

## **Comorbid Complicated Course of Vegetative Diabetic Polyneuropathy. Clinical Case. Multidisciplinary Approach in Treatment**

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### **Abstract**

Diabetic polyneuropathy is the most difficult to diagnose manifestation of diabetes mellitus, affects many organs and systems, exacerbates the course of complications of both an infectious and non-infectious nature. Currently, tuberculosis remains one of the socially significant pathologies in modern medicine. Extrapulmonary form (for example, liver tuberculosis) is one of the most difficult to diagnose forms of this pathology. Mortality in the presence of an asymptomatic course is up to 40% in the population of patients with liver tuberculosis. The article provides an example of diagnostic search, supervision and combination treatment with a multidisciplinary approach on the example of a specific clinical case from practice.

**Keywords:** *Autonomic Polyneuropathy; Diabetes Mellitus; Tuberculosis; Liver; Abdominal Form*

### **Introduction**

Diabetic neuropathy (DN) is symptom complex caused by degeneration of peripheral and autonomous nerves in patients with diabetes. It is based on two mechanisms - metabolic and vascular disorders. Previously, diabetic neuropathy was defined as clinical complication associated with diabetes symptoms or without symptoms, characterized by a lesion as peripheral and autonomic nervous systems, with the exclusion of others etiological reasons. In the joint consensus document (19<sup>th</sup> Annual Diabetic Neuropathy Study Group of the European Association for the Study of Diabetes (NEURODIAB) and the 8<sup>th</sup> International Symposium on Diabetic Neuropathy in Toronto, Canada, October 13 - 18, 2009) proposed split definitions of typical diabetic polyneuropathy and atypical diabetic polyneuropathy. Atypical diabetic polyneuropathy is significantly different from typical diabetic polyneuropathy, both by pathophysiological mechanisms of development, and with the flow and clinical manifestation. This option is characterized by the so-called, intercurrent flow, development and manifestation is possible in any Diabetes moment. Symptoms may occur acutely, subacute, or wear chronic nature, both with a monophasic version of the course, and with waved. The presence of autonomous dysfunction is also characteristic. Unambiguous diagnostic criteria and determine the severity of the course there are currently no atypical forms of Diabetic neuropathy. Atypical picture Diabetic neuropathy is neuropathy of thin (small-caliber) myelinated and non-myelinated fibers. As a result, approximately 50% of patients with

long-term diabetes mellitus organ function impaired gastrointestinal tract and hepatobiliary zone, which is accompanied by dysphagia, a tendency to erosion and strictures due to slow passage of drugs. Currently, less than a third of doctors recognize signs of diabetes-related polyneuropathy (IDF.org, IDF Diabetes Atlas - 9<sup>th</sup> Edition, 2019). Untimely diagnosis and treatment against the background of the atypical course of this pathology significantly contributes to high morbidity, disability, deterioration in the quality of life and mortality.

Tuberculosis has been and remains an urgent problem of medicine, as it is a universal disease that affects any organs and systems. In addition, tuberculosis is one of the medical and socially significant pathologies [1]. According to international statistics, in 2016, 6309134 new cases of tuberculosis and its relapses were detected in the world, of which 15% were extrapulmonary [2]. In the Russian Federation, tuberculosis of extrapulmonary localization in 2016 amounted to 3% [3]. Among extrapulmonary localizations of tuberculosis, the proportion of patients with abdominal tuberculosis is from 2 to 6% of the entire cohort of patients with this pathology. In abdominal tuberculosis, the liver and spleen are affected in every third case (32.3%) [1,3,4].

Abdominal tuberculosis is a specific lesion of the digestive system, peritoneum, lymph nodes of the mesentery of the small intestine and retroperitoneal space, which does not have pathognomonic symptoms [1], therefore, the majority of patients with tuberculosis of the abdominal organs are examined in the general treatment network under various diagnoses. The clinical picture of abdominal tuberculosis is polymorphic, there are no clear diagnostic criteria, therefore, as a rule, it proceeds under the guise of other diseases of the abdominal organs [1,4].

