CLINICAL OBSERVATION OF MACROAMYLASEMIA ON THE BACKGROUND OF SPLENOSIS DUE TO THE POST-TRAUMATIC SPLENECTOMY (LITERATURE REVIEW AND CLINICAL OBSERVATION)

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Macroamylase is represented by large molecules with molecular weight ranging from 150000 to 2000000 (molecular weight of normal human amylase is equal to 50000–55000) [34].

Macroamylasemia arises due to the enzymatically active macro-molecular amylase complexes in blood flow. Large size of these complexes precludes their excretion by the kidneys and provokes their retention in blood. It's proved by the increased amylase activity in blood and decreased activity of this enzyme in urine [9].

Several reviews on macroamylasemia have been published with the aim of specifying pathogenesis, diagnostics of this state and defining its types [30, 54].

Types (stages) of macroamylasemia [9]:

type 1 is a classic form that was described first. It's characterized by persistent hyperamylasemia, reduced amylase activity in urine, and a relatively high concentration of macroamylase complex (i.e. amylase complex with a protein) in serum;

type 2 is also associated with hyperamylasemia, but amylase concentration in urine may be diminished insignificantly, and the ratio of macroamylase to normal amylase in serum is much less than in type 1;

type 3 is characterized by normal serum amylase activity, normal indices of urinary amylase, and usually a low ratio of macroamylase to normal amylase in serum.

Macroamylase molecule could theoretically represent itself as:

1) a polymer of normal amylase;
2) an abnormal amylase;
3) a complex of normal serum amylase with a serum protein;
4) a complex of normal serum amylase with a non-protein substance, such as a carbohydrate.

Results of conducted researches confirm a possibility of existence of the complex of normal serum amylase with serum proteins or carbohydrates upon macroamylasemia, while the evidence of existence of the polymers of normal and abnormal variants of amylase isn't adduced [9].

Upon macroamylasemia amylase in blood is more frequently bound with such substances as high molecular weight proteins — particularly, immunoglobulin A and, rarely, immunoglobulin G [9].

The fact that macroamylase usually represents amylase-immunoglobulin complex formed by the reaction of 'antigen-antibody' is proved by detection of this complex upon various autoimmune diseases and diseases accompanied with malabsorption: celiac disease [15, 35], systemic lupus erythematosus [47], rheumatoid arthritis [13], as well as AIDS [17], and other infections [13]. There are cases of macroamylasemia in pregnant women [27], healthy children and children with frequent episodes of acute respiratory diseases [39, 45]. Macroamylasemia can occur upon various diseases which are based on the inflammatory process, e.g. acute appendicitis [36].

Macroamylase complexes can be formed in the presence of abnormal proteins in blood connected with amylase, e.g. upon multiple myeloma, myeloid leukemia [29, 37]. Macroamylasemia can be combined with the presence of other macroenzymes in blood [8, 24, 43].

While normal amylase in blood is represented by two types of isoamylase (pancreatic — P-type, salivary — S-type), macroamylase includes mainly S-type isoamylase. Upon pancreatic pathology, activity of P-type amylase increases in blood, while there's an increasing activity of S-type amylase upon pathology of salivary glands.

Macroamylase can include not only immunoglobulins, but also other proteins, e.g. α-antitrypsin [38]. Macroamylase molecules can also be formed as a result of binding amylase with polysaccharides or glycoproteins [11, 52].

Macroamylasemia is diagnosed according to the three-stage algorithm by J. E. Berk (1995) [9]. In the first stage blood lipase activity and the ratio of amylase (C_{am}) and creatinine clearances (C_{cr}) are determined, in the second — precipitation with polyethyleneglycol is performed, and in the third — chromatography is done.

Algorithm of differential diagnosis

(by J. E. Berk (1995) [9])

1. Assay serum lipase activity and determine the C_{am}/C_{cr} ratio.
   a) Increased serum lipase activity and normal (1% to 4%) or elevated (>4%) C_{am}/C_{cr} ratio would favor pancreatic disease and make macroamylasemia unlikely.
   b) Normal lipase activity [31] or an abnormally low C_{am}/C_{cr} ratio in the absence of impaired renal function would support macroamylasemia and warrant advancement to step 2.
2. Perform a PEG test.
   a) Precipitation of more than 73% of the amylase activity would fairly securely confirm the presence in the serum of a macroamylase. However, if the clinical features and pancreatic imaging strongly suggest pancreatic disease, or if isoamylase fractionation of the supernatant discloses an abnormally high level of P-type activity, advancement to step 3 should be considered.
   b) If less than 73% of amylase activity is precipitated but the clinical evidence for pancreatitis is not altogether convincing, perform agarose electrophoresis of a serum sample and look for a smeared pattern characteristic of macroamylasemia or, better, advance to step 3.
3. The tests in this final step, unfortunately, are complex and are probably obtainable at this time only in research laboratories interested in such procedures or perhaps in some reference laboratories.
   a) One method of established reliability is chromatography, performed by the standard procedure or by means of high-performance liquid chromatography.

