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ARTHRALGIA, WEIGHT LOSS, ANEMIA, AND SUSPECTED PANNICULITIS: A PROLONGED DIAGNOSTIC JOURNEY TO WHIPPLE DISEASE

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ABSTRACT

Introduction. Whipple disease (WD) is an extremely rare systemic infectious disease caused by *Tropheryma whipplei*, with an estimated prevalence of approximately one case per million people. Due to its multisystem involvement and nonspecific clinical manifestations, WD can mimic a wide range of gastrointestinal, rheumatological, infectious, and other disorders, often resulting in substantial diagnostic delay. The aim of this report was to describe and analyze a case of classical WD with a prolonged diagnostic course and several atypical manifestations and to identify factors that may have contributed to the delayed diagnosis.

Materials and Methods. We present the clinical case of a 45-year-old woman with chronic diarrhea, arthralgia, progressive weight loss, anemia, systemic inflammation, lymphadenopathy, and clinically suspected panniculitis. The approximately 3.5-year clinical course was analyzed together with laboratory findings, computed tomography, upper and lower gastrointestinal endoscopy, and histological examination of duodenal biopsy specimens. Cardiac and neurological assessments, including echocardiography and brain magnetic resonance imaging, were performed to evaluate possible extraintestinal involvement.

Results. The initial manifestation was persistent non-bloody diarrhea, which was subsequently accompanied by fever, arthralgia, painful subcutaneous nodules, marked weight loss, and progressive iron-deficiency anemia. Computed tomography revealed retroperitoneal and mesenteric lymphadenopathy. Initial upper gastrointestinal endoscopy, performed without duodenal biopsy, did not establish the diagnosis. Repeat esophagogastroduodenoscopy revealed duodenal mucosal edema, granularity and hyperemia, thickening of the mucosal folds, and marked villous alterations. Histological examination of duodenal biopsy specimens demonstrated focal aggregates of macrophages with abundant eosinophilic cytoplasm containing PAS-positive granules. In combination with the characteristic clinical presentation, these findings supported the diagnosis of classical WD. Cardiac and central nervous system involvement was not detected. Panniculitis was clinically suspected but was not confirmed by histological or other specific investigations. Treatment was initiated with intravenous ceftriaxone for 14 days, followed by oral doxycycline and hydroxychloroquine. After four weeks of therapy, the patient reported substantial symptomatic improvement.

Discussion. The approximately 3.5-year diagnostic delay was likely related to the extreme rarity of WD, the nonspecific and multisystem nature of its manifestations, the patient's female sex, which is less typical of classical WD, previous courses of antimicrobial therapy associated with partial and temporary improvement, and the absence of duodenal biopsy during the initial endoscopic examination. This case demonstrates that the absence of characteristic macroscopic duodenal abnormalities on endoscopy does not exclude WD. The combination of persistent diarrhea, arthralgia, weight loss, manifestations suggestive of malabsorption, anemia, and lymphadenopathy should increase clinical suspicion of the disease.

Conclusions. Whipple disease should be considered in the differential diagnosis of patients with prolonged, unexplained gastrointestinal and systemic manifestations, particularly when diarrhea, arthralgia, weight loss, malabsorption, anemia, and lymphadenopathy occur in combination. Duodenal biopsy remains a cornerstone of diagnosis and should be considered even when the endoscopic appearance of the duodenal mucosa is normal or nonspecific.

Keywords: Whipple disease, Tropheryma whipplei, diarrhea, arthralgia, panniculitis, duodenal biopsy, diagnostic delay.

INTRODUCTION

Whipple disease (WD) is an extremely rare infectious disease, occurring in approximately one person per million [1]. More than 80 years elapsed from its first description by George Hoyt Whipple in 1907 [2] to the identification of its etiological agent, the bacterium *Tropheryma whipplei* [3], which causes multisystem involvement with highly variable clinical manifestations [4]. Currently, four main clinical forms of WD are recognized [1]: classical WD (characterized by arthralgia, weight loss, diarrhea, and abdominal pain), localized chronic infection (predominantly endocarditis), acute infection (e.g., pneumonia or gastroenteritis), and asymptomatic carriage. None of these forms is specific, and each may mimic a wide range of diseases, from irritable bowel syndrome and celiac disease to rheumatoid arthritis and dementia [5]. Therefore, the diagnosis of WD is often challenging and requires a comprehensive diagnostic approach involving a multidisciplinary team [1, 4].

