium	7.3 mg/dL	8.6–10.2 mg/dL
Iron profile		
Serum iron	29 µg/dL	47.5–160 µg/dL
Total iron binding capacity	411 µg/dL	250–350 µg/dL
Thyroid profile		
Thyroid-stimulating hormone	4.0 µU/mL	0.34–5.60 µU/mL
Triiodothyronine (total)	0.58 ng/mL	0.97–1.70 ng/mL
Thyroxine (total)	6.75 µg/dL	5.53–11.0 µg/dL
Thyroid profile		
Malaria parasite by the immunochromatographic test method	Negative	Negative
Red blood cell morphology		
Anisocytosis	++	-
Microcytosis	+	-
Hypochromia	+	-
Spherocytes	+	-
Poikilocytosis	++	-
Macrocytosis	+	-
Target cell	+	-
Teardrop cell	+	-
Urine protein[a]	+	Negative
Urine culture	*Escherichia coli* was isolated, which was sensitive to Tazocin and Fosfomycin	Negative

Notes:+Slight/a few numbers; ++Moderate/moderate number;
[a]+corresponds to approximately 30 mg of protein per dL.

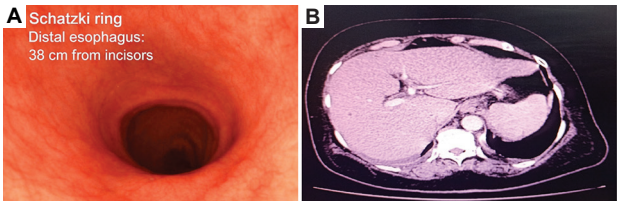

Figure 1. Endoscopic and radiologic findings demonstrating a Schatzki ring and hepatosplenomegaly. (A) Endoscopic view of a Schatzki ring at 38 cm from the incisors, near the gastroesophageal junction. (B) The computed tomography scan revealed hepatosplenomegaly.

and reduced fat spaces, findings suggestive of an underlying MPD.

The stomach and duodenum were normal. Further workups showed microcytic anemia with iron deficiency. Table 1 shows the laboratory tests performed.

The ultrasound sonography test of the abdomen showed splenomegaly, while all other findings were normal. Her computed tomography (CT) image of the chest and abdomen showed hepatosplenomegaly with lymphadenopathy. The liver size was 20 cm in craniocaudal dimension with no focal lesion, and the spleen was enlarged with a splenic index of 645 cm^3. A few subcentimeter, enlarged pre-tracheal, paratracheal, peri-cranial, subcarinal, perivascular, and hilar lymph

nodes were observed; a larger one was in the right hilar region measuring 3.2 × 2.7 × 1.6 cm (anteroposterior [AP] × transverse [TR] × craniocaudal [CC]). A few atelectatic bands were seen in the basal and inferior lingular segments. Aerated lung fields were devoid of any active infiltration.

Bilateral mild pleural effusions were noted, more on the left, with considerations of underlying basilar lung segment collapse. Multiple enlarged right axillary lymph nodes were observed, the largest measuring 13 × 10 mm. Stomach, opacified and un-opacified bowel loops were unmarkable. Opacified abdominal aorta, inferior vena cava, portal, and mesenteric vessels were normal. Multiple subcentimeter and enlarged pre-aortic, para-aortic, and aortocaval, along the celiac axis at porta-hepatis and mesenteric lymph node were seen, and among them, the largest one at porta-hepatis was measured as 2.9 × 2.7 × 2.3 cm (AP × TR × CC) in size. The CT features of hepatosplenomegaly with mediastinal, hilar, right axillary, and abdominal lymphadenopathy were suggestive of lymphoproliferative disorder. An abnormally elevated right hemidiaphragm was also noted, possibly due to eventration or right phrenic nerve palsy (Figure 1).

In our case, the patient was not treated with iron therapy and died shortly after the diagnosis of PVS; the clinical course may have been driven by underlying malignancy (MPD and NHL).

3. Discussion

PVS is a rare disease with unknown etiology, linked with iron deficiency anemia, dysphagia, and proximal esophageal web. PVS cases were reported in women and children.[18-21] The exact pathogenesis is unclear, the incidence and prevalence data are not well-defined, and many laboratory abnormalities are non-specific and are not diagnostic on their own. Factors, including malnutrition, iron deficiency, genetic predisposition, and autoimmune diseases that can occur in middle-aged women, may produce symptoms such as anemia with low-grade dysphagia similar to PVS, making its pathogenesis difficult to identify. PVS is generally associated with proximal esophageal webs, but our case is unusual in demonstrating a Schatzki ring coexisting with features of PVS. In our study, the patient was a 55-year-old female who presented with dysphagia and iron-deficiency anemia and was found to have a distal Schatzki ring.