Moreover, this method allows to reveals raised P-type isoamylase activity, thus confirming pancreatic disease (pancreatitis).

b) Another method, still to be assessed, is a combined immunochemical procedure [41, 42].

Rate of macroamylasemia isn't studied thoroughly, as special identifying researches were not conducted. Published fragmentary information is presented in table 1. These data confirm that macroamylasemia isn't an exotic state; they reflect significant spread of the disorder that deserves serious clinical studies [22]. Furthermore, the diagnostics of macroamylasemia is crucial for the exclusion of organic diseases of the pancreas.

As macroamylasemia doesn't have any clinical manifestations, it seems that it doesn't require any special treatment. Treatment should be directed to the disease which may have triggered the development of macroamylasemia (see above).

At present there is no method to separate macroamylase complexes effectively *in vivo*. Even if it becomes possible in the future, it's not clear what it may give from a clinical point of view. After all, nowadays there's no evidence that separation of macroamylase complexes relieves abdominal pain.

It's absolutely obvious that macroamylasemia could be temporary and transient. What does the disappearance of macroamylasemia mean? This important from a clinical point of view question cannot be answered nowadays due to the lack of information. It's proved that patients with macroamylasemia type 3 have temporal relationship between the disappearance of macroamylase from blood and recovery from disease provoking macroamylasemia [33].

Similar relationship was observed in some patients with macroamylasemia type 1 and 2 [28, 40], particularly in patients with malabsorption upon celiac disease after prescription of gluten-free diet [15].

What happens to patients with persistent macroamylasemia? Data on these cases are too limited and contradictory, what doesn't allow well-reasoned responding. There's one described clinical case of macroamylasemia in combination with extraordinarily high elevation of carbohydrate antigen CA 19-9 [21]. However, this does not give a right to draw any serious conclusion.

As for pancreatitis, macroamylasemia can be combined with it [43]. It's very likely that in these cases macroamylasemia shouldn't be considered as a manifestation or complication of pancreatitis. Such situations rather complicate the differential diagnostics, especially when there is no exacerbation of CP.

We didn't find any description of the clinical observations of macroamylasemia combined with splenosis in the literature. In this regard, the following clinical case, in our opinion, seems to be rather interesting.

Descriptions of the ectopic spleen and splenosis are extremely rare ones in the modern literature. Role of these states isn't sufficiently studied from the clinical and morphofunctional points of view. In studying this problem, it becomes clear that there is no uniform classification of ectopic splenic tissue, and that the existing discrepancy of such definitions as "ectopic spleen", "additional spleen", "accessory spleen" and "splenosis" leads to some confusion.

According to the Big Medical Encyclopedia, accessory spleen («lien accessories») is a proliferation of splenic tissue outside the spleen, which may be congenital anomaly or occur as a result of post-traumatic implantation of splenic parenchyma cells into other organs and tissues. If accessory spleen is present from birth, such a variant becomes normal one in particular individual. The incidence of congenital accessory spleen makes up 14–30%, with its most common localization in the gates and ligaments of the spleen and in the greater omentum [2, 14, 20]. Typically it's small-sized, with an ultrasonic structure identical to normal spleen [3, 7].

Splenosis is a post-traumatic focal ("nodular") implantation of splenic tissue [10], occurring more frequently in the abdominal cavity — in the parietal and visceral peritoneum, as a fragment of splenic tissue in the bed of a remote organ, as well as in the retroperitoneal space and in the extra-abdominal one — in the pleural cavity, pericardium, and postoperative scar and even in the meninges [2, 16, 18, 23, 25, 26, 44, 49, 50, 51]. The rate of post-traumatic splenosis makes up to 67% after open splenectomy [48] and to 80% after laparoscopic one [19, 32]. Purposeful autotransplantation of splenic tissue is one of the splenosis' variants used to prevent

postsplenectomy syndrome, the effectiveness of which is so far under discussion [6, 46].

Unlike accessory spleen, splenosis is being developed due to destruction of the splenic pulp with its dissemination. In some cases this can lead to septic complications such as necrosis, abscess formation, and bleeding upon traumatic injury. Both upon the added spleen, and upon splenosis there's a need to differentiate them from tumors of the abdominal cavity with high-tech methods of research, but publications of clinical observations of these states in the literature are single. Ultrasonography doesn't have quite high sensitivity — up to 68% [53]. Scintigraphy labeled red blood cells has better sensitivity with (Fig. 1). This method also gives a possibility to estimate the functional activity of residual foci of splenic tissue after splenectomy [4].