AIM

The aim of this study was to describe and analyze a clinical case of the extremely rare Whipple disease, the prolonged diagnostic journey leading to the final diagnosis, the potential barriers to and causes of delayed diagnosis, and possible strategies for addressing these challenges.

MATERIALS AND METHODS

This case report describes a 45-year-old woman with a prolonged course of gastrointestinal and systemic symptoms. The clinical history, laboratory findings, imaging studies, endoscopic examinations, and histological assessment of duodenal biopsy specimens were analyzed. Additional cardiac and neurological examinations were performed to assess possible extraintestinal involvement of Whipple disease.

RESULTS

Clinical case. A 45-year-old woman first sought medical attention in August 2022 with complaints of non-bloody diarrhea that had persisted for more than 2 months. She attributed the symptoms to stress that began following the onset of Russia's full-scale invasion of Ukraine. Initial investigations were unremarkable, except for a positive fecal *H. pylori* antigen test. Accordingly, a preliminary diagnosis of chronic *H. pylori*-associated gastritis and functional diarrhea was established, and standard triple eradication therapy was prescribed: pantoprazole 40 mg twice daily, amoxicillin 1000 mg twice daily, and clarithromycin 500 mg twice daily for 14 days. The treatment resulted in partial symptomatic relief but did not lead to complete resolution of the symptoms.

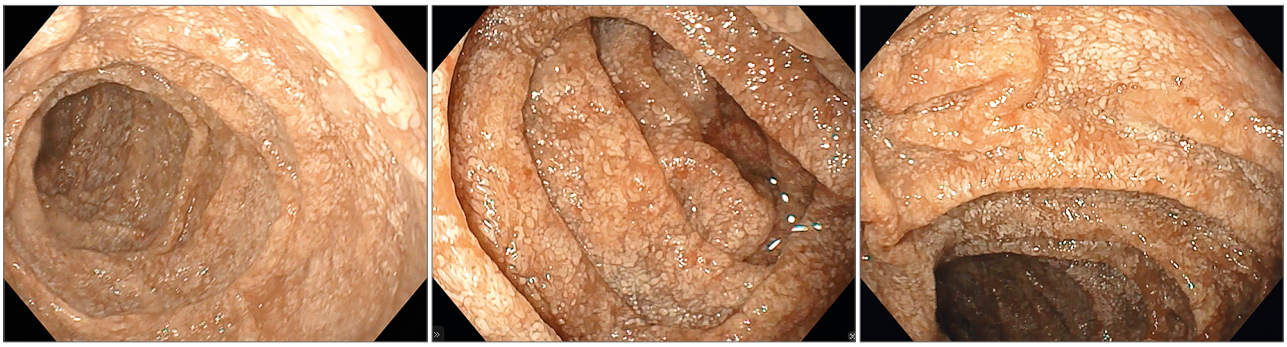
Given the partial response to the initial treatment, the patient did not seek further medical attention until August 2024, when she developed fever up to 38°C, increased stool frequency, painful skin nodules, knee arthralgia, and weight loss from 75 to 57 kg over 3 months. Further investigations revealed hemoglobin 87 g/L, MCV 70 fL, MCH 22.5 pg, platelet count 461 × 10^9/L, ESR 30 mm/h, ferritin 33.5 ng/mL, transferrin saturation 4.51%, C-reactive protein (CRP) 64.4 mg/L, and fecal calprotectin 619 μg/g. Abdominal computed tomography revealed retroperitoneal and mesenteric lymphadenopathy.

