# THE CLINICAL COURSE OF WILSON'S DISEASE AND TREATMENT ADHERENCE

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Wilson's disease is a rare autosomal recessive disorder (prevalence 1 : 30,000) caused by pathogenic variants in the *ATP7B* gene (OMIM 277900). Early diagnosis and pathogenetic therapy enable stable remission. Poor treatment adherence is the key factor adversely affecting the prognosis. This family case study has demonstrated that inadequate attitude towards therapy contributes to the progression of organ damage and increases the risk of disability and mortality. Clinical observation confirms a direct link between compliance with the treatment regimen and clinical outcomes.

**Keywords:** Wilson's disease, *ATP7B* gene, copper, pathogenetic therapy, treatment adherence, liver cirrhosis

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# КЛИНИЧЕСКОЕ ТЕЧЕНИЕ БОЛЕЗНИ ВИЛЬСОНА–КОНОВАЛОВА И ПРИВЕРЖЕННОСТЬ ТЕРАПИИ

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Болезнь Вильсона–Коновалова (БВК) — редкое аутосомно-рецессивное заболевание (распространенность 1 : 30 000), обусловленное патогенными вариантами гена *ATP7B* (OMIM 277900). Ранняя диагностика и патогенетическая терапия позволяют достичь устойчивой ремиссии. Ключевой фактор неблагоприятного прогноза — низкая приверженность лечению. Исследование на примере семейного случая продемонстрировало, что недостаточная приверженность терапии способствует прогрессированию органных поражений и повышает риск инвалидизации и смертности. Клиническое наблюдение подтверждает прямую связь между соблюдением режима лечения и клиническими исходами.

**Ключевые слова:** болезнь Вильсона–Коновалова, ген *ATP7B*, медь, патогенетическая терапия, приверженность лечению, цирроз печени

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**Соблюдение этических стандартов:** все участники исследования либо их законные представители подписали добровольное информированное согласие на участие в научном исследовании.

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Wilson's disease (ICD-10 code E83.0) is an orphan autosomal recessive disorder caused by pathogenic variants of the *ATP7B* gene (OMIM 277900). The prevalence of this pathology is relatively low — about 1 in 30,000 — but studying it is clinically important [1].

There is an effective pathogenetic therapy for Wilson's disease that significantly improves patient's prognosis and quality of life if the diagnosis is made in a timely manner and treatment is appropriate. Modern therapeutic strategies focus on removing excess copper from the body and preventing its further accumulation, thereby controlling disease progression, preventing damage to the liver and central nervous system, and achieving stable remission provided the patient adheres to the treatment long term [2, 3].

Adherence to treatment is an extremely important factor determining the outcome of Wilson's disease [4]. According to a retrospective study, about 74.1% of patients with this disorder regularly receive medication without significant interruptions [5]. Almost 98% of cases in which pharmaceutical therapy was followed as prescribed showed improvement or stabilization of the patient's condition. These findings clearly demonstrate how important uninterrupted treatment is for a therapeutic effect [5].

Studies of the economic burden of Wilson's disease in various countries demonstrate its significant financial and

social consequences [6, 7]. However, most authors of such studies focus primarily on costs directly associated with the therapy, overlooking indirect expenses such as the treatment of comorbidities, long-term patient management, and economic losses resulting from health deterioration and disability in patients who do not adhere to therapy.

In most cases, the course of Wilson's disease worsens not because effective treatments are lacking, but because patients do not adequately adhere to prescribed therapy. This leads to the progression of organ damage and increases the risk of disability and mortality.

This study presents a family case of Wilson's disease; in this case, how the patients followed treatment recommendations directly affects with clinical outcomes, which emphasizes the critical role of treatment adherence.

This study aimed to examine the correlation between compliance with the treatment regimen and the clinical course of Wilson's disease in a family case.

## Clinical case description

This family case involves siblings (brothers) with Wilson's disease. Patient I (proband), born in 2001, had the first symptoms manifesting at the age of 14; they were increasing nausea and vomiting. Later, the list of symptoms extended to include general weakness, nosebleeds, peripheral edema, and diffuse abdominal pain particularly noticeable in the right hypochondrium. Based on this clinical picture, doctors at the patient's residence diagnosed him with Wilson's disease. Despite the absence of confirmatory laboratory and instrumental studies, copper-chelation therapy with D-penicillamine (500 mg/day) was initiated. Key indicators (24-hour urinary copper excretion and biochemical blood tests) were not monitored regularly.