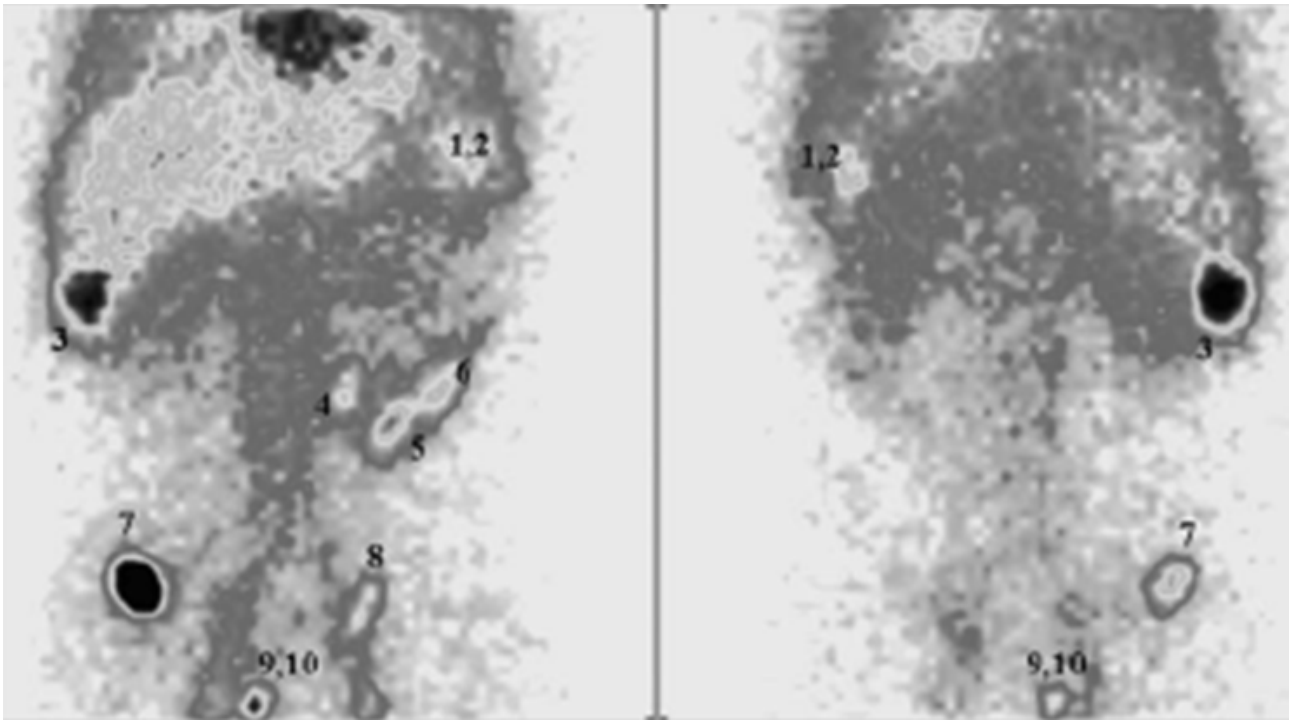

Fig. 1. Scintigraphy with red cells ^{99m}TC-labeled and damaged by heating (by K. A. Apartsyn et al., 2009 [1]). Foci of intraperitoneal splenosis in front (A) and posterior (B) projections

Considering the inaccessibility of radionuclide diagnostic method, ultrasound and laparoscopy remain the main methods of detection of splenosis, which can be supplemented by computed (CT) and magnetic resonance tomography for the differential diagnostics of tumors of the abdominal cavity. Studying the

morphofunctional state of accessory spleen confirms that it has all the basic structural elements of the main spleen and produces all its functions.

V. M. Timerbulatov et al. (2007) believe that splenosis is a normal and necessary state of organism, and define it as a proliferation of the splenic tissue outside the area of its natural localization, which may be congenital (as an accessory spleen and ectopia in the tissues of other organs) or acquired (as a result of splenic injury and implantation of its cells in adjacent organs) and designed to compensate for the lost splenic function (Fig. 2). Upon incidental revealing of congenital splenosis in clinical practice and in the absence of pathological changes, it's necessary to confine a statement of fact and inform the patient. Acquired splenosis demands for the organ-preserving tactics with the aim of prevention and correction of hyposplenism [5].

Fig. 2. Classification of splenosis (by V. M. Timerbulatov et al, 2007 [5])

Clinical observation

Patient U., 21 years old, student of Donetsk National Medical University, directed to the clinic of internal medicine n. a. A. Y. Gubergrits of DonNMU for the further diagnosis and correction of treatment. At the moment of visit she had no *complaints*. Preserved appetite. Normal body temperature. Normal stool and diuresis. Stable weight.

