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# Evaluation Of Plummer-Vinson Syndrome in Pediatric Patients with Dysphagia

## Disfaji Olan Çocuk Hastalarda Plummer-Vinson Sendromunun Değerlendirilmesi

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**Abstract**

**Objective:** Dysphagia is a condition of increasing prevalence in childhood with numerous potential etiologies. Our study focuses on patients diagnosed with Plummer-Vinson syndrome (PVS), characterized by iron deficiency anemia and dysphagia, after excluding other contributing factors. We aim to highlight the importance of recognizing dysphagia cases responsive to anemia treatment and emphasize the potential for pediatricians to manage these patients effectively.

**Materials and Methods:** This study included pediatric patients presenting to our hospital with dysphagia between May 2019 and November 2020. After systematically ruling out other causes of dysphagia, patients diagnosed with PVS were evaluated retrospectively. We analyzed patient data including age, gender, dysphagia type, characteristics, and duration, and discuss these findings in the context of existing literature.

**Results**: The study included 200 pediatric patients presenting with dysphagia. Of these, 24 (14 female, 10 male) were diagnosed with Plummer-Vinson syndrome (PVS). The age range of patients was 12 to 213 months, with a mean age of 97.5 months (±72 months). The average duration of dysphagia symptoms was 11.2 months. Dysphagia characteristics were classified as follows: difficulty swallowing liquids only (n=4), solids only (n=12), or both solids and liquids (n=8). Anemia was diagnosed based on complete blood count results, with hemoglobin and hematocrit levels falling below two standard deviations for the patient's age. Cobalamin deficiency was defined as vitamin B12 levels below 200 pg/ml. Contrast esophagography revealed esophageal webs in 4 patients. Esophagogastroduodenoscopy (EGD) was performed, and web-associated stenosis was treated endoscopically in these patients. Following six months of iron deficiency anemia treatment, dysphagia resolved in 20 patients.

**Conclusion**: While Plummer-Vinson syndrome (PVS) is a less common diagnosis in pediatric populations, it remains an important consideration for children presenting with dysphagia and iron deficiency anemia. For patients whose symptoms persist despite appropriate iron supplementation, referral to a pediatric gastroenterologist is warranted for further evaluation and management.

**Keywords**: Child; Plummer-Vinson Syndrome; Iron-Deficiency Anemia; Esophageal Web, Dysafgia.

**&**

**Öz**

**Amaç**: Disfaji sıklığı çocukluk çağında giderek artmakta ve birçok nedene bağı olarak gelişmektedir. Çalışmamızda pediatrik çağda disfajinin diğer tüm nedenleri dışlandıktan sonra demir eksikliği anemisi ve disfaji olan Plummer-Vinson sendromu ( PVS) tanısı ile takip ve tedavi edilen hastalar sunulmaya çalışılmıştır. Amacımız sadece anemi tedavisi ile düzelebilecek disfaji olabileceğini ve çocuk sağlığı ve hastalıkları uzmanı tarafından tedavi ile düzelebileceğini vurgulamaktır.

**Gereç ve Yöntemler:** Hastanemizde Mayıs 2019 ve Kasım 2020 arasında disfaji şikayeti ile başvuran, disfajinin diğer tüm nedenleri dışlandıktan sonra Plummer-Vinson sendromu tanısı alan hastalar retrospektif olarak değerlendirildi. Hastaların yaş, cinsiyet, disfaji şekli, özelliği ve süresi retrospektif olarak değerlendirilerek literatür eşliğinde sunuldu.

**Bulgular**: Disfaji şikayetiyle başvuran 200 çocuk hastadan 14' ü kız 10' u erkekti, toplam 24 çocuk hastada PVS saptandı. Hastaların yaşları en küçük 12 ay, en büyük 213 ay olup ortalama yaşları 97,5 (+-72 ay) aydı. Hastaların disfaji şikayet süresi ortalama 11,2 aydı. Dört hastada sadece sıvı gıdaları, 12 hastada katı gıdaları, 8 hastada ise hem katı hem de sıvı yutma güçlüğü vardı. Tam kan sayımı yapılarak ve hemoglobin, hematokrit düzeyleri yaşa uygun 2 standart sapma değerinin altında olanlar anemi; vitamin B12 düzeyi 200 pg/ml değerinin altında olanlar kobalamin eksikliği olarak tanımlandı. Kontraslı özofagografi çekilen 4 hastada özofageal web saptandı. Web saptanan hastaların özofagogastroduodensokopide darlık endoskopik olarak tedavi edildi. Yirmi hastada ise altı aylık demir eksikliğine bağlı anemi tedavisi sonrası yutma güçlüğünün devam etmediği görüldü.

