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БОЛЕЗНЬ КАСТЛЕМАНА. АССОЦИАЦИЯ С СИСТЕМНОЙ СКЛЕРОДЕРМИЕЙ

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Castleman's Disease. Association with System Scleroderma

Резюме

В статье представлено наблюдение редко встречающегося доброкачественного лимфопролиферативного заболевания — болезни Кастлемана, с выраженными системными симптомами. Клинический случай представляет интерес не только редкостью патологии, но и особенностями клинических проявлений, включающих паранеопластический псевдосклеродермический клинико-иммунологический синдром, который ранее в контексте болезни Кастлемана описан не был, синдром Рейно, выраженную легочную гипертензию и предполагаемый (не доказанный морфологически) вариант экстранодального поражения с ранее также не наблюдаемой его локализацией в стенке сигмовидной кишки.

Ключевые слова: *болезнь Кастлемана, склеродермия, синдром Рейно, легочная гипертензия*

Конфликт интересов

Авторы заявляют, что данная работа, её тема, предмет и содержание не затрагивают конкурирующих интересов

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Abstract

The article presents an observation of a rare benign lymphoproliferative disease — Castleman's disease, with pronounced systemic symptoms. The clinical case is of interest not only for the rarity of pathology, but also for the peculiarities of clinical manifestations, including paraneoplastic pseudosclerodermic clinical and immunological syndrome, which was not previously described in the context of Castleman's disease, Raynaud's syndrome, severe pulmonary hypertension and a suspected (not proven morphologically) variant of extranodal lesion with its previously also not observed localization in the sigmoid colon wall.

Key words: *Castleman's disease, scleroderma, Raynaud's syndrome, pulmonary hypertension*

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AB — antibodies, CD — Castleman disease, EBV — Epstein-Barr virus, HVCD — hyaline-vascular CD, PA DIA — diastolic pressure in pulmonary artery, DNA — deoxyribonucleic acid, IL — interleukin, BMI — body mass index, CT — computer tomography, PA — pulmonary artery, PH — pulmonary hypertension, MCCD — multicentric CD, MSCT — multispiral computed tomography, MRI — magnetic resonance imaging, PCCD — plasma cell CD, PET — positron emission tomography, IA — imaging agent, PA SYS — systolic pressure in pulmonary artery, SLE — systemic lupus erythematosus, CRP — C-reactive protein, DSD — diffuse scleroderma, CMVI — cytomegalovirus infection, TEECG — trans-esophageal echocardiography, EchoCG — echocardiography, ANA — antinuclear antibodies, EGF — epidermal growth factor, HHV-8 — herpesvirus simplex, type 8, HIV — human immunodeficiency virus, Ig — immunoglobulin, VEGF — vascular endothelial growth factor

Angiofollicular hyperplasia of lymph nodes, also known as Castleman disease (CD), is a rare benign lymphoproliferative disorder, which can develop to non-Hodgkin lymphoma and which is associated with a number of diseases — cancer and autoimmune disorders. The incidence of CD has not been established.

As for CD etiology, there are several hypotheses, including infectious and autoimmune origin. It is assumed that CD is associated with Epstein-Barr virus (EBV), human immunodeficiency virus (HIV) or herpes, type 8 (HHV-8). It has been proven that there is direct correlation between the level of blood IL-6 contributing to pathogenesis of inflammatory, autoimmune and oncological diseases, including rheumatoid arthritis, and clinical symptoms in patients with CD [1]. In pathogenesis of hyaline-vascular CD, probably like in other forms of CD, an important role is played by follicular dendritic cells, proliferation and dysplasia of which in combination with POEMS-syndrome (Polyneuropathy, Organomegaly, Endocrinopathy, M-protein, and Skin Changes) leads to expression of vascular endothelial growth factor (VEGF), promoting active vascular proliferation [2].

In terms of morphology, the following Castleman disease variants are distinguished: hyaline-vascular CD (HVCD), affecting over 70 % of cases, and plasma cell CD (PCCD), accounting for approximately 30 %; sometimes a mixed type is discussed as well. A CD mass is localised in chest (root of the lung) or mediastinum, less often in retroperitoneum, peripheral lymph nodes and very rarely — in lymphoid tissue of nasopharynx and tongue, in tonsils, orbits [3, 4]. Exclusive extranodal lesions are described in soft tissue of chest, neck and retroperitoneal space, limb muscles, periorbital area [5]. Clinical variants of CD include the most common local (unicentric) and more rare diffuse (multicentric) variants. Paucisyptomatic or asymptomatic local forms are associated mostly with lymphadenopathy in one anatomic area, while diffuse forms are manifested through clinical and laboratory symptoms and aggressive course of the disease [6, 7]. The multicentric clinical variant usually has a histological pattern of plasma cell CD and can be associated with HIV, HHV-8 or is characterised as idiopathic multicentric CD [8, 9].

