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Patient with Dyspnea, Dysphagia and Menorrhagia: Plummer — Vinson Syndrome Against the Background of von Willebrand Disease

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Aim: to present a clinical observation of a patient with Plummer — Vinson syndrome against the background of von Willebrand disease.
Key points. A 40-years-old woman presented to the hematology department of our hospital with fatigue, dizziness, dyspnea on exertion and dysphagia to solid food. Laboratory examination showed microcytic and hypochromic anemia (haemoglobin — 2,0 g/dL; red blood cells — 1.31 × 10^{12}/L), a reduction in serum levels of ferritin to 1.5 ng/mL and a decrease in the von Willebrand factor activity up to 32.4 %. According to esophagogastroduodenoscopy the lumen of the upper esophagus third was circularly narrowed to 8 mm by web formation. Combining these clinical symptoms and examinations, we made the diagnosis of von Willebrand disease and Plummer — Vinson syndrome. Iron supplementation therapy and replacement therapy with von Willebrand factor concentrate were prescribed. At the time of discharge, the patient noted a significant improvement in her general condition, the number of red blood cells was 3.9 × 10^{12}/L, haemoglobin — 8.6 g/L.
Conclusion. Plummer — Vinson syndrome is a rare manifestation of iron deficiency anaemia and therefore can often be overlooked by physicians. The cause of deep tissue iron deficiency in our patient was chronic blood loss against the background of von Willebrand disease. Our literature review did not reveal similar cases of concomitant von Willebrand disease with Plummer — Vinson syndrome.
Keywords: case report, esophageal dysphagia, Plummer — Vinson syndrome, von Willebrand disease, menorrhagia
Conflict of interest: the authors declare no conflict of interest.

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Пациентка с одышкой, дисфагией и меноррагией: синдром Пламмера — Винсона на фоне болезни Виллебранда

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Цель: представить клиническое наблюдение пациентки с синдромом Пламмера — Винсона на фоне болезни Виллебранда.
Основные положения. Пациентка П., 40 лет, поступила в гематологическое отделение Челябинской областной клинической больницы с жалобами на выраженную общую слабость, головокружение и одышку при минимальной физической нагрузке, дисфагию при проглатывании твердой пищи, нарушение менструального цикла по типу меноррагии. По данным лабораторного обследования выявлены гипохромная микроцитарная анемия тяжелой степени (эритроциты — 1,31 × 10^{12}/л, гемоглобин — 20 г/л), снижение ферритина до 1,5 нг/мл, сывороточного железа до 2,6 мкмоль/л, снижение активности фактора Виллебранда до 32,4 %. В связи с жалобами на дисфагию проведена эзофагогастродуоденоскопия, по данным которой просвет пищевода в области устья циркулярно сужен до 0,8–0,9 см на протяжении 0,4 см. На основании клиники и результатов дообследования пациентке выставлен диагноз болезни Виллебранда, вторичной железодефицитной анемии на фоне гиперполименореи и синдрома Пламмера — Винсона. На фоне стационарного лечения больная

отметила значительное улучшение общего состояния и регресс дисфагии. По данным общего анализа крови на момент выписки количество эритроцитов составило 3,9 × 10^{12}/л, гемоглобина — 86 г/л. Пациентке даны рекомендации по продолжению приема препаратов железа, с целью заместительной терапии назначен VIII фактор свертывания крови и фактор Виллебранда во время менструаций и оперативных вмешательств.
Заключение. Синдром Пламмера — Винсона является редким проявлением железодефицитной анемии, в связи с чем часто может упускаться из виду практикующими врачами. Причиной глубокого тканевого дефицита железа у нашей пациентки послужила хроническая кровопотеря на фоне болезни Виллебранда. Проведенный нами обзор литературы не выявил похожих случаев сочетания синдрома Пламмера — Винсона и болезни Виллебранда.
Ключевые слова: клинический случай, пищеводная дисфагия, синдром Пламмера — Винсона, болезнь Виллебранда, меноррагия
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Introduction

Plummer — Vinson syndrome, also known as Paterson — Brown — Kelly syndrome or sideropenic dysphagia, is a rare condition that presents with a triad of symptoms: dysphagia, iron deficiency anemia and webs in the upper third of the esophagus. The first description of dysphagia in a female patient with anemia was described by Henry Stanley Plummer in 1914, then in 1922 Porter Paisley Vinson supplemented Plummer's observations, in connection with which the syndrome was called "Plummer — Vinson syndrome" or "dysphagia associated with anemia" [1]. Most often, this syndrome is associated with concomitant diseases that cause iron deficiency anemia, especially due to chronic blood loss or impaired iron absorption.