Anamnesis of disease. Considers herself a patient for 2–3 years, when the aching pain in the right hypochondrium emerged after the intake of fatty, fried foods. Didn't visit a doctor, didn't receive a treatment. Noticed growing pain syndrome in 2011. Examination revealed a slight decrease in hemoglobin level to 11.7 g/dL (norm 12.0–16.0 g/dL), increase in platelet level to $366\times10^3/mm^3$ (норма 180–$320\times10^3/mm^3$), increased activity of blood amylase to 147.1 U/L (норма 3.6–100.0 U/L). Other biochemical parameters (total protein, total and conjugated bilirubin, AST, ALT, alkaline phosphatase, γ-glutamyl transpeptidase, lipase, pancreatic isoamylase, cholesterol, triglycerides, glucose, glycated hemoglobin, urea, creatinine, uric acid and blood iron) are normal. Tumor markers (α-fetoprotein, CEA, CA 19-9) are negative. In May 2011, ultrasound of the abdomen revealed echo-signs of enlarged lymph nodes of the abdominal cavity, moderate diffuse changes in the liver and pancreas, echo-signs of cholecystitis, salt diathesis. Fibrogastroduodenoscopy determined normal endoscopic picture of the esophagus, erythematous gastro- and duodenopathy. Treatment by a gastroenterologist (Primer, Quamatel, Duspatalin, Cynarix, Ermital 25000, then Ermital 10000, Bifi-form Complex, Momordica compositum, Mucosa compositum) with slight effect. Later, normalization of hemoglobin level was showed in the dynamics, while platelets and blood amylase remained elevated.

Re-examination in 3 months revealed IgG antibodies to CMV and EBV-NA. In July 2011, ultrasound detected isoechogenic formation 1.96×1.72 cm in size, with smooth, clear circuits, close to the splenic parenchyma by its structure, in the projection of bed of the rejected spleen. Changes of other organs are still the same.

In July 2011, spiral CT of abdomen and retroperitoneum with preliminary per os contrast (Fig. 3) showed an increase in the head of the pancreas to 3.5×3.2 cm,

with a presence of small plots of low density in it; soft-tissue formation, with clear, irregular circuits 3.6×2.0 cm in size is detected in the projection of bed of the rejected spleen; rounded soft-tissue formations, with smooth, clear circuits, up to 0.9–1.3 cm in size are detected parasagittally in abdomen, under the anterior abdominal wall at the level of L2–L4. Conclusion: changes in the head of the pancreas may be caused by inflammation. Formations in abdomen of described above localization may appear due to the enlarged lymph nodes? remaining slices of the rejected spleen? CT with intravenous bolus enhancement is recommended in order to exclude neoplastic genesis.