In a majority of cases, HVCD does not have any clinical manifestations. Enlarged lymph nodes in various locations (external, visceral) can be single or can present as a chain of adjacent nodes. One out of ten patients has constitutional symptoms (fever, skin rash, loss of weight, dyspepsia, shortness of breath) and abnormal laboratory findings (hyperthrombocytosis, anemia, increased C-reactive protein levels, etc.). Mediastinal or retroperitoneal lymphadenopathy can be associated with signs of organ compression — cough, shortness of breath, pain, or can be an incidental finding during preventive examinations. On MRI or CT scans, a mass presents as a solid mass with a clear, even contour, which intensively accumulates the contrast agent during both vascular phases [10].

In local plasma cell CD, most often the disease affects abdominal lymph nodes (one or several) [3, 6, 7]; a majority of patients experience systemic clinical symptoms and abnormal laboratory findings. A rare variant of MCCD which affects elderly people is associated with marked constitutional symptoms, enlarged spleen/Banti's syndrome, peripheral lymphadenopathy, marked and various laboratory value abnormalities [7, 11]. Multicentric CD can terminate with remission, can have frequent recurrences, can be stable or can develop to malignant lymphoma.

Differential diagnosis of CD variants is possible with the help of morphological and immunohistochemical findings of a removed lymph node. Differential diagnosis of CD is challenging, also due to morphological features; it can resemble non-Hodgkin lymphoma, reactive lymphadenopathy, IgG4-associated lymphadenopathy [12, 13].

An important characteristic of CD is the ability of associated clinical conditions: systemic autoimmune disorders or cancer, such as non-Hodgkin lymphoma,

multiple plasmacytoma, Hodgkin lymphoma, amyloid disease, POEMS-syndrome, lymphoproliferative disorders associated with HIV infection or presence of HHV-8 vIL-6 [2, 14, 15].

The presence of autoimmune disorders is proven by occurrence of autoantibodies (antinuclear antibodies, anti-DNA antibodies, anti-thyroglobulin antibodies, antibodies against parietal cells and adrenal cells) in CD, development of autoimmune hemolytic anemia, autoimmune thrombocytopenia, glomerulonephritis [16]. Pronounced plasmacytosis in lymph nodes resembling plasma cell Castleman disease is sometimes observed in rheumatoid arthritis [17]. There are reports on multicentric CD resembling systemic lupus erythematosus (SLE) [18]. In SLE-associated lymphadenopathy, morphological signs of hyaline-vascular and plasma cell CD [19] were observed. The totality of clinical and, what is more important, morphological signs in SLE and CD allowed identifying a combination of these two diseases in a number of cases [20].

At the same time, we did not find reports on any cases of concomitant CD and other collagen disorders, specifically systemic sclerosis, whereas there are multiple cases of pseudosystemic sclerosis described in patients with multiple plasmacytoma, lymphoblastic lymphosarcoma, other lymphoproliterative and tumour processes [21].

Below is a case study.

21-year-old patient F. was admitted to Rheumatology Department complaining of hand pain, finger and toe blue and white discolouring in the cold, pain in right shoulder and cervical spine; shortness of breath with minimal physical activity, at rest, palpitations; recurrent temperature rises to 38.0–38.5°C (max. to 41°) with chills, associated with severe muscle pain, abdominal skin flaking; pronounced weakness, tiredness.

Since the age of approximately 12 years old, she has been noting enlarged lymph nodes on her neck with long-lasting, dry cough; the patient was examined and treated by phthisiologist with recovery. X-ray images showed a peripheral mass in her left lung, which was interpreted as residual changes of a specific process. The patient was allergic to antivirals.

According to the patient, she has been ill for 2 years, when after circumscribed abscess (quinsy exacerbation), treated with antibacterial and non-steroidal anti-inflammatory drugs, she had persistent periods of febrile and subfebrile fever, joint and muscle pain, enlarged lymph nodes on her neck. Approximately one and a half years ago, she noticed finger and toe discolouration (cyanosis, blanching).

Prior to admission to Rheumatology Department, she was examined in an oncology dispensary due to suspected non-Hodgkin lymphoma: lymphadenopathy of the neck and upper chest area, moderately enlarged spleen, single enlarged axillary and perineal lymph nodes. Her tongue was covered with painless aphthae.