Given the diagnosis of iron-deficiency anemia and evidence of inflammation, esophagogastroduodenoscopy (EGD) and colonoscopy were performed without biopsy. The examinations revealed erythematous gastropathy, an axial hiatal hernia, and hemorrhoids, which unfortunately did not bring the diagnostic process closer to identifying the underlying disease. Subsequent testing for intestinal infections, including *Yersinia enterocolitica*, *Clostridioides difficile*, HIV, and tuberculosis, was negative. An infectious disease specialist diagnosed an unspecified gastrointestinal infection, and empirical treatment with rifaximin 1200 mg/day was initiated, resulting in partial symptomatic improvement.

The patient took rifaximin several times until July 2025, when repeat bacteriological stool testing revealed *Klebsiella pneumoniae*, and treatment with trimethoprim/sulfamethoxazole 960 mg/day for 10 days was initiated. This therapy also provided only partial symptomatic relief.

However, in February 2026, the patient was hospitalized because of exacerbation of diarrhea and severe anemia: erythrocyte count 3.6 × 10^{12}/L, hemoglobin 71 g/L, hematocrit 21.3%, MCV 59 fL, MCH 19.6 pg, platelet count 439 × 10^9/L, ESR 30 mm/h, ferritin 5.7 ng/mL, and transferrin saturation 3.8%. A blood transfusion was performed, and repeat EGD and colonoscopy were recommended. Colonoscopy revealed no clinically significant abnormalities, except for a tubular adenoma in the ascending colon. In contrast, EGD revealed several abnormalities in the postbulbar portion of the duodenum (D1-D2): purulent contents, mucosal edema, granularity, hyperemia, thickening of the mucosal folds, and marked thickening and shortening of the villi (Fig. 1).

Histological examination of duodenal biopsy specimens revealed focal, compact aggregates of macrophages with abundant eosinophilic cytoplasm containing PAS-positive granules in the lamina propria of the duodenal mucosa, as well as lymphohistioplasmacytic infiltration with eosinophils and neutrophils. No histological features of celiac



A. B. C.

Fig. 1. Endoscopic images of the duodenal mucosa before treatment

Table 1. Chronology of the Clinical Case

Date/Period	Summaries of visits	Results of diagnostic tests
August 2022	tress, chronic non-bloody diarrhea (presumed functional). Triple *H. pylori* eradication therapy with partial symptomatic improvement.	Positive stool *H. pylori* antigen test.
August 2024	Fever, chronic non-bloody diarrhea, painful skin nodules, arthralgia, and weight loss. Diagnosis of unspecified gastrointestinal infection. Rifaximin therapy with partial symptomatic improvement.	Hemoglobin 87 g/L, platelet count 461 × 10^9/L, ESR 30 mm/h, ferritin 33.5 ng/mL, transferrin saturation 4.51%, CRP 64.4 mg/L, fecal calprotectin 619 μg/g. CT: retroperitoneal and mesenteric lymphadenopathy. Endoscopy: erythematous gastropathy and hemorrhoids. Stool tests for intestinal infections were negative.
July 2025	Suspected gastrointestinal infection associated with *Klebsiella pneumoniae*. Trimethoprim/sulfamethoxazole therapy with partial symptomatic improvement.	Positive stool culture for *Klebsiella pneumoniae*.
February 2026	Worsening diarrhea and anemia. Blood transfusion. Suspected panniculitis. Diagnosis of Whipple disease. Treatment with ceftriaxone, doxycycline, and hydroxychloroquine initiated.	Erythrocyte count 3.6 × 10^{12}/L, hemoglobin 71 g/L, platelet count 439 × 10^9/L, ESR 30 mm/h, ferritin 5.7 ng/mL, transferrin saturation 3.8%. EGD: purulent contents, mucosal edema, granularity, hyperemia, thickening of mucosal folds, and marked thickening and shortening of duodenal villi. Duodenal biopsy: PAS-positive macrophages.
March 2026	Symptomatic improvement. Doxycycline and hydroxychloroquine continued for 12 months.	

disease were identified. Thus, based on the classical clinical manifestations and characteristic histological findings, a definitive diagnosis of WD was established.