Von Willebrand disease, first described by Erik von Willebrand in 1926, is currently considered the most common inherited bleeding disorder [2]. Von Willebrand disease is caused by quantitative deficiency and/or qualitative defects of von Willebrand factor — a glycoprotein with many functions that generally promotes platelet adhesion to vessel walls after vessel injury, also von Willebrand factor serves as a carrier protein for factor VIII. Bleeding history associated with von Willebrand disease leads to chronic blood loss and as a consequence to iron deficiency anemia. According to L. Srivaths et al. [3], in 60 % of patients with reduced activity of von Willebrand factor, iron deficiency anemia is registered, despite this, our literature analysis did not reveal any reported cases of Plummer — Vinson syndrome in patients with von Willebrand disease.

Case report

A 40-years-old woman presented to the Hematology Department of Chelyabinsk Regional Clinical Hospital with fatigue, dizziness, dyspnea on exertion, dysphagia to solid food, tachycardia, menorrhagia, 15 kg weight loss for 3 years and tinnitus. Her medical history revealed iron deficiency anemia for more than 20 years, but haemoglobin concentration was not monitored over the past five years, and she did not receive iron supplementation therapy. During a detailed collection of complaints, the patient noted that over the past two years she periodically began to experience a feeling of obstruction when passing a food bolus at the level of the upper third of the esophagus, but this symptom did not progress. During the week before hospitalization, she noted increasing fatigue, increased dyspnea, and dizziness. She turned to the emergency room of our hospital. The patient's blood test showed microcytic and hypochromic anemia (haemoglobin — 2.0 g/L; red blood cells — 1.31 × 10^{12}/L) with white cell count of 3.95 × 10^{9}/L and platelet count of 630 × 10^{9}/L. The patient was hospitalized in the hematology department with a diagnosis of severe iron deficiency anemia. According to personal and family history, the patient had heavy and prolonged menstrual bleeding from the onset of menarche. The patient's mother and grandmother also had menorrhagia since childhood. No data for alimentary iron deficiency were found during the survey. A physical examination revealed a general condition of moderate severity; the body mass

index was 18 kg/m^2. Visible mucous membranes were pale, dry skin was noted. Vital function assessment revealed increased respiratory rate to 20 per minute, heart rate to 94 beats per minute, systolic blood pressure was 110 mmHg, diastolic blood pressure was 70 mmHg. Physical examination of the digestive and genitourinary systems did not reveal significant abnormalities. Laboratory examination showed a reduction in serum levels of ferritin to 1.5 ng/mL, serum iron to 2.6 μmol/L and an increase in serum erythropoietin concentration to 750 mU/mL with normal levels of vitamin B_{12} and folic acid. A decrease in the von Willebrand factor activity up to 32.4 % was revealed. No abnormality was found in liver and renal function tests, coagulation panel, platelet aggregation studies and routine urine analyses. Electrocardiography data revealed sinus tachycardia with a heart rate of 94 beats per minute. According to the ultrasound examination of the abdominal organs, moderate diffuse changes in the liver were recorded. On the first day of hospitalization, the patient received a transfusion of red blood cells and was prescribed iron protein succinylate. Esophagogastroduodenoscopy was performed to evaluate her dysphagia. According to esophagogastroduodenoscopy, the lumen of the esophagus at the near upper esophageal sphincter was circularly narrowed to 8 mm by web formation, and a disturbed passage of the scope was noted (Fig. 1).

A barium swallow study was performed, which revealed persistent narrowing of the upper esophagus third at the level of the C_{VII} vertebra, over 7 mm, the contrast did not linger at this level (Fig. 2). According to the results of the neck soft tissues multispiral computed tomoangiography no signs of mass formation were revealed.

