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ОПЫТ ДВУХЛЕТНЕГО НАБЛЮДЕНИЯ ПАЦИЕНТА С БОЛЕЗНЬЮ ДЕРКУМА НА ФОНЕ ТЕРАПИИ МЕТОТРЕКСАТОМ

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Experience of Two-Year Observation of a Patient with Dercum Disease During Methotrexate Therapy

Резюме

Болезнь Деркума, также известная как болезненный липоматоз, нейролипоматоз, синдром Андера — редко встречающееся заболевание, главным клиническим проявлением которого является наличие болезненных образований подкожной клетчатки, с локализацией в различных частях тела: на конечностях, туловище, ягодицах. К возможным этиологическим факторам относят генетические мутации, наличие аномальных клеточных белков, эндокринные нарушения, изменения со стороны нервной системы. Чаще это заболевание встречается среди женщин старше 35 лет. Случаи развития болезни Деркума у детей и подростков встречаются редко. Пациенты с этим заболеванием зачастую имеют избыточную массу тела. Выделяют 4 типа болезни Деркума: генерализованная диффузная, генерализованная узловая, локализованная узловая, юкста-артикулярная. В некоторых случаях повышаются островоспалительные маркеры: скорость оседания эритроцитов, С-реактивный белок. В представленном клиническом случае также отмечен высокий уровень фактора некроза опухоли-α со снижением в динамике, что требует дальнейшего изучения прогностических возможностей данного биомаркера в оценке активности заболевания. Гистологические исследования подкожных элементов у пациентов с болезнью Деркума не имеют специфических изменений (морфологическая картина соответствует липоме). В литературе обсуждаются различные методы терапии, включающие липосакцию, массаж, а также нестероидные противовоспалительные препараты, глюкокортикоиды, метотрексат и др. Представленный клинический случай описывает раннюю диагностику болезни Деркума с проведением дифференциальной диагностики с панникулитами другой этиологии и достижение стойкой ремиссии на фоне терапии метотрексатом у пациентки 42 лет с жалобами на наличие болезненных локальных узелковых образований кожи верхних и нижних конечностей разного размера.

Ключевые слова: *болезнь Деркума, липоматоз, метотрексат, панникулит*

Конфликт интересов

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

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Abstract

Dercum disease, also known as painful lipomatosis, neurolipomatosis, Ander's syndrome, is a rare illness. The main clinical manifestation of this disease is the presence of painful formations of subcutaneous tissue, localized in various parts of the body: on the limbs, trunk, buttocks. Possible etiological factors include genetic mutations, the presence of abnormal cellular proteins, endocrine disorders, changes in the nervous system. This disease is more common among women over 35 years old. Cases of Dercum disease in children and adolescents are rare. Patients with this disease are often overweight. There are 4 types of Dercum disease: generalized diffuse, generalized nodular, localized nodular, juxta-articular forms. In some cases, acute inflammatory markers increase: the erythrocyte sedimentation rate, C-reactive protein. A high level of tumor necrosis factor-α with a decrease over time was also noted in the presented clinical case, it requires further study of the prognostic capabilities as a marker of disease activity. Histological examination of subcutaneous elements did not reveal specific changes (the morphological picture corresponds to lipoma). Various methods of therapy are discussed in the literature, including liposuction, massage. Non-steroidal anti-inflammatory drugs, glucocorticoids, methotrexate also may be used. *The clinical case presents a 42-year-old female patient with complaints of the presence of painful local nodular skin lesions of various sizes on the upper and lower extremities. We describe the early diagnosis of Dercum disease with differential diagnosis with panniculitis of other etiologies and the achievement of stable remission against the background of methotrexate therapy.*

Key words: *Dercum disease, lipomatosis, methotrexate, panniculitis*

Conflict of interests

The authors declare no conflict of interests

Conformity with the principles of ethics

The patient consented to the publication of laboratory and instrumental research data in the article «Experience of Two-Year Observation of a Patient with Dercum Disease During Methotrexate Therapy» for the journal «The Russian Archives of Internal Medicine» by signing an informed consent

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ANCA — anti-neutrophil cytoplasmic antibodies, DD — Dercum disease, CRP — C-reactive protein, TNO-α –tumour necrosis factor-α