**Sonuç**: PVS tanısı çocuklarda nadir görülür, demir eksikliği anemisi ile ilişkili yutma güçlüğü çeken tüm çocuklarda akla gelmeli ve tedavi verilmelidir. Uygun dozda ve sürede tedavi sonrası şikayeti devam eden hastalar çocuk gastroenteroloji değerlendirmesi için yönlendirilmelidir.

**Anahtar Kelimeler:** Çocuk; Disfaji, Plummer-Vinson Sendromu; Özofageal Web; Demir Eksikliği Anemisi.

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## Introduction

Swallowing is a complex process enabled by the coordinated voluntary and involuntary actions of multiple structures, including the oral cavity, pharynx, larynx, and esophagus. Healthy, efficient swallowing requires the synchronized function of the oral cavity, pharyngeal muscles, cranial nerves, upper and lower esophageal sphincters, esophageal muscles, and stomach. Dysphagia, or difficulty swallowing, can result from mechanical obstruction during food transfer from the mouth to the stomach, or from decreased strength or impaired coordination of the muscles involved in the swallowing mechanism. Pediatric dysphagia can manifest at any age, often in conjunction with feeding or breathing difficulties, and potentially hindering growth and development.

First described by Plummer (1912) and Vinson (1919), Plummer-Vinson syndrome (PVS) is a triad characterized by dysphagia, iron deficiency anemia, and, in some cases, an upper esophageal web. Webs, a rare cause of upper cervical dysphagia, occur in approximately 5-15% of patients with dysphagia. While definitive data on PVS incidence and prevalence are lacking, it is considered very rare in childhood and generally presents as case reports within the literature (1,2). Improved nutrition and earlier diagnosis and treatment of iron deficiency likely contribute to the decreased incidence of PVS in children today. Dysphagia in PVS typically begins with solids and may be intermittent or progressive. Esophageal webs, thin transverse membranes extending into the esophageal lumen, can be single or multiple. While an esophageal web is not always present in PVS with iron deficiency, dysphagia remains a necessary diagnostic criterion (3).

## Materials and Methods

This study included 200 pediatric patients presenting to our hospital's Pediatric Gastroenterology outpatient clinic with dysphagia complaints between May 2019 and November 2020. We evaluated patient data, including age, gender, dysphagia type, characteristics, and duration. To assess for iron deficiency anemia and potential concomitant vitamin B12 deficiency, the following laboratory tests were performed: hemogram, iron, iron-binding capacity, ferritin, and vitamin B12 levels. Contrast esophagography was performed to identify anatomical abnormalities, and patients with non-web anatomical etiologies were excluded from the study. We also excluded pediatric patients with burns from corrosive substance ingestion, esophagitis due to infection, eosinophilic esophagitis, and heterotopic gastric mucosal lesions within the esophagus. To investigate potential causes of dysphagia and anemia, esophagogastroduodenoscopy (EGD) with duodenal and stomach biopsies was performed. Patients received oral iron supplementation at a dose of 5mg/kg/day for the treatment of anemia.

Patients were instructed to return for follow-up after six months or sooner if dysphagia symptoms recurred. Data were analyzed retrospectively and discussed in the context of existing literature.

The study was approved by the Clinical Researches Ethics Committee of Adana City Training and Research Hospital (approval date: 17.08.2023, meeting number:133, decision no:2772).

### Statistical Analysis

Descriptive statistics were calculated for numerical data, including mean, standard deviation, and median (min-max). Categorical data were expressed as percentages. A p-value of $<0.05$ was considered statistically significant.

## Results

Among the 200 children presenting with dysphagia, we diagnosed 24 with Plummer-Vinson syndrome (PVS) after excluding all other potential causes. The average duration of their dysphagia complaints was 11 months, with the shortest complaint period being 1 month and the longest being 48 months. Our study included 10 male patients (37.5%) and 14 female patients (62.5%). We classified dysphagia into three groups based on the type of food causing difficulty: solid food (12 cases), liquid food (4 cases), and both solid and liquid food (8 cases). Table 1 provides a detailed breakdown of patient characteristics within these groups.

We performed complete blood counts to assess for anemia, defined as hemoglobin and hematocrit levels two standard deviations below the age-appropriate norm. Cobalamin deficiency was defined as a vitamin B12 level

below 200 pg/ml. Out of the 24 patients with PVS, 3.5% also exhibited cobalamin deficiency. Contrast esophagography was conducted in 79% of PVS cases, revealing esophageal webs in four patients.