### Case Report and Discussion

Clinical case from own practice. Patient M., born in 1964, turned to a gastroenterologist for an appointment with complaints of periodic pain in the right hypochondrium of an aching nature, not associated with eating, sometimes intensifying at night; periodic increase in body temperature to 37.2 - 37.4°C, headaches. From the anamnesis: type 2 diabetes mellitus, subcompensated for 16 years (diet therapy, metformin 1000 mg 2 times/day, diabeton MB 60 mg/day), no occupational hazards, annually underwent medical examinations as part of the maternity group for the past 16 years, from previous diseases noted spondylarthrosis of the lumbar spine, acute pneumonia, sinusitis; there were no injuries. At the initial examination, pain in the right hypochondrium, liver + 2 cm from the edge of the costal arch to the right draws attention. Stool, urination in N. A.D. 140/80 mm Hg, Ps 90/min. The vesicular breathing, no wheezing, no restriction of respiratory movements. Heart sounds are clear, rhythmic. Recommended: general blood test, general urine test, biochemical blood test, ultrasound examination of the abdominal organs, fibroesophagogastroduodenoscopy.

Analysis data: Hgb - 126 g/l, Er -  $5 \times 10^{12}$ /ul, HCT - 38.7%, LY - 38.9%, MO - 8.6%, PLT -  $115 \times 10^3$ /ul, L -  $6,6 \times 10^9$ /ul, erythrocyte sedimentation rate - 20 mm/hour. Venous glucose - 7.5 mmol/liter, cholesterol - 4.9 mmol/liter, total bilirubin - 6.65  $\mu$ mol/liter, direct bilirubin - 2.03  $\mu$ mol/liter, indirect bilirubin - 4.62  $\mu$ mol/liter, ALT - 15.6 units/liter, AST - 20.7 units/liter, alkaline phosphatase - 103 units/liter, GGT - 175 units/liter, amylase - 40.1 units/liter, total protein - 69 g/liter, albumin - 43.75, serum iron - 13.03, creatinine - 99.9  $\mu$ mol/liter. APTT - 33 sec, prothrombin time - 14 sec, INR - 1.1, IPI - 93%, fibrinogen - 0.4, blood sugar 8.7 mmol/liter.

Ultrasound examination of the abdominal organs: liver 154 × 74 mm, contours are even, clear, heterogeneous structure, echogenicity is hyperechoic, hypoechoic formation 19 × 13 mm is visualized in the left lobe, gall bladder is 65 × 25 mm, walls are not thickened, content is homogeneous, sediment, no stones, v.porta 12 mm, common bile duct 4.6 mm, pancreas sizes within normal limits, head 26 mm, spleen 98 × 55 mm, contours clear, even, structure homogeneous, echogenicity unchanged.

Fibroesophagogastroduodenoscopy: esophagus - freely pass along the entire length, the mucosa without an inflammatory reaction, cardiac pulp is hypotonic; stomach - peristalsis is preserved, a moderate amount of mucus is in the lumen, the mucous membrane is hyperemic, the pylorus is freely passable, the bulb is 12 p. intestine without features. Urease test for *H. pylori* “+++”.

Dz: Biliary dyskinesia? Liver cyst?

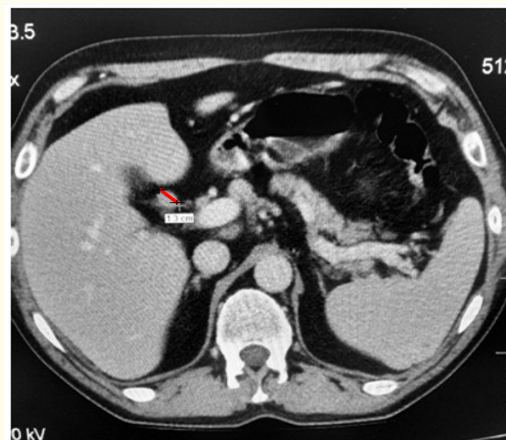
Given the presence of a volumetric formation in the left lobe of the liver, the patient was sent for a consultation with a surgeon.

After a joint examination, the patient is directed to a multi-spiral computed tomography of the abdominal organs and additional examinations.

Multi-spiral computed tomography of the abdominal organs. The study was performed in a spiral mode with a slice thickness of 1.0 mm, without and with iv contrast (100.0 ultravist-300, bolus excretion). Conclusion: multi-spiral computed tomography signs of the pathological zone of the left lobe of the liver (Figure 1). Hepatolienal syndrome, portal hypertension, cirrhosis of the liver? Lymphadenopathy (Figure 2). Local change in the left lobe of the liver of a dystrophic nature (local hepatosis). Multiple focal changes in the spleen of cystic nature (Figure 3).