Introduction

Dercum disease (DD), also known as lipomatosis, lipomatosis of the nerve, Ander syndrome, is a rare disease of unknown origin, the main characteristic of which is painful subcutaneous tissue lesions in various locations [1]. The first to describe this pathology was American doctor Francis Xavier Dercum, who in 1888 published two articles and proposed the term "lipomatosis dolorosa" for the disease, which was later named after him [1]. There is no specific genetic basis for DD. However, it is argued that there is family predisposition for "lipomatosis dolorosa"; this condition was described in immediate family members who had liposome proteins of a specific structure. Also, in 1973 Cantu J.M. et al. proposed to characterise DD as an autosomal dominant disease; however, in opposition to this, numerous authors point out the sporadic nature of this pathology [1]. Initially, endocrine disorders (dysfunctional thyroid gland, pancreas, hypophysis) were believed to be the aetiological factor of DD. However, in the first half of the XX century this idea was abandoned because there were no clinically significant laboratory abnormalities of the endocrine system [2-6].

One of the most significant manifestations of the disease is a very intense pain syndrome, which can be associated with higher activity of the sympathic nervous system, resulting from the presence of such provoking factors as hypoxia, production of some substances (protons, serotonin, substance P, etc.), which affect pain receptors, vasospasm, inflammatory reactions, necrosis [7].

Currently, lipid metabolism defects attract attention as an element of DD pathogenesis, but this mechanism is not clear. In their academic paper, Blomstrand R. et al. (1971) described decreased synthesis of monounsaturated fatty acids in affected adipose tissue vs. healthy tissue. However, another study contains opposite results: monounsaturated fatty acid levels were higher in patients with DD (Fagher B. et al., 1991) [2, 8].

There are reports of decreased reaction of the affected adipose tissue to noradrenaline and anti-lipolytic effects of insulin [5, 9].

In periarticular DD, adipose fascia inflammation impairs lymph flows in these areas, causing fluid accumulation in interstitial tissue and fascia induration and development of fibrosis around fat lobules and making them palpable. Pain in these lesions is a result of inflammation in fascia and nerves. It is also believed that development of this disease can be related to poor tissue regeneration following a traumatic injury, which causes chronic inflammation and damage to adjacent structures [10].

The incidence is higher in females at a ratio of 5–30:1 [11]. Disease manifests at the age of 35–50 years old. There are just few reports on DD in children and adolescents [12].

In 1901, J. Roux et al. were the first to propose diagnosis criteria, which included four clinical symptoms:

- Painful subcutaneous lesions;
- Generalised obesity;
- Asthenic syndrome;
- Mental symptoms (depression, dementia, confusion) [13].

Later, these criteria were modified: mental symptoms and asthenia were removed, probably because these symptoms were observed in a majority of patients with DD. In 1910, Stern H. separated two fundamental signs — obesity and painful adipose lesions [14].

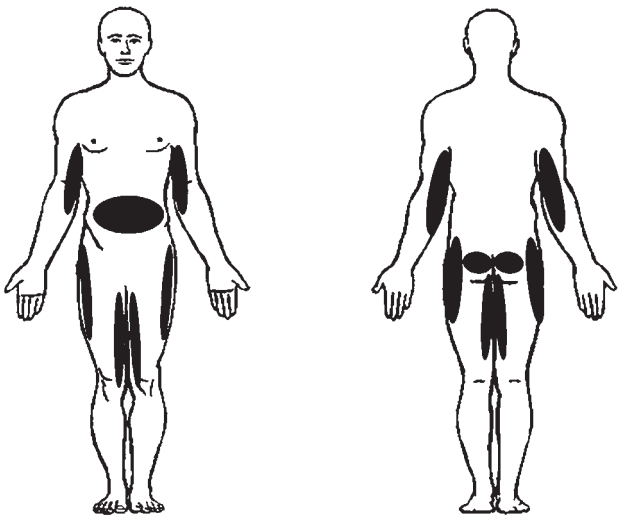

Figure 1A. *The most common localization of the formations in Dercum's disease (cited from [18]).*