Esophagogastroduodenoscopy (EGD) was performed in 14 patients (58.3%) who presented with both dysphagia and anemia. This procedure aimed to diagnose and treat potential esophageal webs, as well as rule out Helicobacter pylori gastritis and pernicious anemia (other potential causes of anemia). EGD confirmed the presence of webs in 4 patients, and endoscopic stenosis dilation was performed. Slight bleeding occurred as a procedural complication, but no other complications were observed. All patients received iron deficiency anemia treatment, and those with coexisting vitamin B12 deficiency received supplementation. Patients were followed up at 3-month intervals over a 6-month period. After six months of iron deficiency treatment, patients whose dysphagia resolved were discharged from follow-up (Table 2 provides a comparison of pre- and post-treatment values). Cases with confirmed webs remained under the care of pediatric surgery.

**Table 1.**

Demographic And Clinical Features of Patients by Groups.

| | Solid dysphagia | Liquid dysphagia | Solid and Liquid dysphagia |
|---|---|---|---|
| Gender | 10 F/ 2M | 2 F/ 2 M | 2 F/ 6 E |
| Average age | 119 months | 45.5 month | 115 months |
| Contrast esophagography | 9 patients | 3 patients | 7 patients |
| Esophagogastroduodenoscopy | 9 patients | 3 patients | 2 patients |