**Figure 1:** The formation of the left lobe of the liver. Marked by cursor.

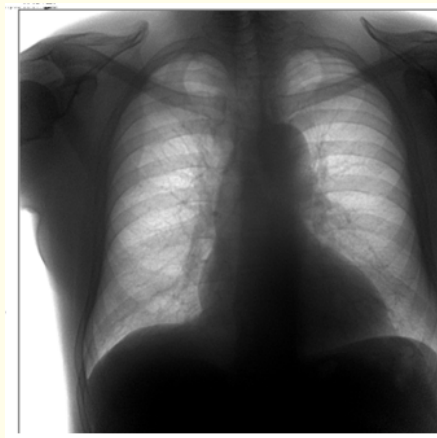


**Figure 2:** Hepatic lymphadenopathy. One of the lymph nodes is indicated by the cursor.



**Figure 3:** Cystic hypoechoic formation of the spleen.

Additional examination: 1. Alpha-fetoprotein (AFP) - 1.2 IU/ml, 2. HBsAg "-", HCV "-", 3. Ultrasound examination of blood vessels of the hepatolienal zone: echological signs of focal changes in the liver and spleen without obvious signs of portal hypertension, 4. X-ray of the chest organs (Figure 4): pulmonary pattern without focal and infiltrative changes, structural roots, sinuses free, mediastinum not expanded, heart, aorta within normal limits, no bone pathology detected, 5. Total video colonoscopy: apparatus performed in the dome of the cecum, throughout the mucous pink, is not changed, haustration usual, the tone in N.



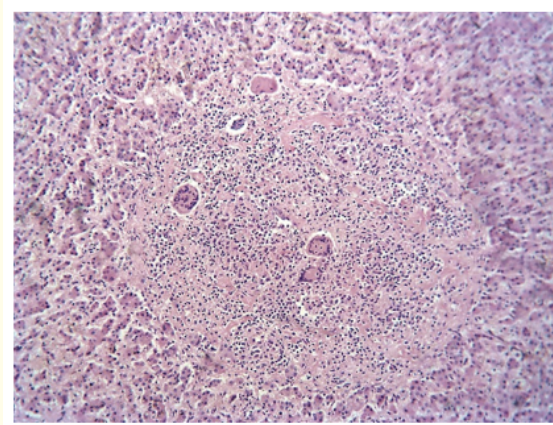
**Figure 4:** Chest x-ray.

Given the conflicting and partially mutually exclusive data of physical examinations and certain complaints, the patient, after a second joint examination with the surgeon, was referred for consultation to the Regional Clinical Oncology Center. Where computed tomography of the abdominal cavity and retroperitoneal space was performed with examination of the basal parts of the lungs and pleural sinuses.

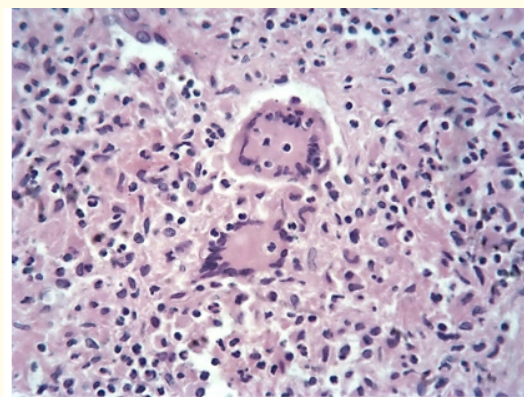
With dz: Susp. MTS hepar. Significant total hepatic lymphadenopathy, epigastric and retroperitoneal lymphadenopathy, the patient was recommended and performed trepan biopsy of the liver and spleen.

Macrodrug: In the column of liver tissue there is no tumor growth, discompletion of the beam structure, moderate fibrosis of the portal tracts with lymphohistiocytic infiltration.

Ying the diagnosis of D13.4 according to ICD-10 (benign liver tumors), the following operations were recommended and performed: atypical liver resection. The postoperative period was uneventful, without complications, the sutures were removed on day 12, wound healing by primary intention. Macro drugs were sent for histological and cytological examination. Macro drug: a fragment of the left lobe of the liver 12 × 7 × 4.5 cm, on the diaphragmatic surface under the capsule a node in diameter of 1.5 cm; fatty tissue 12 × 6 × 1 cm soft; a fragment of the right lobe of the liver in a diameter of 1.5 cm with nodulation in the center up to 6 mm. Microscopic examination: in the liver, multiple epithelioid cell granulomas with foci of caseous necrosis in the center; on the periphery, single multinuclear giant cells of the Pirogov-Langhans type, lymphocytic infiltration (Figure 5 and 6). No cancerous growth.



