

MEDICAL SCIENCES

DIFFICULT PATIENT IN THE PRACTICE OF A DOCTOR GULLO SYNDROME

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ABSTRACT

The article presents a clinical case, in patients in biochemical tests an increase in serum pancreatic enzymes was observed in the absence of symptoms of pancreatic disease.

Keywords: Gullo syndrome, asymptomatic pancreatic hyperfermentemia, pancreas, amylase, chronic pancreatitis.

Gullo syndrome- an asymptomatic increase in the level of pancreatic enzymes often leads to an erroneous diagnosis of "acute or chronic pancreatitis" and in many similar cases, an increase in enzymes - hyperfermentemia is benign [2,3].

The complexity of the diagnosis of Gullo syndrome was that in the absence of structural pathology of the pancreas according to the results of instrumental studies in combination with pronounced fluctuations in the activity of pancreatic enzymes.

For the first time, a series of cases of persistent asymptomatic hyperfermentemia in practically healthy individuals was described by the Italian internist researcher Lucio Gullo in 1996. He also proposed the term "benign pancreatic hyperfermentemia" [1, 3].

Prof. Gullo continued to monitor most of these patients from 1987 to 2006 and stated that during this period persistent hyperfermentemia persisted in the absence of an obvious pancreatic disease or other known causes.

The author concluded that the increased activity of pancreatic enzymes in these patients is benign, and therefore he called the described anomaly chronic non-pathological pancreatic hyperfermentemia, or benign pancreatic hyperfermentemia, or Gullo syndrome [6].

In most cases, with this syndrome, the level of at least two pancreatic enzymes is increased, in other cases there is an isolated increase in the activity of amylase or lipase, more often a small one (1.5—4 times). So far, about 240 cases of Gullo syndrome have been recorded worldwide [5]. Further studies made it possible to identify familial pancreatic hyperfermentemia, which occurs in 4.0–19.5% of all cases of asymptomatic benign increase in the activity of pancreatic enzymes, that laboratory abnormalities in these patients are not associated with pancreatic pathology [4, 7].

Purpose of presenting clinical observation.

Demonstrate the features of clinical manifestations of Gullo Syndrome and diagnosis of the disease; compare with literature data.

Features of clinical observation. A 17-year-old patient and a 35-year-old patient were diagnosed with Gullo syndrome, which is characterized by a pathological increase in serum pancreatic enzymes in the absence of other signs of the pancreas. As a clinical example of Gullo Syndrome, we offer our own one-year follow-up at an outpatient appointment.

Features of clinical observation. A 17-year-old patient and a 35-year-old patient were diagnosed with Gullo syndrome, which is characterized by a pathological increase in serum levels of pancreatic enzymes in the absence of other signs of the pancreas. As a clinical example of Gullo syndrome, we offer our own one-year outpatient follow-up.

A 17-year-old teenager was sent by the military registration and enlistment office to consult a gastroenterologist due to weight deficiency. He makes no complaints. From the anamnesis: I have not previously been examined by a gastroenterologist. He has been smoking since he was 14 years old, smokes 1 pack a day, often on an empty stomach.

On examination - Height 182 cm, weight - 59 kg. Skin of ordinary coloring, acne on the skin of the forehead and chin. The tongue is slightly overlaid with a whitish coating. The abdomen is soft, not swollen, soft and painless on palpation. A chair every 2-3 days, independently decorated.

Upon examination- Height 182 cm, weight - 59 kg. Skin of normal color, acne on the skin of the forehead and chin. The tongue is slightly coated with a whitish coating. The abdomen is soft, not swollen, soft

and painless on palpation. Stool once every 2-3 days, independently, decorated.

Laboratory data: General analysis of blood, urine, coprogram - a variant of the norm. Biochemical blood tests total bilirubin -18 mmol/l, ALT-0.41 mmol/l, AST-0.38 mmol / l, glucose-4.8 mmol / L, total protein – 75g / l, amylase -259 IU/l. (with a laboratory norm of 220 IU/l.) In urine analysis - amylase 2000 at The norm is 900. Two days later, we retake 2008 With repeated analysis: Biochemical blood tests total bilirubin -17 mmol / l, ALT-0.39 mmol / L, AST-0.38 mmol / L, glucose-4.5 mmol / L, total protein – 78g / L, amylase - 460 IU / L. (at normal laboratory 220 IU/l.) In urine analysis - amylase 2010 at a rate of 900. Two days later, we retake 2018 With a second examination (after 6 months), the level of alpha-amylase in the blood is 360 U / l, in urine - 200 U / l, creatinine in the blood is 80 mmol / L.