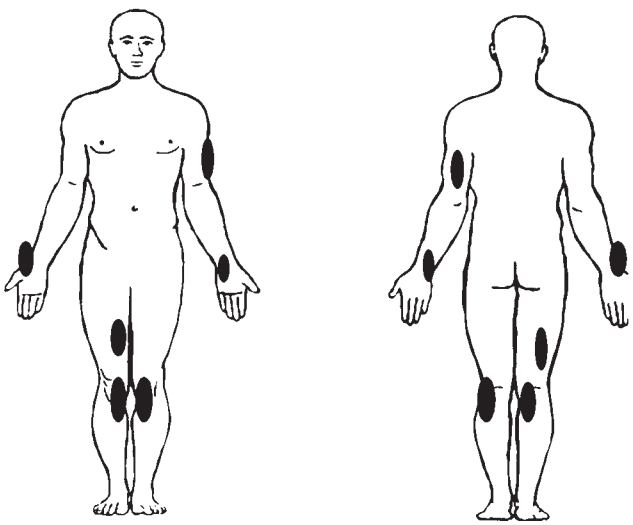

Figure 1Б. *Localization of the formations in the presented patient*

Emotional fluctuations and asthenic syndrome are considered to be sequelae of the existing pain syndrome and obesity, which can cause sleep disturbances and fatigue [15].

DD is associated with severe burning pain; however, some patients experience mild discomfort up to paroxysmal pain episodes. Usually, skin above lesions is normal.

Lesions can be located in any part of the body; gluteal region involvement is reported in 70 % of cases [1]. According to a questionnaire by Herbst K.L. et al. (2007) of over 100 patients with confirmed DD, this disease can affect buttocks, extremities, and trunk (Fig. 1A) [16].

Trentin C. et al. (2008) describe a case where DD caused mastalgia in a patient, who was suffering from multiple painful lesions in her breast [17].

The risk group of this disease includes individuals with various metabolic disorders (overweight, impaired glucose tolerance). In addition to painful lesions in adipose tissue, patients with DD can experience an array of symptoms. These include weakness, bruising, sleep disturbances, shortness of breath, joint pain, nerve and mental symptoms, such as mood swings, depression, epilepsy, disorientation, and dementia [5]. Neurological symptoms are often associated with metabolic disorders in obese patients, e.g. diabetic neuropathy. There is a report of septic shock in a patient with DD caused by lipoma necrosis after lymphatic oedema resulting from constriction of lymph and blood vessels in the affected area [19].

Literature sources contain contradictory information on the role of inflammatory biomarkers in DD diagnosis: increased C-reactive protein (CRP) levels were recorded in 33 % of patients, while erythrocyte sedimentation rate was higher than normal values in 38 %. However, direct correlation between DD and high values of these parameters has not been proven, because some patients participating in the study had autoimmune comorbidities, which could cause higher ESR and CRP values [16].

According to Herbst K.L. et al. (2009), fine-needle aspiration did not show any morphological differences between DD lesions and lipomas. Biopsy samples contained excessive connective tissue [1].

Currently, there is no approved unified classification of DD. Three versions of DD classification are used. The first one is the classification by V. Giudiceandrea (1900), where three types of pathologies were identified (Table 1) [20].

A modification of the mentioned classification is the classification by J. Roux et al. (1901) (Table 2) [13].

The most up-to-date is the classification proposed by E. Hansson as modified by Kosseifi S. et al. (2010) (Table 3).

Table 1. *Classification of Decrum disease types (according to V. Giudiceandrea) [20].*

Type	Description
Type 1. Nodal form	Localization of lipomas of various sizes on the upper and lower extremities, back, chest. Formations can merge with each other
Type 2. Diffuse form	Diffuse pain in adipose tissue of a symmetrical nature
Type 3. Mixed form	The presence of fatty formations and diffusely painful adipose tissue at the same time

Table 2. *Classification of Decrum disease types (according to J. Roux et al.) [13]*

Type	Describtion
Type 1. Nodal form	Many painful lipomas
Type 2. Limited diffuse type	Painful fatty deposits (often on the inside of the knees and/or thighs)
Type 3. Generalized diffuse type	Diffuse pain in adipose tissue (often in the limbs and trunk)