Combined with the patient's medical history, the final diagnosis was von Willebrand disease, secondary iron deficiency anemia and Plummer — Vinson syndrome. Iron supplementation therapy was started. Replacement therapy with von Willebrand factor concentrate and coagulation factor VIII were prescribed during menstruation and surgical interventions. At the time of discharge, the patient noted a significant improvement in her general condition, the number of red blood cells was 3.9×10^{12}/L, haemoglobin — 8.6 g/L, and platelets — 163×10^9/L. During 1 year of observation of the patient, no adverse events or side effects to the therapy were recorded. In the course of the therapy, the patient's dysphagia resolved, but according to the results of repeated esophagogastroduodenoscopy

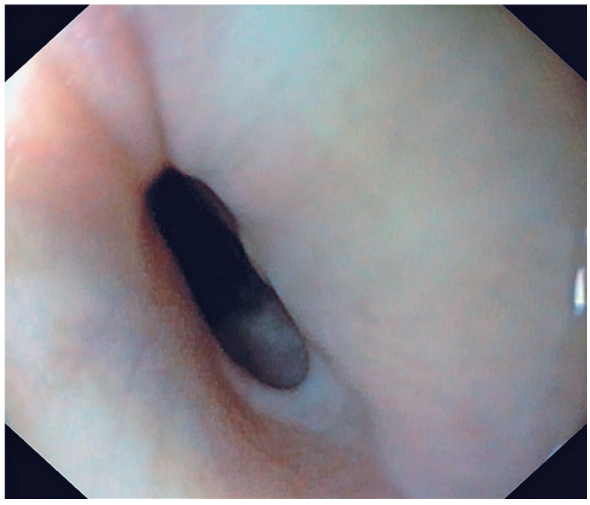

Figure 1. Results of the patient's esophagogastroduodenoscopy (circular narrowing of the lumen of the esophagus in the area of the mouth up to 0.8 cm due to the formation of a connective tissue membrane)

Рисунок 1. Результаты эзофагогастродуоденоскопии пациентки (циркулярное сужение просвета пищевода в области устья до 0,8 см за счет образования соединительнотканной мембраны)

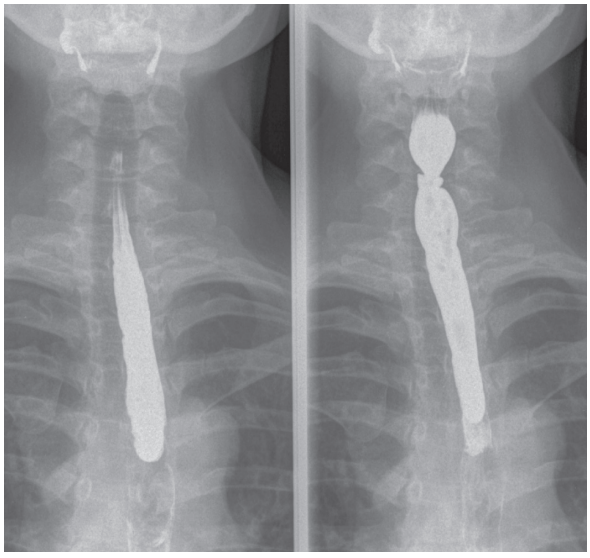

Figure 2. Results of X-ray examination of the patient's esophagus (persistent narrowing of the upper third of the esophagus at the level of the C_{VII} vertebra up to 6 mm, over a length of 7 mm)

Рисунок 2. Результаты рентгеноскопии пищевода пациентки (стойкое сужение верхней трети пищевода на уровне C_{VII} позвонка до 6 мм на протяжении 7 мм)

1 year after hospitalization, a thin transparent circular web is visualized in the upper esophagus third, which does not narrow the lumen of the esophagus.

Discussion

The above-described clinical case provides an opportunity to perform differential diagnosis for several therapeutic syndromes: hypoxic, sideropenic, hemorrhagic, and such a polyetiological symptom as dysphagia. On admission to the hospital, the predominant clinical syndromes of our patient were sideropenic and hypoxic, the cause of which was iron deficiency anemia. According to modern concepts, the main mechanisms leading to iron deficiency anemia include blood loss, impaired iron absorption, increased need for iron, and insufficient intake with food. The predominant mechanism in our clinical case was blood loss. Given the long history of menorrhagia, in the absence of gynecological diseases and other causes of iron deficiency anemia, the patient underwent additional examination to exclude hemorrhagic diathesis, according to the results of which a decrease in the level of von Willebrand factor was recorded. The most common clinical manifestations of von Willebrand disease are epistaxis, bleeding after tooth extraction, easy bruising, gingival bleeding and excessive post-traumatic or surgical bleeding. According to literature data, which is confirmed by the given clinical case, for women the most frequent, and sometimes the only clinical manifestation, especially in the first type of von Willebrand disease, is heavy menstrual bleeding. According to various studies, the frequency of von Willebrand disease in patients with menorrhagia ranges from 6.6 to 20 % [4–6].