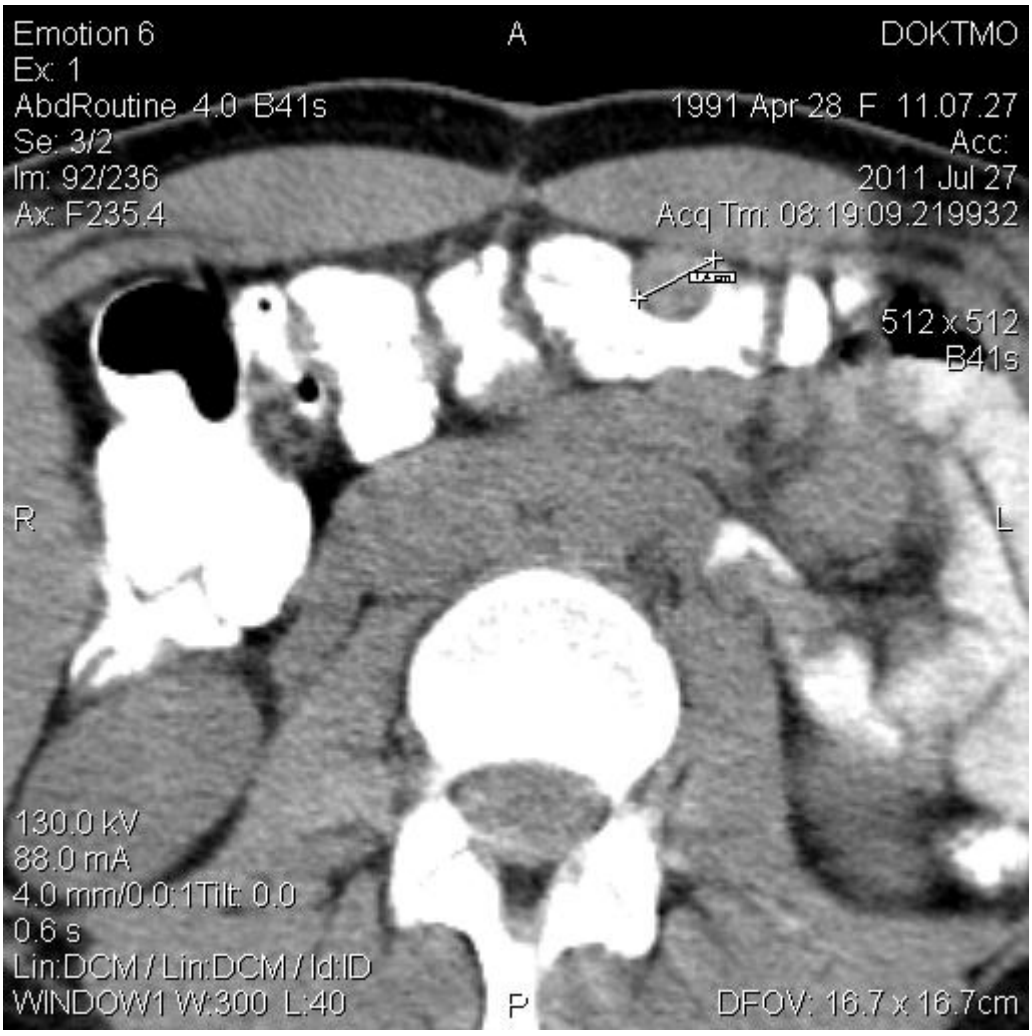

Fig. 3. Spiral CT of abdomen and retroperitoneum with preliminary per os contrast. Rounded soft-tissue formations are detected parasagittally in abdomen, under the anterior abdominal wall at the level of L2–L4 (splenosis?)

Examined by a surgeon. Diagnosis: additional slice of the spleen. No need of a treatment.

Upon repeated dynamic examination by gastroenterologist attention was attracted by the pain

syndrome and persistent increase of blood amylase to 1.5–3.0 norms with normal indices of urine amylase. Prescribed treatment (Quamatel, Ursofalk, Creon 10000, Laktovit-forte, broths of horsetail, mountain knotgrass, common knotgrass) turned out to be non-effective. In this regard, she was directed to the consultation at the clinic of internal medicine.

Anamnesis of life. Lymphadenopathy has been marked since the age of 4. At the age of 7 a laparotomy, splenectomy concerning blunt abdominal injury (fall from a height), traumatic splenic rupture, intraperitoneal bleeding was performed.

Patient denies tuberculosis, typhus, malaria, sexually transmitted diseases, dysentery, hepatitis, HIV.

Allergic anamnesis is not burdened.

Family anamnesis: mother twice underwent cyst drainage of the upper pole of the left kidney (?) in April and May 1995, had a disability group III for chronic continuously relapsing pancreatitis on the background of the operated pancreas in May 1995 (resection of the tail of the pancreas, splenectomy). Histological conclusion: cyst of the tail of the pancreas intimately welded with splenic parenchyma, the inner surface is focally lined with high prismatic epithelium, covered with granulations, in other areas — with necrotic and necrotic suppurative detritus, with focal giant-cell reaction of the "foreign bodies". Wall — coarse-fibered connective tissue. Mother was also noted to have double lymphadenopathy: axillary since 2001, supraclavicular and submandibular since 2011.

Objective data. General state is satisfactory. Body-build coincides with female type, nutrition is adequate. Skin and visible mucous are of normal color. Tongue is moist and clean. Thyroid gland is not enlarged, painless upon palpation. Peripheral lymph nodes — submandibular ones are enlarged on the both sides to 2×1 cm, in the other groups — small. Clear vesicular resonance. Vesicular breathing upon auscultation, without wheezing. Borders of the relative dullness of heart aren't expanded. Heart activity is rhythmic, with the tones of sufficient volume. Blood pressure — 120/70 mm Hg. Art. Pulse — 74 in 1 minute. Abdomen's volume isn't

increased. On examination, postoperative scar is marked in the midline; there're few dark spots in the area of the left hypochondrium. Abdomen is soft, painless upon the superficial palpation. Upon deep palpation, sensitivity appears in projection of the head and body of the pancreas. Bowel segments are of normal palpable characteristics, painless. Liver 0.5 cm comes out of hypochondrium, has elastic consistency, painless. Spleen isn't palpable (rejected). Kidneys aren't palpable. No peripheral edema.

Data of laboratory and instrumental examination.

Total blood analysis: leukocytes — $5.3\times10^3/mm^3$ (norm $4.0–9.0\times10^3/mm^3$), erythrocytes— $3.88\times10^6/mm^3$ (norm $3.70–5.00\times10^6/mm^3$), hemoglobin — 12.8 g/dL, hematocrit — 36.6% (norm 36.0–48.0%), platelets — $348\times10^3/mm^3$, stabs — 2% (norm 1–6%), segmanted — 52% (norm 47–72%), eosinophils — 3% (norm 0.5–5.0%), basophils — 0 (norm 0–1%), lymphocytes — 38% (norm 19–37%), monocytes — 5% (norm 3–11%), ESR — 4 mm/h (norm 2–15 mm/h).