Table 3. *Classification of Decrum disease types (according to E. Hansson et al.) [14]*

Type	Describtion
Type 1. Generalized diffuse form	Painful sensations in adipose tissue, very small fat deposits localized in all areas of the body are determined. Pain can occur in areas without visible compactions
Type 2. Generalized nodular form	Painful sensations at the site of localization of lipomas located in several parts of the body
Type 3. Localized nodular form	Painful lipomas in certain areas of the body
Type 4. Juxta-articular form	Painful folds of fat inside or around large joints (knees, hips or elbows)

DD should be differentiated from fibromyalgia, cellulitis, endocrine disorders, primary mental disorders, multiple symmetrical lipomatosis (Madelung's disease), multiple family lipomatosis, Proteus syndrome and benign adipose tissue tumours [12].

Clear guidelines for the management of DD are not available, and healthcare providers have to select a strategy on their own. In some cases, lipoplasty has favourable effects with pain syndrome regression, confirmed in a study by Hansson E. et al. (2012) [14]. Another non-drug therapy is massage of adipose tissue and fascia, which sometimes also helps to reduce pain syndrome [21].

Given the presence of pain syndrome, drug therapy involves pain management: use of intralesional and intravenous lidocaine, non-steroidal anti-inflammatory drugs. Also, therapy can include pregabalin, interferon α-2b, glucocorticoids, metformin, as well as infliximab and methotrexate [1].

DD is a very rare finding (less than 100 PubMEd publications, access date: August 17, 2024), since there are no large-scale studies of this disease. Available publications are either clinical case studies or literature reviews. In Russian literature, there are just individual clinical cases of this condition [22].

The objective of this clinical case study is to demonstrate the experience with DD diagnosis and successful methotrexate therapy.

Clinical case study

On May 04, 2022, a female patient, 42 years old, visited MEDSI Medical Centre in Michurinskiy Avenue (Moscow) and complained of painful subcutaneous lesions in her right knee and proximal phalanges of her right hand, low-grade fever for a month, episodes of significant weakness, and pain in her both feet. She was examined by a surgeon, thyroid specialist, rheumatologist. Subcutaneous lesions appeared within the past month. Also, the patient reported short-term painful episodes of weakness with fever up to 37.0 °C.

According to the medical record, the patient has degree 1 obesity (158 cm, 75 kg, BMI 30 kg/m^2), insulin resistance, hypothyroidism caused by chronic autoimmune thyroiditis. She takes L-thyroxine prescribed by the thyroid specialist.

The patient used unprescribed non-steroidal anti-inflammatory gels; no favourable effect. Initial examination: satisfactory condition; clear skin and visible mucosa; joints: unremarkable. Blood pressure: 132/65 mm Hg, heart rate: 78 bpm. Respiratory and GI systems: unremarkable. As for the objective status, the patient had oedema of both shanks (up to the lower third), palpable local nodular lesions of the skin of upper and lower extremities (various diameter, the largest measuring up to 3 cm), moderately painful (Fig. 1B). Calcaneal region allodynia was diagnosed. Additional examinations were performed.

Complete blood count and urinalysis results were normal. Blood biochemistry demonstrated 3-fold increase in CRP values to 16.39 (reference values are provided in brackets: 0–5) mg/L (Table 4), uric acid to 454.6 (142–340) μmol/L. Tumour necrosis factor-α (TNF-α) of 12 (0–6) pg/mL was recorded. Creatinine, liver enzymes and glucose levels were normal.

An ultrasound examination of soft tissue of the knee showed irregular areas of lower echogenicity, measuring 30x9x36 mm, 2 mm deep from the skin surface. An ultrasound examination of the proximal phalanges of the third finger of the right hand (dorsal surface) and second finger of the right hand (palmar surface) showed similar changes, measuring 5×4×3 mm. Colour Doppler visualisation: unremarkable. The patient did not have synovitis of the knee and proximal phalanges of the second and third fingers of the right hand.

Right knee CT: clinically unremarkable.

During outpatient follow-up for two months, the patient had increasing pain in individual lesions on her fingers, neuropathic pain in heel region (Table 5), significant weakness lasting for over two hours, which prevented the patient from doing household chores. The patient managed weakness on her own.