Instrumental studies: FGDS –Conclusion: Hypertrophic gastroduodenitis, exacerbation. Ultrasound of the abdominal cavity is a complete description of the norm, the pancreas is of normal size, with clear contours, homogeneous. CT scan with contrast of abdominal organs revealed no pancreatic pathology.

Upon repeated examination (after 6 months), the level of alpha-amylase in the blood is 360 U / l, in urine — 200 U / l, creatinine in the blood is 80 mmol / L. MRSP of the abdominal organs of pancreatic pathology was not detected. IFA on AFP, CA 19.9- negative

During the year, the patient was observed on an outpatient basis, took enzymes (creon 50 thousand units, mezim forte, pangrol 25000), conducted eradication therapy

Upon repeated examination (after a year), the level of alpha-amylase in the blood is 260 U / l, in the urine — 180 U / l. FGDS –Conclusion: Chronic superficial gastritis. Ultrasound of the abdominal cavity - the pancreas is of normal size, with clear contours, homogeneous.

The second clinical case:

Patient: K, 35 years old, dentist, in December 2023, went to the polyclinic at her place of residence with complaints of heaviness in the stomach and bloating.

From the anamnesis of the disease: He considers himself ill since 2015, when for the first time he began to worry about local dull pain around the navel, connects errors in the diet after taking carbonated drinks.

Followed a diet, pain syndromes passed on their own after 2 days.

In 2020, pain syndromes were again bothered (pain of a shingling nature with irradiation to the right hypochondrium), the onset of the disease was associated with the intake of fatty foods, followed a diet and after which the pain syndrome was stopped. At the end of November 2021, after overeating fatty foods, the symptoms of intestinal dyspepsia (bloating) began to bother again and at the beginning of December 2021, pain syndrome joined, she took activated charcoal, es-pumizan, sorbent at home on her own, did not notice much effect, and the CP was taken to the BSMP, examined by doctors, when passing second-hand tests: amylase pancreatic 1043 units and was diagnosed for the first time: Acute pancreatitis, received inpatient treatment and was discharged with minor improvement (upon discharge, alpha amylase 880 units. Then she was observed by a private gastroenterologist, received hospital treatment at home. Every year, the patient received treatment with antifibrinolytics, antispasmodics, antisecretory drugs, and enzymes. In 2021, the patient developed anaphylactic shock upon administration of gordox 20ml, and therefore the antifibrinolytic drug was discontinued. It was observed by m/w, periodically passed second-hand tests in March 2022 alpha amylase -600ED., against the background of diet and creon intake, in June 2022 alpha amylase decreased to -400ED. This deterioration has been associated with errors in the diet since November 2023, symptoms of intestinal dyspepsia began to bother, then pain syndrome joined, when passing second-hand tests, alpha amylase 605 units, and therefore sent for consultation to a gastroenterologist

From the anamnesis of life: Family history- the mother suffers from a duodenal ulcer. Allergic history: Allergic reactions to gordox medications in 2021 in the form of anaphylactic shock.

Objective data: The general condition is satisfactory. Height 168cm, weight 70kg. Digestive organs: The tongue is moist, slightly overlaid with a white coating. The abdomen is symmetrical, participates in the act of breathing. It is mild and painless on palpation. The liver does not protrude from under the edge of the costal arch, the spleen is not palpable. According to the words, the stool is 1-2 times a day for a week, porridge-like, without impurities.

Laboratory instrumental studies:

Complete blood count – no pathology

Table No. 1

Biochemical tests

Indicators	09.11.21	05/12/22	06/01/22	10.08.22	11/16/22	12/29/22	21.11.23
Pancreatic amylase	500IU/l	446IU/l	442IU/l	462IU/l	439IU/l	537IU/l	430IU/l
Lipase	55IU/l			40IU/l	40IU/l	45IU/l	
ALT	20.1U/l	11.8U/l	10.8U/l	11.7U/l	5.8U/l	12U/l	11U/l
AST	18.8 U/l	15.2U/l	13.2U/l	14.4U/l	12.20U/l	14.3U/l	16U/l
Total bilirubin	7.5 mmol/l	8.8 mmol/l	1.8 mmol/l	5.0 mmol/l	4 mmol/l	3.9 mmol/l	11.4 mmol/l
Direct bilirubin	4.1 mmol/l						
Total protein	82.5g/l	70.6 g/l	71.6 g/l	71.3 g/l	72.3 g/l	78.5 g/l	77.2g/l
Creatinine		58.0 mmol/l	59.0 mmol/l	57.0 mmol/l	54.0 mmol/l	84.2 mmol/l	82.1 mmol/l
Sugar	5.0 mmol/l			4.8 mmol/l	4.6 mmol/l	4.79 mmol/l	4.1 mmol/l
Alpha amylase urine	350ED						252ED
Elastase 1 feces							250µg/g
Anti-tissue transglutaminase antibody IgA							4.7U/ml
Anti-tissue transglutaminase IgG antibody							5.0U/ml