Given the possibility of extraintestinal manifestations of WD, cardiac and neurological assessments were performed, including brain MRI and echocardiography; no involvement of these systems was detected. Examination of the skin by a gastroenterologist revealed a painful induration of the skin of the left arm, which was clinically consistent with panniculitis. However, additional investigations to confirm or exclude this manifestation and establish its association with *T. whipplei* were not performed.

The patient was started on ceftriaxone 2 g once daily intravenously for 14 days, followed by oral doxycycline 100 mg twice daily and oral hydroxychloroquine 200 mg twice daily. The patient's baseline weight was 61 kg, and an ophthalmological examination performed before treatment initiation revealed no ocular abnormalities. After 4 weeks of therapy, the patient reported symptomatic improvement. Continued treatment for 12 months was recommended, followed by cardiological and ophthalmological follow-up given the potential adverse effects of the therapy. Long-term treatment outcomes are awaited and were not available at the time of writing this article.

The chronology of this clinical case is presented in Table 1.

DISCUSSION

We described a case of classical WD presenting with diarrhea, weight loss, arthralgia, and anemia in

a middle-aged woman. In our case, the diagnostic delay was approximately 3.5 years from symptom onset. This is relatively typical for this disease, with published data indicating a diagnostic delay of 3-7 years [6]. Several factors may contribute to this delay, ranging from limited physician awareness due to the extreme rarity of WD to its ability to mimic various gastrointestinal, rheumatological, neurological, and other disorders [4, 6].

It should be noted that classical WD occurs predominantly in men [1], which differs from our case and may also have contributed to the delayed diagnosis. In addition, our patient received *H. pylori* eradication therapy containing two antibiotics, which may have temporarily improved the clinical and endoscopic manifestations of the disease. Consequently, the initial endoscopic examination showed no apparent abnormalities and did not bring us closer to the diagnosis. However, one finding that should have raised clinical suspicion was retroperitoneal and mesenteric lymphadenopathy, which may be present in approximately 50% of patients with WD [7].

Another important manifestation is arthralgia. Joint involvement may occur in up to 90% of patients with WD [1, 8]. However, the nonspecific nature of these symptoms, particularly in the context of fever and systemic inflammation, represents a limiting factor in the diagnostic evaluation. Subsequent treatment of our patient with several courses of antibacterial agents (rifaximin and trimethoprim/sulfamethoxazole) produced some clinical improvement but ultimately prolonged the diagnostic pathway to the correct final diagnosis.

Unfortunately, there are no specific clinical guidelines for the diagnosis and treatment of WD. According to the diagnostic algorithms presented in UpToDate [9] and cohort studies [10], clinical suspicion based on characteristic manifestations and exclusion of more common diseases (infections, inflammatory bowel disease, etc.) should be followed by EGD with duodenal biopsy. It should be emphasized that visible endoscopic abnormalities may be absent in more than 70% of patients [10], which may further contribute to diagnostic delay.

Classical macroscopic findings of WD include enlarged or broadened villi, dilated lymphatic vessels, and duodenal mucosal edema, as well as signs of duodenitis [10]. In our case, the second endoscopic examination revealed similar abnormalities, which finally led to the correct diagnosis. However, the initial endoscopic examination was unremarkable, which did not exclude WD. Therefore, duodenal biopsy is essential for confirming the diagnosis [5, 9, 10].

The most characteristic histological finding is the presence of PAS-positive macrophages in the duodenal mucosa [5, 9]. According to the available literature [4, 9, 10], identification of this finding may be sufficient to establish a definitive diagnosis of WD when there is a strong clinical suspicion based on symptoms and other clinical manifestations. Additional diagnostic tests may include PCR analysis of duodenal biopsy specimens or other biological materials, such as cerebrospinal fluid, as well as immunohistochemical testing for *T. whipplei*. These tests may be useful for confirming the diagnosis, particularly in equivocal cases or when PAS-positive macrophages are absent, and should be performed whenever feasible [1, 4, 5, 9].