The Schatzki ring in this case was located at 38 cm from the incisors (distal esophagus), distinguishing it from the proximal web. This anatomical position is a key distinguishing feature of a Schatzki ring, a smooth, concentric mucosal ring at the gastroesophageal junction in the distal esophagus, often associated with hiatal hernia and gastroesophageal reflux disease.[22]

In our case, the patient presented with iron deficiency anemia and dysphagia, which are classical features of PVS. Endoscopic evaluation demonstrated a distal Schatzki ring auto-dilated with an endoscope; the stomach and duodenum were normal, while the clinical findings supported the PVS diagnosis of anemia, dysphagia, and mucosal changes. Although a proximal esophageal web was not visualized in this patient, the diagnosis of PVS was supported by the hematological profile (iron-deficiency anemia) and clinical presentation. Furthermore, the case demonstrated MPD and NHL. The exact pathogenesis of PVS remains unclear due to limited data availability. Clinicians must carefully rule out coexisting or alternative malignancies to optimize disease management and treatment outcomes. The prognosis is dramatically worsened if PVS is associated with other malignant conditions, such as squamous cell carcinomas of the hypopharynx and upper esophagus. Endoscopic dilatation remains the most commonly applied treatment for esophageal webs, while surgery is not routinely recommended.[23]

A mutation in the *TMPRSS6* gene was noted in PVS, leading to iron-deficiency anemia. In iron deficiency, the synthesis of iron-dependent oxidative enzymes is compromised, promoting anaerobic metabolic activity and leading to myasthenic changes in esophageal muscles, which can progress to esophageal webs and dysphagia.[24,25] In PVS syndrome, the risk of hypopharynx or upper esophagus squamous cell carcinoma is high, which is associated with chronic iron deficiency (irreversible mucosal changes that lead to malignant degeneration). The dysphagia can be due to several conditions, including malignancy, strictures, esophageal burns, and heterotopic gastric mucosae, that should be ruled out. The patients should follow up on the upper gastrointestinal endoscopy for the diagnosis of neoplastic changes with webs. Iron-dependent metabolic pathways, such as oxidative phosphorylation, were compromised, leading to anaerobic metabolic changes in the esophageal muscle that can cause esophageal webs.[26,27]

The current case report shows that prognosis is poorer when PVS is associated with MPDs. We suggest that if iron therapy does not help in the recovery phase, other core malignancies should be investigated. Core malignancies that are not ruled out accurately may lead to patient death. A barium swallow can detect esophageal webs, but it is difficult to differentiate between web rings and tumor growth. Upper gastrointestinal endoscopy should be used as a confirmatory diagnostic procedure.[19,21]

Occult blood loss, underlying malignancies, and iron intolerance should always be ruled out. Iron-deficiency anemia can be treated with 150–200 mg of an iron supplement. Dysphagia in several patients often resolves

following iron supplementation.[4] Esophageal webs can be dilated via Savary–Gillard dilators or endoscopic balloon dilatation.[28,29] We suggest that PVS can be treated with the primary cause, not ignoring the risk of squamous cell carcinoma of the pharynx and esophagus. The patient should be followed closely, and genetic profiling is required to investigate the underlying mechanism. A follow-up upper gastrointestinal endoscopy is recommended for patients' surveillance 6 months after iron therapy. Laboratory investigations, such as hemoglobin level, mean corpuscular volume, hematocrit, serum iron level, total iron-binding capacity, and ferritin levels, should be repeated after iron therapy, which may lead to iron overload-related complications and poor treatment outcomes.

PVS is a complex disease process, and an early diagnosis is of utmost importance; patients may develop malignancies, such as MPD and NHL, and esophageal webs may develop into esophageal or pharyngeal squamous cell carcinoma. Esophageal webs of PVS with core malignancy should be ruled out.

4. Conclusion

PVS is a rare disease that generally affects middle-aged women. We presented a clinical case of a 55-year-old female diagnosed with dysphagia, MPD, and NHL. Dilatation was performed, and the patient was advised on treatment for iron-deficiency anemia. The patient died shortly after the diagnosis; the cause of death was not available. The interval between diagnosis and death was short, and iron therapy could not be initiated because of the underlying malignancy (suspected NHL and MPD). Therefore, endoscopic dilatation or incision was performed to relieve dysphagia. In conclusion, no specific and sensitive test is available for diagnosing PVS; therefore, various parameters, such as complete blood count and iron profiling (serum iron, total iron-binding capacity, and serum ferritin level), should be considered before investigating dysphagia complications. Barium esophagography, endoscopy, and video fluoroscopy can be used to detect esophageal abnormalities.

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Conflict of interest

The authors declare that they have no competing interests.

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Ethics approval and consent to participate

This case report was a part of a PhD research study, and the ethical approvals were obtained from Pakistan Institute of Nuclear Science and Technology (PINST/DC-31/2021), Islamic International University (108-FBAS/PhDBT/F-19), and Pakistan Institute of Medical Sciences (F.1-1/2015/ERB/SZABMU). Written informed consent was obtained from the patient.

Consent for participation and publication

Written informed consent was obtained from the patient's legal guardian for publication of this case report and any accompanying images.

Availability of data

The data are available from the authors upon request.

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