**Figure 5:** Tuberculous granuloma in the liver. Hematoxylin and eosin stain. x 200.



**Figure 6:** Detail of figure 5: Giant multi-core cells of the Pirogov-Langhans type. Hematoxylin and eosin stain. x 400.

In the postoperative period, a consultation with a TB specialist is recommended to exclude mycobacteriosis.

Consultation of a TB doctor at the Regional Clinical Tuberculosis Dispensary. Tuberculin tests were performed: diaskin test (DST) “-”, Mantoux test (RM 2 TE) “7 mm papule”.

In connection with the controversy of the diagnosis, he was sent for examination to clarify the diagnosis at the Federal State Budgetary Institution “St. Petersburg Research Institute of Phthisiopulmonology” with a direct diagnosis: Abdominal tuberculosis. Liver tuberculosis?

Also sent cartridges with macro and micropreparations.

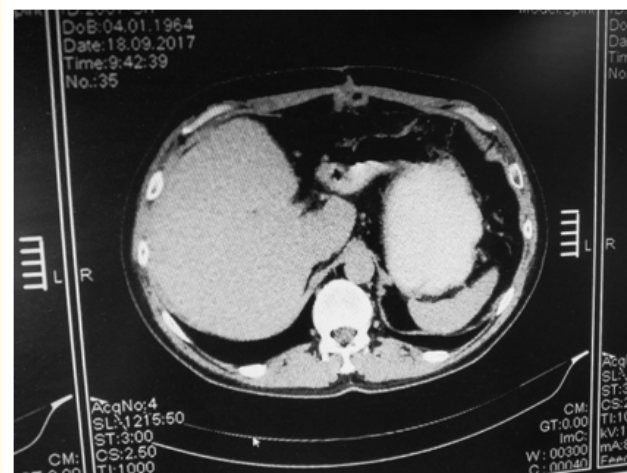
In St. Petersburg NIIF, the doctor issued and confirmed: A18.3 Tuberculosis of the intra-abdominal lymph nodes, tuberculosis of the liver.

Received course therapy for 5 months: H 0.6, R 0.3, Z 1.5, E1.2.

### Re-puncture biopsy

Then 15 days: H 0.6, R 0.45, vit B6 at 3 t/day. PTCT (H 0.6, R 0.45 - FP) for 6 months.

Control multi-spiral computed tomography of the abdominal organs 5 months after surgical treatment with intravenous bolus contrast, multiplanar and three-dimensional reconstruction: a few foci up to 4 mm in the spleen, no data for lymphadenopathy, condition after atypical liver resection (Figure 7).



**Figure 7:** After the operation. The left lobe of the liver is resected, the lymph nodes are removed.

Within 24 months, it is observed on an outpatient basis by a gastroenterologist, surgeon, endocrinologist. 1 year after the pathogenetic treatment, an x-ray of the chest organs was made, an ultrasound examination of the abdominal organs was examined by a phthisiator. Dz: Clinical cure for tuberculosis of the intra-abdominal lymph nodes, liver tuberculosis. Condition after atypical liver resection. Currently continues to be observed by a gastroenterologist, surgeon and endocrinologist. No complaints.

## Conclusion

Thus, as this clinical case illustrates, tuberculosis of the liver and intra-abdominal lymph nodes, especially against autonomic polyneuropathy does not always proceed according to the criteria described in the literature [1,5,6]: 1. Combined with tuberculosis of the spleen, 2. It manifests itself in generalized specific processes in combination with tuberculosis of the lungs, 3. Morphologically manifests itself as a miliary form, 4. It is always accompanied tuberculosis intoxication and clinical and laboratory signs of hepatitis of moderate laboratory activity. Diagnosis of this pathology in clinical practice causes certain difficulties, both among specialists in this area and among doctors of related specialties. Therefore, an individual approach and a clear caution in relation to patients against the background of multidisciplinary examination and treatment are necessary.

## Disclosure

The authors report that there are no financial interests or conflicts of interest in this article.

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