Biochemical blood analysis: amylase — 144.6 U/L, γ-glutamyl transpeptidase — 13.8 U/L (norm 5.0–55.0 U/L), alkaline phosphatase — 53.1 U/L (norm 34.8–129.0 U/L), total bilirubin — 11.3 μmol/L (norm 5.0–21.0 μmol/L), direct bilirubin — 2.11 μmol/L (norm 0.0–3.4 μmol/L), creatinine — 63.7 μmol/L (norm 58.0–127.0 μmol/L), urea — 3.01 mmol/L (norm 0.10–8.30 mmol/L), lipase — 28.60 U/L (norm 0.0–60.0 U/L), pancreatic amylase — 21.30 U/L (norm 13.0–53.0 U/L), total cholesterol — 4.25 mmol/L (norm <5.16 mmol/L), triglycerides — 0.69 mmol/L (norm 0.40–1.53 mmol/L), glucose — 5.24 mmol/L (norm 4.10–5.90 mmol/L), glycated hemoglobin — 5.1% (norm <6.0%), calcium — 2.54 mmol/L (norm 2.20–2.65 mmol/L), iron — 14.7 μmol/L (norm 10.7–32.2 μmol/L), ferritin — 26.6 ng/ml (norm 10.0–250.0 ng/ml).

Total urine analysis: straw-yellow color, transparent, relative density — 1021 (norm 1018–1026), pH — 6.0 (norm 5.0–7.0), protein, glucose, ketones, bilirubin— not detected, urobilinogen — marks, pavement epithelium — covers all the fields of view, transitional epithelium — singular, leukocytes — 2–3 in the field of view, intact erythrocytes — singular, bacteria aren't detected.

Urine α-amylase — 60.10 U/L (norm 0.0–460.0 U/L).

Viral markers: HBsAg, anti-HBcore IgG+M, anti-HCV IgG+M, DNA-HSV-1/2, anti-CMV of class IgM, DNA CMV in blood, antibodies of class IgM to nuclear AG EBV-NA — not found; anti-CMV of class IgG — 24.2 U/ml (norm <3.0 U/ml), antibodies of class IgG to nuclear AG EBV-NA — 57.8 U/ml (norm <3.0 U/ml), DNA EBV in blood — found (++).

Antibodies to toxoplasma of class IgM — not found, class IgG — 3.2 IU/ml (norm <10 IU/ml), DNA of toxoplasma — not found. Antibodies of class IgG to toxocara, to antigens of Trichinella, to antigens of Opisthorchis, to antigens of Echinococcus — not found.

HIV-test — negative.

Tumor markers: CA 19-9, CA 125 — within the normal range.

Ultrasound diagnostics (Prof. A. D. Zubov, June 25, 2012) (Fig. 4a, b): no free fluid in the abdominal cavity and pleural sinuses. Liver: total size (left and right lobe) — not increased, smooth circuit, capsule isn't thickened, no signs of hepatoomentopexy, total echogenicity — normal, no focal changes. Portal vein — 0.9 cm in diameter, blood velocity 24.0–30.0 cm/s, hepatopetal undulating blood flow. Hepatic artery: blood velocity — to 80.0 cm/s, low-resistant type of blood flow. Induration the round ligament of the liver with the presence of effect of distal attenuation from it, without signs of recanalization. Gall bladder — common anatomical location, form, circuit, volume; wall — not thickened. Choledoch — 0.3 cm in diameter. No concrements. Pancreas — no liquid collectors in the projection of omental bursa, total volume isn't increased, smooth circuit, echogenicity is slightly increased, in the projection of the cervix — body not dilated Wirsung's duct is visualized (0.2 cm in diameter). Spleen — rejected. Closer to the anterior abdominal wall, near the border of the parietal peritoneum — 3 solid rounded formations, transversely located in the epigastrium, at a distance of up to 2.0 cm apart from each other, hypoechoic, with a smooth circuit, homogeneous structure, upon energy mapping at the "noise" border — with single loci of blood flow, moderately movable during instrumental palpation, movable upon respiratory excursion. Kidneys, adrenal glands — without visible pathological changes.