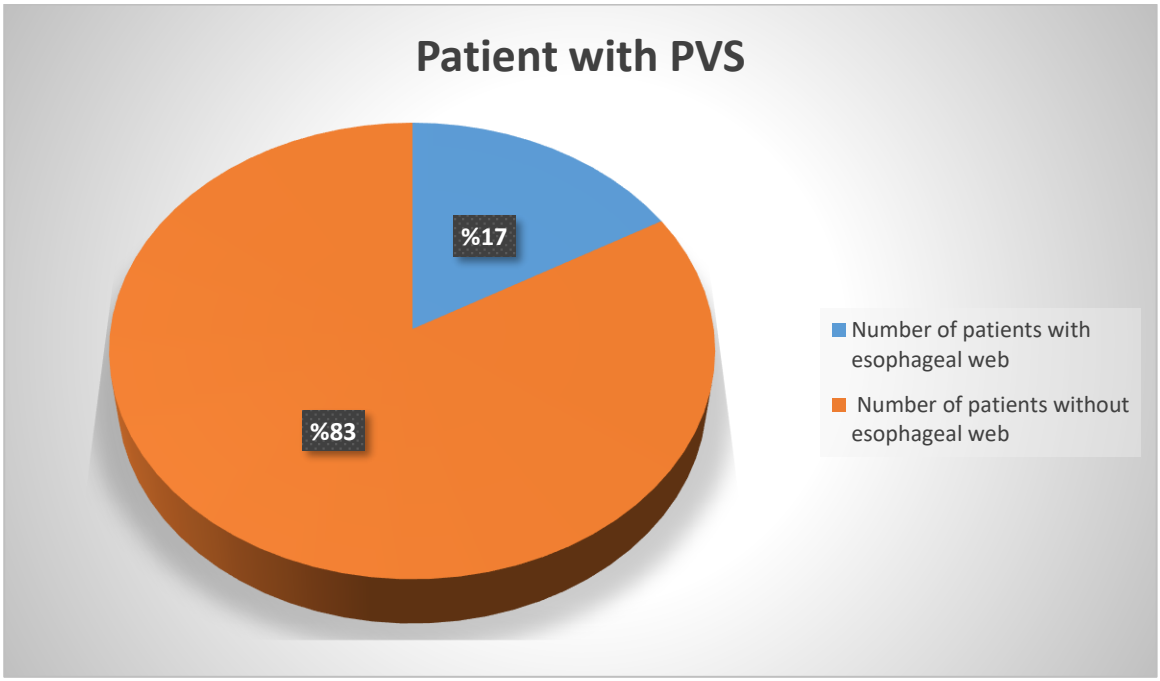


**Figure 1.** Distribution of pediatric patients with Plummer-Vinson syndrome according to webs.

## Discussion

Plummer-Vinson syndrome (PVS) is named after the physicians who contributed to its understanding. Henry Stanley Plummer first described the condition in 1912, focusing on abnormal esophageal contractions without anatomical stricture. In 1919, Porter Paisley Vinson highlighted dysphagia caused by a surgically treatable esophageal stricture. Later, British physicians Donal Ross Paterson and Adam Brown-Kelly described the classic PVS triad: dysphagia, esophageal webs, and iron deficiency anemia. Due to this historical progression, the syndrome is known as Paterson-Brown-Kelly syndrome in the United Kingdom and Plummer-Vinson syndrome in the United States (4). Plummer-Vinson syndrome (PVS) is a clinical condition characterized by the triad of dysphagia, upper esophageal webs, and iron deficiency anemia (5, 6). Esophageal webs, a less common cause of upper cervical dysphagia in children, occur in approximately 5-15% of dysphagia cases (7). They are thin mucosal membranes composed of mucosa and submucosal tissue, typically found just below the cricopharyngeal muscle within the cervical esophagus. Esophageal webs often adhere asymmetrically to the anterior esophageal wall (8). Our study aligns with findings in adult literature, where PVS predominantly affects females (89% of cases with inflammatory PVS on biopsy) (9). In our pediatric cohort, we similarly observed a higher proportion of female patients (62.5%) compared to males (37.5%). The mean age of our patients was 97.5 months, with an average dysphagia duration of 11 months. The complaint duration ranged from a minimum of 1 month to a maximum of 48 months. Dysphagia in PVS typically presents with difficulty swallowing solid foods. It is often painless and may be intermittent or progressive. In some cases, it can evolve to include difficulty with liquids (2, 10). Out of our patient cohort, contrast-enhanced radiographs could not be obtained for one patient with liquid dysphagia, three patients with solid dysphagia, and one patient with both types. EGD results did not reveal significant differences across these groups. Esophagogastroduodenoscopy (EGD) was not performed in 10 patients due to family preference: 3 patients with solid food dysphagia, 1 patient with liquid dysphagia, and 6 patients with both types. We observed no statistically significant difference in average age between the dysphagia groups. While the liquid food dysphagia group appeared younger on average, the small sample size likely contributed to the lack of statistical significance. Esophageal webs can be multiple, and dysphagia typically presents when the esophageal lumen narrows below 2 cm. However, many webs remain asymptomatic (11). All webs identified in our study were singular and significantly narrowed the esophageal lumen (approximately 80%), contributing to dysphagia with both solids and liquids. All patients maintained normal body weight and height. Unfortunately, we could not access other physical examination findings due to incomplete records within the computer system. This limitation highlights the importance of comprehensive medical record-keeping for future studies.

The precise etiopathogenesis of PVS remains elusive, with potential contributions from genetic, environmental, nutritional, immunological, and infectious factors. The iron deficiency theory posits that reduced levels of iron-dependent oxidative enzymes lead to atrophy and myopathy in pharyngeal muscles and mucosa, promoting web formation. Interestingly, despite web persistence after iron treatment, esophageal motility normalization and dysphagia improvement support this theory. However, the reason why webs composed of squamous epithelium develop in the proximal hypopharynx secondary to submucosal inflammation and fibrosis remains unclear. Regardless of the underlying trigger, iron deficiency disrupts iron-dependent enzymes crucial for cellular renewal. This disruption leads to mucosal degeneration and mesh formation. Research indicates that iron deficiency impairs esophageal motility by reducing the amplitude of muscle contractions (11, 13). Miranda and Dantas demonstrated that iron deficiency anemia patients exhibited slower esophageal transit times in the proximal and middle esophagus compared to healthy individuals, but no difference in the distal esophagus (14). This finding could explain the propensity for web formation in the proximal esophagus. While motility studies, particularly manometry, offer valuable insights into esophageal function, they can be challenging to perform in pediatric populations. Successful esophageal manometry requires patient cooperation and responsiveness to instructions, which can be particularly challenging in young children (especially those under 7 years old). Despite having patients over the age of 7 in our study (41.6%), we were unable to conduct esophageal motility tests due to a lack of this specialized equipment at our center. Our findings highlight a critical need for larger multicenter studies incorporating motility assessments to deepen our understanding of this condition in pediatric patients.

An alternative theory proposes an immunological mechanism involving the formation of autoantibodies directed against the esophagus (11). In patients with PVS, the possibility of coexisting autoimmune disorders should be investigated. These may include rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, pernicious anemia, thyroiditis, or celiac disease (15, 16, 17). The potential link between PVS and autoimmune disorders (such as rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, pernicious anemia, thyroiditis, or celiac disease) warrants careful investigation (15, 16, 17). While the precise mechanisms remain unclear, these disorders could contribute to PVS through autoantibody production or by triggering inflammatory processes (18). In our study, we evaluated thyroid function tests and celiac antibodies to screen for other potential causes of dysphagia. These tests yielded normal results. Gastric biopsies obtained during esophagogastroduodenoscopy did not indicate pernicious anemia. While other autoimmune diseases were not specifically evaluated due to an absence of suggestive clinical findings, their potential role should be considered in future studies.