At the same time, the patient was diagnosed with chronic EBV infection (active period) and primary latent CMV; she underwent specialised therapy. Complete blood count demonstrated mild neutrocytosis, moderate anemia, higher ESR value, 2.5-fold increase in C-reactive protein. Pharynx scraping came back with EBV DNA; blood draw tests showed mycoplasma pneumoniae IgA, IgG (low titre) and IgM (border-line titre); β-hemolytic streptococcus was isolated from pharynx. Urinalysis results showed leukocyturia, protein (0.21 g/L). Hypoalbuminemia (22.96 g/L), hypergammaglobulinemia (57.17 %) were found; serum and urine M-protein was not found. An ultrasound examination revealed enlarged lymph nodes on the neck (max. 46 × 15 mm). Chest, abdominal and pelvic CT results: intrathoracic lymphadenopathy (max. 24.7 × 7.1 mm), enlarged perineal, para-aortic, portal fissure, ileac, paracolic lymph nodes; enlarged liver (207 × 75 × 214 mm) and spleen (167 × 68 mm). PET CT results: moderately enlarged lymph nodes: jugular supraclavicular and axillary on both sides, also mediastinal, abdiminal, pelvic, retroperitoneal, femoroinguinal (max. 20 mm), diffuse imaging agent (IA) hyperfixation in hypertrophic lymphoid tissue of the pharyngeal lymphoid ring, diffuse IA hypermetabolism in parenchyma without any signs of focality.

Microscopic examination of the lymph node (Hematopathology Department of I. P. Pavlov First St. Peteresburg Medical University): the follicular pattern is preserved; all parts of the node contain lymphoid follicles of varying size with clear boundaries, they are separate from one another; several follicles have signs of fusion and an irregular germinal center, mantle area; germinal center zoning is preserved, with lost of macrophages; there are plasmic cell accumulations in between macrophages. Immunohistochemistry: lymphoid cells of follicular structures express CD20; germinal center cells express bc1-6, CD10, there is no bc1-2 expression in them; moderate T-cell (CD3+) inclusion, cells are located mostly between follicular structures; in a reaction with anti-CD23 antibodies, dendritic stroma in a majority of follicles is fragmentarily absent, in a reaction with anti-CD21 antibodies, this pattern is less pronounced; IgD marks the mantle area; MNDA+ cells are located loosely in peripheral sections of follicles; in a reaction with anti-IgLkappa and anti-IgLlambda antibodies, no signs of monotyping were found. Proliferation index (Ki-67) in follicular structures (germinal centers) is high, with max. 2 % outside the centers. The identified changes correspond to plasma cell Castleman disease.

A board of hematology specialists verified mild Castleman disease on the basis of histology and histochemistry

results, results of CT, ultrasound (enlarged lymph nodes, enlarged liver and spleen), presence of hypoalbuminemia, hypergammaglobulinemia, febrile body temperature rises and absence of M-protein in serum and urine.

Upon admission to Rheumatology Department, the patient was complaining of body temperature rise to 38 °C, neck pain (more to the right), visibly enlarged lymph nodes on her neck, muscle and joint pain.

The condition is satisfactory. Body composition is normosthenic. BMI = 22.05 kg/m^2. Skin is clean, pale, lips are slightly cyanotic. Hypomimia, pinched nose. Forehead skin does not form a fold. Hands and feet are cold to the touch, from time to time they turn white, then become cyanotic and/or erythematous, the condition is most vivid on 2–3 fingers **(Fig.)**.

Chill, finger and toe numbness. Scleredema. Her tongue is covered with single painless aphthae. Enlarged (up to 4 cm) solid painless cervical, submandibular, axillary lymph nodes can be palpated. By percussion, pulmonary sounds are above the lungs, breathing is vesicular, weak in lower parts on the right. Respiratory rate: 18 breaths per minute. BP right 100/70 mm Hg, BP left 95/70 mm Hg, heart rate is 79 bpm. Cardiac border is extended to the right; tone I — apical, clear, short systolic murmur; tone II is clearly above the pulmonary artery. Abdomen is soft, painless; liver edge is beyond the costal margin, spleen is not palpated. At palpation, cervical paravertebral points are slightly painful. Symptom of transverse hand, feet and right shoulder squeezing is positive.

Figure. *Patient F. Raynaud's syndrome*

Complete blood count: RBC 3.44×10^{12}/L, Hb 103 g/L, Ht 0.27, L. 11.2×10^9/L, bands 1 %, segm. 56 %, LYMPH 34 %, Mon 8 %, EOS 1 %, platelets 508×10^9/L, ESR 54 mm/h.