It should also be noted that PAS-positive macrophages may occur in other diseases, including tuberculosis. However, PCR and IHC testing are not widely available and, unfortunately, are not accessible in Ukraine, which represents a limitation of this case report. In addition, we decided not to perform further testing to exclude rare causes of PAS-positive macrophages in the duodenum, given the patient's classical clinical presentation.

The presence of extraintestinal manifestations may also contribute to clinical suspicion of WD [1, 9]. In addition to the manifestations described above, our patient had a painful induration of the skin of the left arm that was clinically consistent with panniculitis. Cutaneous manifestations of WD are frequently overlooked [11], whereas panniculitis may represent a relatively specific manifestation of the disease. Only 15 cases of panniculitis associated with WD have been described in the literature [11]. However, in our case, this finding was not confirmed, and differential diagnosis of other potential causes of painful cutaneous nodules should be performed with the involvement of a dermatologist and rheumatologist. This represents an additional limitation of our case report.

There is a limited number of randomized studies on the treatment of WD [12], which depends on the clinical presentation and the presence of extraintestinal involvement [9]. In patients with classical WD without endocarditis or central nervous system (CNS) involvement, as in our case, treatment with oral doxycycline and hydroxychloroquine for 12 months is recommended [9]. In cases involving CNS disease or endocarditis, treatment is recommended to begin with intravenous ceftriaxone or penicillin G, followed by trimethoprim/sulfamethoxazole for 12 months [9].

Given the unavailability of cerebrospinal fluid PCR testing to completely exclude CNS invol-

vement, as well as the possibility of subclinical CNS disease, we decided to initiate treatment with intravenous ceftriaxone for 2 weeks, followed by oral doxycycline and hydroxychloroquine. It should be noted that the most recent randomized study [12] demonstrated that oral therapy was non-inferior to sequential intravenous-to-oral treatment in patients with classical WD, even in the presence of CNS involvement. However, further randomized studies are needed to expand these findings and confirm the above conclusions, particularly for other clinical forms of WD.

Unfortunately, there is no national registry of patients with WD in Ukraine; therefore, the exact incidence and prevalence of the disease in the country are unknown. We identified only a few cases reported in the literature [13, 14]. Naturally, not all cases are described and published. Moreover, given the numerous barriers to establishing the correct diagnosis, the actual number of patients with WD may be considerably higher. Patients may pass from one physician to another without receiving a definitive diagnosis, appropriate treatment, or resolution of their underlying clinical problem.

CONCLUSIONS

We described a case of classical WD with a prolonged diagnostic journey before the correct diagnosis was established and appropriate treatment initiated. Given its numerous multisystem manifestations, WD should be considered in patients presenting with diarrhea, arthralgia, weight loss, clinical signs suggestive of malabsorption, lymphadenopathy, clinically suspected panniculitis, and other extraintestinal manifestations. Duodenal biopsy is an essential component of the diagnostic process and should be considered even when the endoscopic appearance of the duodenal mucosa is normal.

Article Declarations

Prospects for further research. Further research should focus on evaluating different therapeutic approaches to WD, particularly in randomized controlled trials.

Study limitations. The main limitations of this case report include the unavailability of PCR and IHC testing for *T. whipplei*, the absence of additional testing to exclude other causes of PAS-positive macrophages in the duodenum, and the lack of histological confirmation of panniculitis and its association with WD.

Primary data and materials. All patient data obtained from the source documents were anonymized.

Research ethics. The study was conducted in accordance with all protocols and procedures stipulated by the Biomedical Research Ethics Committee, the requirements of Ukrainian healthcare legislation, and the principles of the Declaration of Helsinki (2000). The patient provided informed consent for publication of the case and accompanying clinical data. The article was approved by the local ethics committee.

Declaration of AI use. Artificial intelligence tools were not used in the preparation of this article.