Against the background of blood transfusions and ferrotherapy, the patient's anemia symptoms significantly regressed, but complaints of dysphagia persisted. At the first stage of the differential diagnosis of esophageal dysphagia, it is necessary to exclude mechanical causes: obstruction of the esophagus by a foreign body, narrowing of its lumen due to inflammation, fibrosis or tumor process and compression of the esophagus from the outside. The primary and most informative diagnostic method in this clinical situation is an endoscopic examination of the esophagus, and, if necessary, barium swallow study of the esophagus. Esophagogastroduodenoscopy made it possible to record a thin connective tissue membrane in the upper third of the esophagus, which, together with the presence of anemia and deep sideropenia, made it possible to confirm Plummer — Vinson syndrome. It is worth noting that histological examination of esophageal biopsy specimens has no diagnostic value for confirming Plummer — Vinson syndrome but may be necessary to exclude other causes of esophageal dysphagia, such as eosinophilic esophagitis, infectious esophagitis, and neoplastic processes.

Exact data about prevalence of Plummer — Vinson syndrome are not available, but there is currently a downward trend in the incidence of Plummer — Vinson syndrome in developed countries, which is probably associated with a decrease in the prevalence of iron deficiency anemia and improved nutritional status. According to case reports, Plummer — Vinson syndrome is often associated with the presence of comorbidities in patients, which contributes to the development of iron deficiency anemia. The largest number of Plummer — Vinson syndrome observations was recorded in patients with Crohn's disease, colorectal cancer, factor VII deficiency, amyloidosis and celiac disease [7–12]. Despite the above data, a thorough analysis of the Pubmed search engine did not record clinical cases of Plummer — Vinson syndrome in patients with von Willebrand disease.

The pathogenesis of Plummer — Vinson syndrome is not fully understood. Proposed causal mechanisms include iron deficiency anemia, poor nutrition, genetic predisposition, autoimmune inflammation, and pyridoxine and riboflavin deficiencies. It is suggested that the appearance of connective tissue membranes in the upper third of the esophagus is associated with a decrease in the activity of iron-dependent oxidative enzymes, which leads to gradual dystrophy of the pharyngeal muscles, atrophy of the overlying mucosa, and the appearance of membranes [13]. This hypothesis is indirectly supported by the rapid regression of dysphagia in patients with Plummer — Vinson syndrome receiving iron therapy [1], but it does not explain why connective tissue membranes are localized in the upper third of the esophagus. These mucosal changes in the postcricoid esophagus probably arise due to a higher risk of injury by a solid bolus of the upper esophagus and an unfortunate topographic location: anterior and posterior bone elements [13]. An alternative explanation for the formation of membranes is the possible existence in some patients of ectopia of the gastric mucosa in the upper esophagus, the secretion of acid from which causes local inflammation and the formation of stricture. However, this hypothesis does not explain the absence of membranes in the lower esophagus, given that the mucous membrane of the lower third of the esophagus is more often and intensely exposed to hydrochloric acid [14]. An indirect confirmation of the autoimmune genesis of this disease is the frequent combination

of Plummer — Vinson syndrome and other autoimmune disorders, including Sjögren's syndrome, rheumatoid arthritis and celiac disease [15, 16]. In general, it is most likely that the formation of connective tissue membranes has a multifactorial nature and the question of which factor plays the main role in the pathogenesis of this condition remains open.

Interest in such a rare disease as Plummer — Vinson syndrome is also associated with its high potential for malignancy. The strongest association has been established between Plummer — Vinson syndrome and the development of squamous cell carcinoma of the esophagus [17]. Therapy for Plummer — Vinson syndrome is primarily aimed at eliminating the cause of anemia, iron supplementation therapy, and using endoscopic treatment methods. Endoscopic methods of therapy for Plummer — Vinson syndrome include the use of endoscopic balloon dilation, Savary — Gilliard dilators, endoscopic laser dissection, and electrodissection of strictures [18–20].

Conclusion

Plummer — Vinson syndrome is a rare manifestation of iron deficiency anemia and therefore can often be overlooked by physicians. The cause of deep tissue iron deficiency in our patient was chronic blood loss against the background of von Willebrand disease and the long absence of preventive examinations and von Willebrand factor replacement therapy. Our literature review did not reveal similar cases of concomitant von Willebrand disease with Plummer — Vinson syndrome.

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