Urinalysis: relative density 1025, pH 5.5, protein 10 mg/L, WBC 6–7–10 HPF.

Coagulation profile: ART 66 s, PT 11.9 s, INR 1.01, APTT 34 s, fibrinogen 5.38 g/L, fibrinolytic activity 11 min, Quick's value 88.8 %, ethanol gelation test: neg., D-dimer 0.96 μg/mL.

Biochemical blood assay: GGT 109 U/L, LDH 305 U/L, ALAT 24 U/L, ASAT 32 U/L, alkaline phosphatase 243 U/L, creatine phosphokinase 55 U/L, creatine phosphokinase-MB 24 U/L, CRP 12.2 mg/L, total protein 106 g/L, albumin 34 g/L, glucose 4.2 mmol/L, total/direct bilirubin 8/1.4 μmmol/L, urea 4.2 mmol/L, uric acid 390 μmmol/L, Fe 6.0 μmmol/L, pro/BNP 1157 pg/mL, creatinine 62 μmmol/L. Blood electrolytes, lipid profile, thyroid hormones are within the normal range. Blood procalcitonin is below 0.5 ng/mL (negative).

No blood microflora growth was observed. Anti-HIV, HHV-8: negative. Anti-EB-VCA IgG AB: positive, IgM: negative, anti-early EBV protein AB (IgG): 60.6 U/mL (normal value: up to 40), anti-nuclear EBV antigen AB: 266 U/mL (normal value: up to 20).

Immunology examination: antinuclear AB IgG: positive, antinuclear antibodies: SS-A/Ro, SS-B/LA, RNP 70, Sm, RNP/Sm, centromere B, Jo-1: 5.8 (normal value: up to 1.2). Anti-aDNA antibodies: 56.1 (normal value: up to 25). Rheumatoid factor: 27 IU/mL (positive). Anti-SCL-70 IgG: negative. Anti-CPP IgG antibodies: 8.9 I/mL (negative). Lupus anticoagulant: 1.09 units (negative).

ECG. Sinus tachycardia (121 bpm). Vertical cardiac axis position. Impaired myocardium repolarization in apical and lower sections of LV. 24-hour ECG monitoring: tachycardia, 107–143 bpm (mean value: 120 bpm).

EchoCG, TEECG. Global myocardial contractility is preserved. Left atrial cavity is enlarged. Induration of mitral valve leaflets and subvalvular structures, aortic demilunes. Prolapsed anterior mitral valve leaflet up to 4.7 mm, mitral regurgitation. Prolapsed tricuspid valve leaflets, valve insufficiency; regurgitation of pulmonary artery valve. Signs of significant pulmonary hypertension (PA SYS = 66 mm Hg, PA DIA = 24 mm Hg).

Contrast-enhanced chest CT: pulmonary trunk 32 mm, right branch 18 mm, left branch 16.5 mm, even contrast accumulation along vessel length. Bilateral basal pleuropneumofibrosis, right-sided hydrothorax, hyperplastic subcarinal lymph nodes, cervical, supraclavicular, axillary lymphadenopathy. Conclusion: signs of pulmonary hypertension.

Abdominal, retroperitoneal and renal MSCT. Bilateral renal artery duplication, small amount of fluid in pelvis. Enlarged liver and spleen. Enlarged portal fissure, ileac, paracolic, perineal, para-aortic lymph nodes near renal arteries fuse to form a conglomerate of up to 28 × 18 mm. Uneven wall induration and unclear contours of sigmoid with regional lymphadenopathy, which should be differentiated between an inflammatory process and mass.

Colonoscopy did not reveal any organic pathology of sigmoid.

Ultrasound examination of abdomen and kidneys: enlarged liver and spleen, diffuse changes in hepatic and pancreatic parenchyma, contour deformity of gall bladder, fluid (17 mm thick) in right pleural space.

Final clinical diagnosis. Mild Castleman disease, plasma cell type with systemic symptoms, associated with progressive systemic sclerosis — high-activity subacute systemic scleroderma with vascular involvement (Reynaud's syndrome), skin involvement (scleredema, mask-like face), pulmonary involvement (pulmonary arterial hypertension, bilateral basal pleuropneumofibrosis, right-sided hydrothorax), cardiac involvement (cardiomyopathy). Extranodal lesion (sigmoid wall mass)? ANA immunologic activity. Chronic Epstein-Barr virus infective, inactive.

Medication: prednisolone (35 mg/day) together with cyclophosphan 400 mg (with further dose escalation to 600 mg) every two weeks; pentoxifylline 600 mg/day in cycles, sildenafil 25 mg three times daily, symptomatic joint pain management.