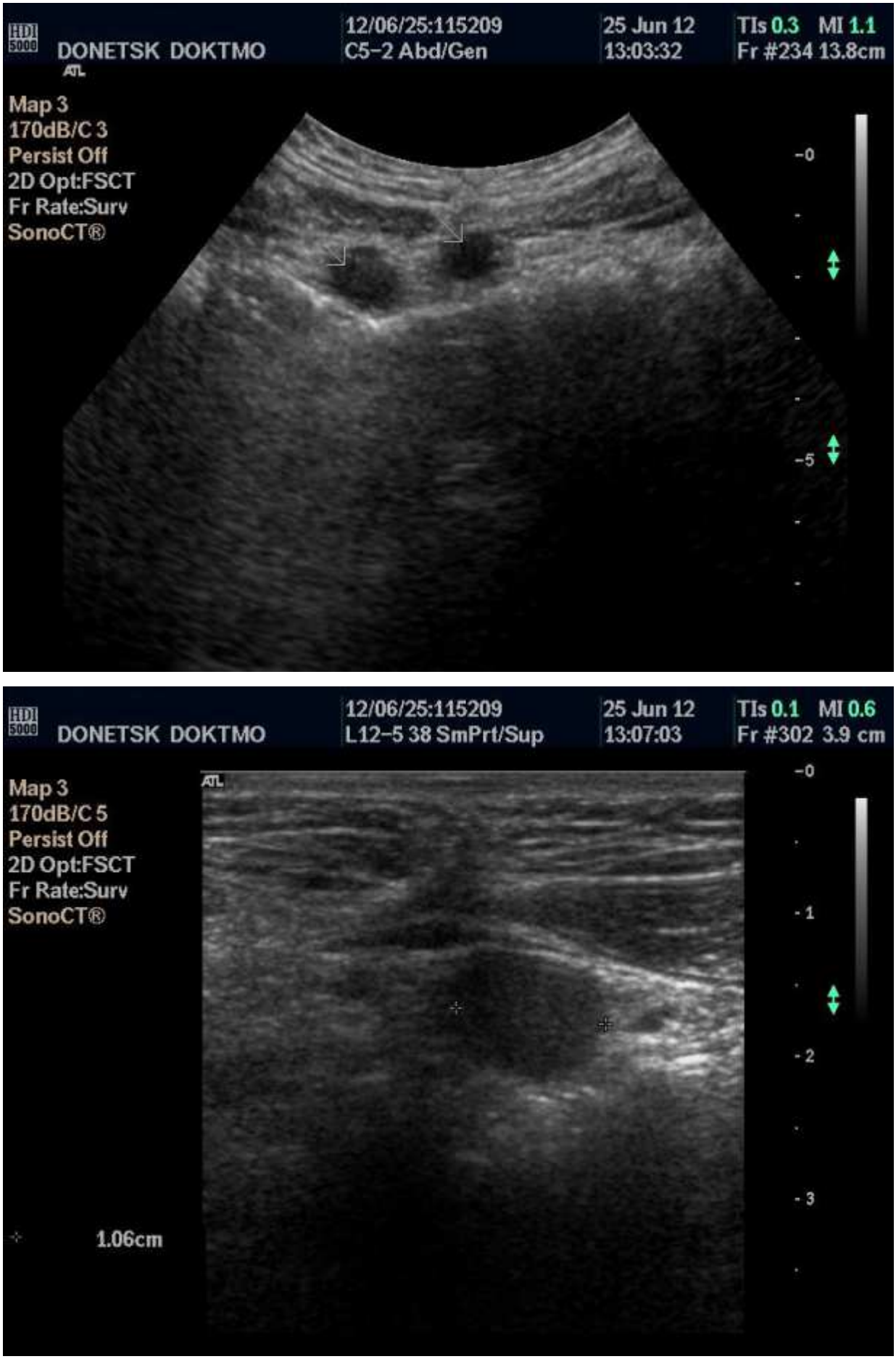

Fig. 4a, b. Ultrasound diagnostics of the organs of abdominal cavity. Closer to the anterior abdominal wall, near the border of the parietal peritoneum — 3 solid

rounded hypoechoic formations, transversely located in the epigastrium, with smooth circuits, homogeneous structure

Submandibular and parotid salivary glands on the left and right are uniformly increased in volume, including retroauricular portion. Thickened capsule, parenchyma — inhomogeneous, with slightly increased echogenicity, lobed, with minor linear echo-positive inclusions. No focal changes in the parenchyma. Excretory ducts are visualized without evidence of sialolithiasis and dilatation. Relative dilatation of outlet veins. Upon EC — vascularization closer to the increased one. Tumor formations — not detected. Near the left angle of the mandible — a single lymph node 1.6×0.8 cm with a smooth circuit and relative violation of differentiation of cortex medulla (echo-signs of lymphadenitis). Other groups of lymph nodes — within the normal range.