ELISA for HP from 04/01/22 IgA-positive
ELISA for HP Ig G from 04/01/22 – positive

ELISA for Vit B12 from 05/12/22 424.1 (normal), folic acid - 6.24 (normal)

Video esophagogastroduodenoscopy from 11/14/2023 Conclusion: Chronic antral gastritis.

MRI of the abdominal cavity and retroperitoneal space+ with contrast from 12/14/2022

Conclusion: Deformation and congestive changes in the gallbladder. No evidence of invasive neoplasms in the abdominal cavity was identified.

Ultrasound (liver, gallbladder, pancreas, spleen, kidneys) ot 11/13/2023 Conclusion: Moderate congestion in the gallbladder. MKD.

11/20/2023 Feces on I/g There are no helminths in the feces, Absent

11/20/2023 Determination of tumor antigen (CA 19-9) in blood serum by ELISA method CA 19-9, 6.39 (normal)

11.21.2023 ELISA for determination of HBsAg of hepatitis B virus in serum and determination of total antibodies to hepatitis C virus- Negative

11/21/2023 ELISA for determination Ig G to Ascaris lumbricoides (ascariasis) in blood serum by ELISA method Ascariasis IgG, negative Ascariasis IgG (KP), 0.46, Giardiasis IgM, negative Giardiasis IgM (KP), 0.17

Determination of Ig G for Opisthorchis felineus (opisthorchis felineus) and Opisthorchis viverrini (opisthorchiasis) Opisthorchiasis IgG, negative Opisthorchiasis IgG (CP), 0.24, Toxocariasis IgG, negative Toxocariasis IgG (CP), 0.47

Coagulogram dated 11/23/2023 APTT 35.5 sec, INR 1.01, PT 11.40 sec, PTI 99.01%, fibrinogen 3.46 g/l

Thyroid hormones from 11/10/23- TSH 3.43, T4f 1.18 ng/dl, anti-TPO 18.30 IU/ml, insulin 5.31 µIU/ml - without pathology

11/27/2023 by PCR method qualitative RNA of the hepatitis C virus, Negative 11/29/2023 Feces for occult blood negative

Stool examination dated November 29, 2023 (coprogram) without pathology.

PCR stool for HP dated 02.11.23 - positive

MRI of the abdominal cavity and retroperitoneal space+ with contrast from 12/24/2023

Conclusion: MRI picture of diffuse changes in the parenchyma of the liver and pancreas. Deformation and congestive changes in the gallbladder.

No evidence of invasive neoplasms in the abdominal cavity was identified. Compared to MRI dated 12/14/22, no changes.

Taking into account the data, the patient was given a preliminary diagnosis: Chronic gastritis, HP associated, exacerbation stage. Gullet syndrome (benign pancreatic hyperfermentemia). Eradication therapy was prescribed on an outpatient basis for 2 weeks, she took enzymes (Creon 50 thousand units, Suvastin), the condition improved, the symptoms of gastric dyspepsia disappeared, when taking biochemical tests, alpha amylase decreased to 340 units/l

In 2 cases, good health, absence of concomitant diseases or habitual intoxications, abnormalities during physical examination, normal results of laboratory tests, except for amylase, as well as the absence of pathology according to ultrasound, CT and MRSP of the abdominal organs did not clarify the situation.

Conclusion: Thus, in the absence of structural pathology of the pancreas, according to the results of instrumental studies in combination with pronounced fluctuations in the activity of pancreatic enzymes, the diagnosis of benign pancreatic hyperfermentemia (Gullet syndrome) becomes the most likely in our patients. The accidentally detected hyperfermentemia was the reason for an in-depth examination, however, with a detailed history collection, a thorough physical and

laboratory instrumental examination, including ultrasound of the abdominal cavity and computed tomography, MRI of the abdominal cavity with contrast, explaining the increased activity of pancreatic enzymes, it was not possible to identify

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