In 2018, at the age of 17, the patient's condition worsened: there appeared pronounced peripheral edema, ascites, increasing nausea and dyspepsia. He was admitted to the Sechenov University Clinical Center (E. M. Tareev Clinic of Rheumatology, Nephrology and Occupational Pathology) for diagnosis verification and therapy correction. The examination (including laboratory and instrumental tests) revealed the following changes: a marked decrease in ceruloplasmin level, increased serum copper concentration, thrombocytopenia with mild splenomegaly, signs of liver failure, and manifestations of portal hypertension (Table). No central nervous system damage or Kayser–Fleischer ring was detected. A molecular genetic study confirmed the diagnosis: a pathogenic variant, c.3207C>A (p.His1069Gln), in the *ATP7B* gene was found in the homozygous state. As the proband's mother clarified, he repeatedly interrupted his prescribed drug therapy, skipped doses, and interrupted treatment without consulting a doctor. Resuming regular intake of D-PAM at an increased dose (up to 1500 mg/day) stabilized the clinical picture.

The family history is burdened: the patient's older sister died at the age of 28 from liver failure (liver cirrhosis of unspecified etiology). Genetic testing revealed the brother has the same homozygous ATP7B variant as the proband. The parents are clinically healthy (Figure).

At age 22 (in 2023), the patient was readmitted to our medical center with Wilson disease in severe decompensation. Patient interview revealed that the deterioration in his condition was also associated with non-adherence to the recommended diet and irregular intake of D-PAM, including prolonged interruptions in therapy without medical consultation.

Clinically, the disease progressed and brought new neurological symptoms, including gait and sleep disorders. Laboratory tests confirmed liver cirrhosis with hepatic failure: hypoproteinemia, severe hypoalbuminemia, a marked reduction in prothrombin index to 28%, and fibrinogen 1.65 g/L. There were also signs of intravascular hemolysis: hyperbilirubinemia with a predominance of the indirect fraction (total bilirubin 163.5 mmol/L; direct bilirubin 73 mmol/L), and a hemoglobin reduction to 108 g/L. Instrumental and clinical tests confirmed manifestations of portal hypertension (splenomegaly, esophageal varices, ascites) and thrombocytopenia (platelet count 89 × $10^9$/L). An ophthalmological examination revealed a Kayser–Fleischer ring.

After the resumption of D-PAM (at an increased dose to 1500 mg/day) and addition of diuretics and infusion therapy, the patient's condition stabilized. The recommended treatment was liver transplantation. Unfortunately, patient I died at the age of 22.

Patient II, born in 1993, attended the Sechenov University Clinical Center (E. M. Tareev Clinic of Rheumatology, Nephrology and Occupational Pathology) after his younger brother's death.