After a week of therapy, joint and muscle pain passed off, body temperature dropped to low subfebrile values. In 3 months, constitutional symptoms disappared; shortness of breath and clinical signs of Reynaud's syndrome improved; the size of palpable lymph nodes normalised; PA SYS dropped from 66 mm Hg to 54 mm Hg. CT scans demonstrated normal size of thoracic, abdominal and retroperitoneal lymph nodes; spleen CC dimension reduced from 164 mm to 140 mm; sigmoid wall thickness and contours normalised.

Discussion

We presented a case of a rare condition — Castleman disease, in its even more rare form (according to our own data, since it has not been described anywhere), in combination with a systemic connective tissue pathology — systemic sclerosis.

Like any other rheumatoid disorder, systemic scleroderma can be a clinical mask for a number of pathological processes and can hide metabolic disorders, endocrinopathies (porphyria, phenylketonuria, Wilson disease, Werner syndrome, Sheehan syndrome, micromegaly, hypothyroidism), a group of tumours — solid tumours, chronic leukemias, lymphoproliterative processes [21], which are manifested at various stages of advanced symptoms of systemic sclerosis.

In this clinical case, lymphatic pathology was observed 5–6 years before advanced disease: enlarged cervical lymph nodes and a peripheral mass in the left lung, but at that time they were interpreted as signs of possible TB.

Symptoms of systemic sclerosis (Reynaud's syndrome, later joined by hypomimia, pinched nose, scleredema) appeared in patient F. after a circumscribed abscess, which was possibly caused by EBV and was associated with hyperthermia, muscle and joint pain, loss of weight, excessive sweat, diffuse lymphadenopathy, enlarged liver and spleen, high CRP levels, anemia, leukocytosis, hyperthrombocytosis, higher ESR, hypergammaglobulinemia; all these symptoms together with clinical signs of an autoimmune (sclerodermic) syndrome and confirmed plasma cell variant were verified as Costleman disease with systemic symptoms, resembling multicentric (diffuse) CD — MCCD [4, 5], which usually requires glucocorticoids and chemotherapy [17]. Taking into account anti-HIV (-) and HHV-8 (-), the origin of the disease in patient F. is probably associated with EBV infection.

Also, bilateral basal pleuropneumofibrosis, pulmonary hypertension (PH) with high PA pressure, enlarged pulmonary vessel diameter, tricuspid valve insufficiency, were diagnosed.

Overall, the incidence of PH in diffuse scleroderma varies from 9 % to 65 %; PA develops similar to other vascular disorders (Reynaud's syndrome with sclerodermic renal crisis) as a result of progressive re-modelling of small to moderate pulmonary vessels because of an endothelial damage and impaired regulation of intercellular interactions [22, 23]. The mechanism of PH based on an increase in VEGF expression and synthesis by plasmatic cells of light Ig λ-chains, productive vasculitis with microvascular bed with obliteration (sclerosis) was described in Castleman disease with POEMS-syndrome [24].

An immunological examination revealed presence of specific markers of sclerodermia — antinuclear antibodies, anti-antigen SS-A/Ro, SS-B/LA, RNP 70, Sm, RNP/Sm antibodies, anti-centromere B, Jo-1 antibodies, the value of which was 5 times higher than the normal value; also, rheumatoid factor was observed.

MSCT results were of interest: uneven wall induration and unclear contours of sigmoid, which could be an inflammatory process or a tumour in its wall. Extranodal involvement in CD was assumed. Unfortunately, the patient refused to give her consent for endoscopic or laparoscopic examination. However, regression (disappearance) of this mass during therapy makes it possible to assume an extranodal mass with rare (previously not described) location in sigmoid wall.

Thus, development of a number of clinical presentations of diffuse scleroderma in a patient with Castleman disease: skin induration on hands, forehead with hypomimia, pinched nose, immunology shifts in the form of antinuclear antibodies and rheumatoid factor, Reynaud's syndrome and pulmonary hypertension, allowed diagnosing pseudosclerodermia paraneoplastic syndrome — an associated form of disorder with the leading role of CD as a lymphoproliterative process with an active, progressing disease (which can develop into a malignancy in a number of cases), however, the last fact is not essential for paraneoplastic syndrome.

Conclusion

This case study of plasma cell variant of Castleman disease with marked systemic symptoms is of interest not only because this pathology is rare, but also due to its clinical manifestations, including pseudosclerodermia paraneoplastic clinical and immunological syndrome, which has never been described in connection with CD; Reynaud's syndrome; marked pulmonary hypertension and suspected (without morphological confirmation) extranodal involvement of sigmoid wall.

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All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication.

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