PVS is a clinical diagnosis based on dysphagia associated with chronic iron deficiency anemia. While esophageal webs are a characteristic finding, they are only detected radiologically or endoscopically in approximately 10% of cases. Dysphagia is the essential diagnostic criterion (3). Contrast-enhanced radiographs, especially lateral views, are crucial for visualizing webs and potential ring-shaped stenosis (19, 20). Even in the absence of abnormalities on contrast esophagography, severe cases may still present with stenosis. Nosher et al. (1975) demonstrated this, finding 5.5% of esophagi exhibiting one or more webs in their radiographic study (21). Chisholm et al. (1974) found esophageal webs in just 10% of patients with iron deficiency anemia, which aligns with our finding of webs in 17% of PVS cases. However, among the broader population of 200 children with dysphagia, we detected webs in only 2%. This highlights the challenges of evaluating barium radiographs, particularly for upper esophageal anomalies. Difficulty coordinating studies in children and a lack of experienced teams trained in serial image evaluation further complicate the process. With improved resources and expertise, we anticipate that web detection rates will increase. Upper endoscopy can also detect esophageal webs, though they can be easily missed, and there is a risk of perforation during the procedure. Godino et al. found iron deficiency anemia in 38.5% of patients with endoscopically detected webs and low serum ferritin levels (without anemia) in an additional 0.7% of cases (22). Our findings align with this study. We performed esophagogastroduodenoscopy (EGD) in 58.3% of our PVS cases, successfully visualizing and treating webs in 4 patients where they had also been detected by contrast-enhanced radiography. Treatment strategies for PVS include iron supplementation, esophageal dilation, or a combination of both. Iron therapy should continue until hematocrit and ferritin levels normalize. For patients without complete obstruction, this approach often leads to significant dysphagia improvement. Clinical studies demonstrate that iron repletion also enhances esophageal motility. In cases of severe, longstanding dysphagia unresponsive to iron supplementation, mechanical widening of the stenosis is typically necessary, with surgical intervention rarely required (23, 24, 25). The higher rate of endoscopic dilation performed in pediatric PVS patients (64%) suggests a reduced response to iron therapy compared to adults. Often, a single dilation procedure is sufficient in children, contrasting with adult cases (26). A three-year study of 37 PVS patients found a 94% resolution of dysphagia after one endoscopic dilation, with a 10% recurrence rate over a ten-month follow-up (27). Another study reported success with Savary-Gilliard bougie dilation in 153 patients, 132 of whom had PVS. Dysphagia resolved in 90.7% of these cases after a single dilation session (28). Within our pediatric cohort, we identified 12 PVS patients who also exhibited vitamin B12 deficiency alongside iron deficiency. We detected vitamin B12 deficiency in a significant portion of our pediatric dysphagia patients (13 out of 200 cases). Notably, all 12 PVS patients exhibited both iron and vitamin B12 deficiencies, while one patient had isolated vitamin B12 deficiency. Vitamin B12 deficiency can contribute to dysphagia through mechanisms like impaired cytokine expression, reduced oxidative enzymes, white matter demyelination, and atrophy in pharyngeal muscles. Our focus on PVS patients means we did not fully evaluate the broader relationship between dysphagia and isolated vitamin B12 deficiency. This highlights an important avenue for future research. The four patients with confirmed webs received a single endoscopic dilation session alongside iron therapy; we observed only minor bleeding as a complication. We treated all other PVS cases presenting with iron deficiency anemia (without webs) using oral ferrous iron (2+ valent form) at 5 mg/kg/day for 3-6 months. Following treatment, these patients reported complete resolution of dysphagia.

In conclusion, dysphagia is an increasingly common pediatric presentation with diverse etiologies. While Plummer-Vinson syndrome (PVS) remains a rare diagnosis in children, it warrants consideration in any child

presenting with dysphagia alongside iron deficiency anemia. Patients whose dysphagia persists despite six months of appropriate iron therapy should be referred to a pediatric gastroenterologist for further evaluation and management.

**Ethics Committee Approval**: The study was approved by the Adana City Training And Research Hospital Clinical Research Ethics Committee (date: 17.08.2023 and approval number: 133/2772).

**Informed Consent**: Written consent was obtained from the participants.

**Conflict of Interest**: Authors declared no conflict of interest.

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