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АРТРАЛГІЯ, МОЖЛИВИЙ ПАНІКУЛІТ, ВТРАТА ВАГИ ТА АНЕМІЯ: ТРИВАЛИЙ ДІАГНОСТИЧНИЙ ШЛЯХ ДО ХВОРОБИ УІППЛА

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РЕЗЮМЕ

Вступ. Хвороба Уіппла (ХУ) – надзвичайно рідкісне системне інфекційне захворювання, спричинене *Tropheryma whipplei*, частота якого становить приблизно один випадок на мільйон населення. Через мультисистемне ураження та неспецифічність клінічних проявів захворювання може імітувати гастроентерологічні, ревматологічні, інфекційні та інші патологічні стани, що часто призводить до значної затримки встановлення діагнозу. Метою роботи було описати та проаналізувати клінічний випадок класичної ХУ з тривалим діагностичним шляхом та окремими нетиповими проявами, а також визначити чинники, які могли сприяти затримці діагностики.

Матеріали та методи. Представлено клінічний випадок 45-річної жінки з тривалою діареєю, артралгією, прогресуючою втратою маси тіла, анемією, системним запаленням, лімфаденопатією та клінічно підозрюваним панікулітом. Проаналізовано клінічний перебіг захворювання протягом приблизно 3,5 року, результати лабораторних досліджень, комп'ютерної томографії, езофагогастродуоденоскопії, колоноскопії та гістологічного дослідження біоптатів дванадцятипалої кишки. Для оцінки можливого позакишкового ураження проведено кардіологічне та неврологічне обстеження, включаючи ехокардіографію та магнітно-резонансну томографію головного мозку.

Результати. Першим проявом захворювання була тривала неводяниста діарея, яка згодом супроводжувалася гарячкою, артралгією, болючими підшкірними вузликами, значною втратою маси тіла та прогресуючою залізодефіцитною анемією. Комп'ютерна томографія виявила ретроперитонеальну та мезентеріальну лімфаденопатію. Початкове ендоскопічне дослідження верхніх відділів шлунково-кишкового тракту без

виконання біопсії не дозволило встановити діагноз. При повторній езофагогастродуоденоскопії виявлено набряк, зернистість і гіперемію слизової оболонки дванадцятипалої кишки, потовщення складок та виражені зміни ворсинок. Гістологічне дослідження дуоденальних біоптатів продемонструвало скупчення макрофагів із широкою еозинофільною цитоплазмою, що містила PAS-позитивні гранули. У поєднанні з типовою клінічною картиною ці результати стали підставою для встановлення діагнозу хвороби Уіппла. Ознак ураження центральної нервової та серцево-судинної систем не виявлено. Панікуліт був запідозрений клінічно, проте гістологічно або іншими специфічними методами не підтверджений. Лікування розпочато з внутрішньовенного введення цефтріаксону протягом 14 днів із подальшим переходом на доксициклін та гідроксихлорохін. Через чотири тижні терапії пацієнтка повідомила про суттєве симптоматичне покращення.

Обговорення. Діагностична затримка тривалістю приблизно 3,5 року була пов'язана з рідкістю захворювання, неспецифічністю та мультисистемністю його проявів, нетиповою для класичної ХУ жіночою статтю пацієнтки, попереднім застосуванням антибактеріальної терапії з частковим тимчасовим ефектом та відсутністю біопсії під час першого ендоскопічного дослідження. Випадок демонструє, що відсутність характерних макроскопічних змін дванадцятипалої кишки під час ендоскопії не виключає ХУ. Поєднання тривалої діареї, артралгії, втрати маси тіла, ознак мальабсорбції, анемії та лімфаденопатії має підвищувати клінічну настороженість щодо цього захворювання.

Висновки. Хворобу Уіппла слід включати до диференційної діагностики у пацієнтів із тривалими нез'ясованими гастроінтестинальними та системними проявами, особливо за наявності поєднання діареї, артралгії, втрати маси тіла, мальабсорбції, анемії та лімфаденопатії. Біопсія дванадцятипалої кишки є ключовим методом діагностики та має розглядатися навіть за відсутності виражених ендоскопічних змін слизової оболонки.

Ключові слова: Хвороба Уіппла, Tropheryma whipplei, діарея, артралгія, панікуліт, дуоденальна біопсія, діагностична затримка.

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