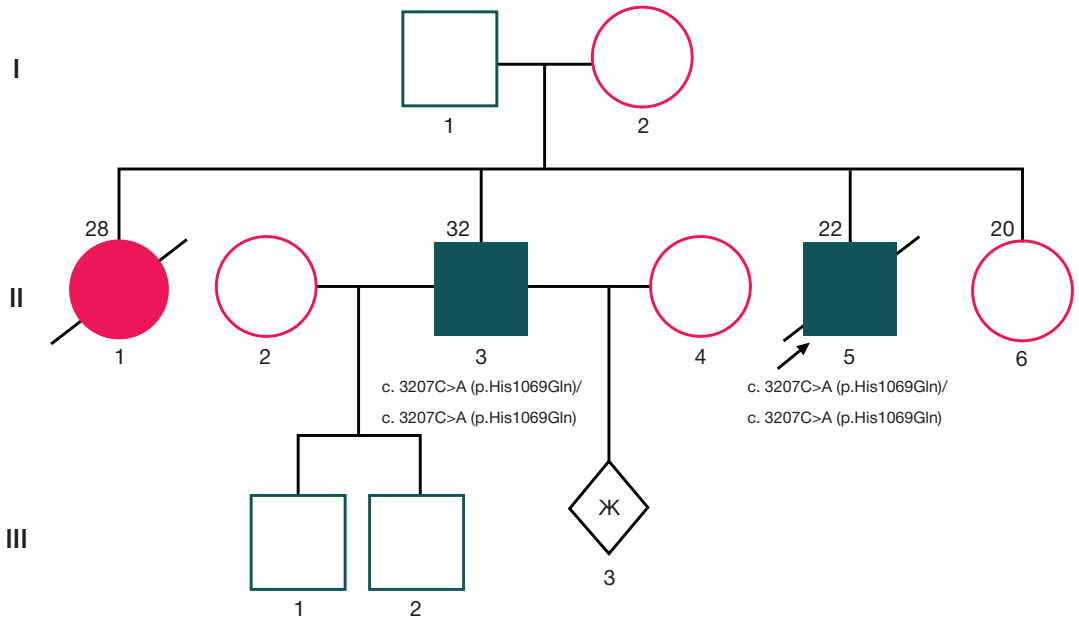


**Fig.** Family tree

**Table.** Clinical manifestations of Wilson's disease in sibs

| Clinical indicators | Sibs | | | |
|---|---|---|---|---|
| | Patient I | | | Patient II |
| Age of manifestation | 14 years | 17 years | 22 years | 29 years |
| Clinical manifestations | Generalized weakness, nausea, vomiting, epistaxis | Nausea, dyspepsia, severe peripheral edema, ascites | Generalized weakness, pronounced peripheral edema, ascites, heaviness in the right upper quadrant, and bilateral Achilles tendon pain | Generalized weakness, peripheral edema, epistaxis |
| Kayser–Fleischer ring | – | – | + | + |
| Neurological manifestations | No | No | Gait and sleep disorders | No |
| Liver function indicators | ALT — 49.9 units/L (0–40), AAT — 51.1 units/L (0–41), ALP — 75 units/L (0–120) | ALT — 62 units/L (0–40), AAT — 83 units/L (0–41), ALP — 75 units/L (0–120) | ALT — 46 units/L (0–40), AAT — 61 units/L (0–41), ALP — 75 units/L (0–120) | ALT — 16.7 units/L (0–40), AAT — 22 units/L (0–41) ALF — 148.8 units/L (0–120) |
| Ceruloplasmin | Not documented | 0.10 g/L (0.20–0.35 g/L) | 0.10 g/L (0.20–0.35 g/L) | 0.119 g/L (0.20–0.35 g/L) |
| 24-hour urinary copper excretion: baseline | Not documented | 13.93 mmol/day (0.6–1.0) | 16.83 mmol/day (0.6–1.0) | 2.22 mmol/day (0.6–1.0) |
| Diagnosis | Wilson's disease, abdominal form | Wilson's disease, abdominal form Liver cirrhosis, Child–Pugh class C | Wilson's disease, mixed form. Liver cirrhosis, Child–Pugh class C (12 points) Grade I hepatic encephalopathy | Wilson's disease, abdominal form, F3 liver fibrosis (METAVIR score) |

The 2018 examination (patient's age 25), which was part of the family molecular genetic screening, verified his diagnosis — Wilson's disease. Despite the established pathology, the patient decided to refrain from additional diagnostic procedures at that time. At admission (age 29), patient II complained of generalized weakness, peripheral edema, and epistaxis. Laboratory tests revealed reduced ceruloplasmin (0.119 g/L; normal range: 0.20–0.35 g/L) and elevated 24-hour urinary copper excretion (2.22 mmol/day; normal range: 0.6–1.0 mmol/day) (Table). Instrumental tests confirmed Kayser–Fleischer rings and F3 liver fibrosis (METAVIR score). Diagnosis: Wilson's disease, abdominal form. Initiated therapy: D-PAM for copper elimination. The patient is under regular follow-up, feels well, continues to work, and leads an active lifestyle.

## Clinical case discussion

In the described family case of Wilson's disease, it is associated with a common variant of the ATP7B gene. However, the clinical picture varies distinctly among family members. The key factor influencing the severity and progression of the disease is patients' adherence to prescribed therapy; interruptions or irregular intake of medications significantly worsens the prognosis.