Multislice CT of the abdomen, retroperitoneum with intravenous bolus enhancement (March 15, 2012): liver — diffusely enlarged, with smooth, clear contours, homogeneous structure, additional formations and foci of pathological density aren't visualized in it. Size of the pancreas isn't changed, homogeneous structure, additional formations aren't found. Rounded formation with a homogeneous structure, clear and smooth contours of 2.1 cm in diameter (probably, additional slice) is detected in the bed of the rejected spleen. Adrenal glands are of normal shape, size, structure, additional formations aren't detected in them. Kidneys are of normal shape, size, position, additional formations aren't detected in them. cavity systems aren't expanded, concrements aren't visualized. Vascular pedicles of the kidneys — without changes. The ureters aren't dilated. In the abdominal cavity, directly under the anterior abdominal wall, 1.0–4.0 cm above the navel — 4 rounded formations of 1.1–1.6 cm in diameter; their size, number and structure haven't changed substantially in comparison with the previous examination on July 27, 2011. Soft-tissue formation with rather clear, smooth circuits of 2.7×2.1 cm in size is detected in the abdomen, under the right lobe of the liver. Above-described formations moderately uniformly accumulate contrast agent. Lymph nodes in the abdomen and retroperitoneum aren't increased. In the parametrial tissue on the left —

single lymph nodes thickened up to 0.9 cm in diameter. No destructive changes in the bone structures at the level of examination. Conclusion: nodular formations in the abdominal cavity without clear organ accessory, the nature of which is not fully understood currently. Additional slice of spleen. Diffuse changes in the liver.

Static scintigraphy of liver with Tc99m (June 2012): liver is of normal shape, with good concentration of the radiopharmaceutical and its relatively uniform distribution. Spleen isn't visualized (rejected). Conclusion: no scintigraphic signs of organic liver lesion.

Consultation of surgeon: formations require differentiation between lymphoid, neoplastic or another process and ectopic splenic parenchyma.

Considering normal activity of blood P-isoamylase upon increased index of blood α-amylase, we concluded that the increase in the latter was due to S-isoamylase. This made us to consult a dentist.

Consultation of dentist: no data on the presence of inflammatory and neoplastic diseases in the maxillofacial area and oral cavity. Lymph nodes are enlarged, painless, of normal consistency. Treatment isn't required.

Consultation of infectionist: taking into account anamnesis, data of laboratory and instrumental examination, formations detected in the abdominal cavity aren't associated with toxocariasis or echinococcosis. EBV-infection, stage of persistence. It's recommended to repeat CT in 3–6 months. Hemogram monitoring.

Consultation of hematologist: present rounded formations under the anterior abdominal wall haven't grown for one year (according to CT). However, determining their nature is possible only after a biopsy of formations (if possible — puncture one). It's necessary to conduct a testing for viral infections, urine inoculation of flora. Absolute norm in the peripheral blood (occasionally eosinophilia). DNA EBV in blood (++), antibodies of class IgG to nuclear antigen EBV and CMV are detected in the patient. Treatment by Valtrex 500 mg 2 times a day for 14 days, then 500 mg for 7 days is prescribed. Re-examination of DNA EBV after 2 months of therapy.

Consultation of oncologist: no data confirming the fact of malignant character of formations in abdominal cavity. More likely, they aren't tumors at all.

Puncture biopsy of formation in the abdominal cavity: splenic tissue in the biopsy material.

C_{am}/C_{cr} ratio: 0.6% (norm 1–4%).

Precipitation of blood amylase activity by polyethylene glycol 6000 is performed. Precipitation of 87% of amylase activity is detected (norm — up to 73%)

Considering the steady increase in indices of blood α-amylase upon normal activity of urine α-amylase and blood lipase, reduction of C_{am}/C_{cr} ratio, increased results of precipitation of amylase activity by polyethylene glycol 6000, we've already diagnosed macroamylasemia in the step II of the algorithm by J. E. Berk (1995) [9].

Clinical diagnosis:

Macroamylasemia. Chronic pancreatitis in remission. Chronic cholecystitis beyond remission. EBV-infection, stage of persistence. Splenosis.

Recommendations: observation (1 ultrasound diagnostics every 6 months).

In conclusion, it should be pointed out that the awareness of doctors on macroamylasemia and variants of splenic ectopy gives a chance in cases, being accidental discovery, to prevent unnecessary examinations and surgical interventions.

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Clinical observation of macroamylasemia on the background of splenosis due to the post-traumatic splenectomy (literature review and clinical observation)

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Key words: macroamylasemia, splenosis, post-traumatic splenectomy, chronic pancreatitis, hyperenzymemia

Article analyzes current data on macroamylasemia and splenosis, their etiology and diagnostics in particular. Authors presented their own clinical observation of a young woman who was diagnosed to have macroamylasemia on the background of splenosis due to the splenectomy after blunt abdominal injury. This is the first time such a combination has been described in the literature.