The pathogenic ATP7B variant c.3207C>A (p.His1069Gln), identified in the family in the homozygous state, is one of the most common genetic variants associated with Wilson's disease. In Slavic populations, it may account for 30–60% of all identified pathogenic variants causing Wilson's disease. Therefore, it is an important marker for screening and molecular genetic diagnosis of the condition [1].

Homozygosity for p.His1069Gln is usually associated with later onset of neurological symptoms and a relatively favorable course of Wilson disease [8]. Nevertheless, the presented family case shows pronounced clinical polymorphism despite an identical genotype, as described in several studies [9]. In patient I, the onset of the disease at the age of 14 was characterized mainly by abdominal symptoms (nausea, vomiting, abdominal pain), with progression of liver damage to cirrhosis and liver failure. At the age of 22, against the background of decompensation, there are appeared neurological manifestations (gait and sleep disorders), which signals transition from the abdominal form to a mixed one [3].

At the time of clinical examination (age 29), patient II, who was also homozygous for the p.His1069Gln variant, presented with less pronounced symptoms including weakness, edema, nosebleeds, and signs of liver fibrosis.

Patients show significant variability in age of onset, severity of liver damage, and neurological symptoms. The differences can be explained by a combination of genetic and environmental factors, including dietary patterns and the modifying effect of other genes [10]. The key factor determining the unfavorable outcome in patient I was non-adherence to pathogenetic therapy. In his case, copper-chelating D-PAM at a dose of 500 mg/day was started early, but key parameters, including 24-hour urinary copper excretion and blood biochemistry, were not monitored, and therapy was repeatedly interrupted. As a result, the disease progressed to liver failure, cirrhosis, portal hypertension, intravascular hemolysis, and neurological disorders.

Literature data indicate that adherence to treatment in Wilson's disease directly affects both the duration and the quality of life of patients [5, 11]. Without treatment, the mean life expectancy after the onset of the first symptoms of Wilson's disease is 5–6 years [12].

Noncompliance with the recommended treatment regimen significantly worsens prognosis, and the risk of liver transplantation in such patients increases three- to fivefold compared with patients who strictly follow medical recommendations [13]. Prolonged interruption of copper-chelating drugs, such as D-PAM, is particularly dangerous. Cases of fulminant liver failure have been reported in patients who discontinued therapy for 9 months or more, even those with a previously stable condition [14]. Associated with Wilson's disease, this type of failure translates into an extremely unfavorable prognosis. Without transplantation, mortality in such cases reaches 60% to 95%, highlighting the importance of early diagnosis, continuous monitoring, and strict adherence to treatment to prevent life-threatening complications.

Timely diagnosis and regular pathogenetic therapy ensure a normal life expectancy in most patients with Wilson's disease: the clinical picture improves or stabilizes in 98% of those who regularly take medications [5]. This confirms the high effectiveness of existing treatments, provided they are

systematically adhered to. Interestingly, an analysis by several authors found no significant relationship between adherence to medical recommendations and several potentially important factors. In particular, neither demographic characteristics (gender), nor clinical parameters (phenotypic picture of the disease), nor therapeutic specifics (type of treatment and its duration), nor side effects had a statistically significant effect on the adherence of patients with Wilson's disease to the prescribed treatment [5].

Existing treatments for Wilson's disease have two major limitations: the risk of side effects from D-penicillamine (affecting 10–30% of patients) and the requirement for lifelong medication. Thus, there is a need for fundamentally new approaches, among which gene therapy appears the most promising. The method delivers a functional copy of the *ATP7B* gene using AAV vectors, which potentially eliminates the disease's root cause and reduces the need for long-term pharmacotherapy [16].

At the same time, the prognosis for Wilson's disease critically depends on treatment adherence; without therapy, the life expectancy after symptom onset is only a few years, whereas with strict adherence to treatment recommendations, patients can live a long, full-fledged life.

## CONCLUSION

Wilson's disease is one of the few hereditary disorders for which an effective pathogenetic therapy exists. Timely diagnosis and regular treatment, provided that therapy is adhered to, can transform the disorder from a life-threatening condition into a controlled chronic disease with a favorable long-term prognosis. To ensure treatment adherence, it is necessary to implement support programs that include patient and family education, regular monitoring of the condition, and multidisciplinary support from specialists of various profiles.

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