When the clinical representation of the disease was analysed, episodes of asthenic, pain syndrome, painful subcutaneous lesions (panniculitis syndrome) were noted. Differential diagnoses ruled out TB, parasitic infections, sarcoidosis, upper and lower extremity panniculitis.

Abdominal ultrasound revealed gallbladder polyps. Mammography, gastroscopy and colonoscopy, as well as skeletal examination to rule out rare bone conditions did not show any pathologies.

There were no markers of autoimmune pathology (anti-neutrophil cytoplasmic antibodies (ANCA), cyclic citrullinated peptide antibodies, antinuclear antibodies, HEp-2 cell antinuclear antibodies).

Based on the medical record, physical examination results, DD was added to differential diagnosis.

Based on the clinical, laboratory and instrumental data obtained during the two months after the initial visit, the final diagnosis was made: Dercum disease of the upper and lower extremities (onset in April 2022), periarticular form, associated with psychopathic pain syndrome and moderate inflammation (high CRP levels). Primary hypothyroidism due to chronic autoimmune thyroiditis, medically compensated. Degree 1 obesity.

Methotrexate therapy was initiated: 15 mg SC once a week, then the dose was increased in 5 mg increments once every three weeks to 25 mg. L-thyroxine was continued under supervision of a thyroid specialist. Three months later, favourable effects were observed: clinical signs disappeared, CRP levels normalised, and TNO-α decreased to 8 ng/mL.

In this clinical case study, methotrexate was discontinued after stable remission for one year. The patient was in remission for six months without GC therapy.

The patient is followed up by a rheumatologist and undergoes follow-up laboratory blood tests (complete

Table 4. *Investigations*

Test, units of measurement	The first visit	The second visit (in 1 month)	Reference values
C-reactive protein, mg/l	25,7	7,4	0-5,0
Antibodies to complement factor C1q, U/ml	0,84		0-10,0
C3 complement component, g/l	1,7		0,9-1,8
C4 complement component, g/l	0,46		0,1-0,4
Interleukin-1 beta, pg/ml		<5,00	<5,00
Interleukin-6, pg/ml		2,0	0-5,9

Table 5. *Diagnostic criteria for chronic neuropathic pain (cited from [23])*

	Diagnosis points	What the patient had
A.	A.1. History of disease or injury to the somatosensory nervous system. A.2. Neuroanatomically logical (dermatomal) distribution of pain.	-
B.	The pain is accompanied by the presence of sensory symptoms with neuroanatomical distribution.	-
C.	Additional diagnostic tests may confirm damage or disease of the somatosensory nervous system that explains the pain.	-
D.	The pain is not explained by another medical condition that causes chronic pain.	+

Diagnostic criteria:
Presence of persistent or recurrent pain lasting ≥3 months and presence of at least points A and D.
The presence of points B and C increases the likelihood of the diagnosis.

blood count, CRP, alanine aminotransferase, aspartate aminotransferase, creatinine, glucose, glicated hemoglobin, thyrotropic hormone, urinalysis) once every three months; the results are within the normal range. In this case, the prognosis is favourable due to the early diagnosis, timely therapy initiation and good response to treatment.

Discussion

DD is a diagnosis by exclusion, an important aspect of which is differential diagnosis with numerous conditions. First of all, these are a group of panniculitis conditions [23]. In general, panniculitis is an inflammatory disease with extensive subcutaneous tissue involvement; also, the process can affect internal organs and locomotor system [24]. The distinguishing characteristic of DD is that the skin in visually unchanged, as opposed to erythema nodosum in sarcoidosis; also, there are no discharges on the skin, as in Weber-Christian disease (Table 6). Patients with DD do not show inflammatory changes in lesions on imaging (ultrasound, MRI).

To diagnose DD (criteria by Stern H., 1910), two symptoms must be present: generalised overweight or obesity and chronic pain in adipose tissue lasting for over three months. Both characteristics were present in this clinical case study, and the patient was diagnosed with the condition after a comprehensive differential diagnostic search.

DD is a diagnosis by exclusion, because the combination of symptoms described in this clinical case study is unique and extremely rare.

There are no specific laboratory tests to suspect DD. This patient had elevated TNO-α levels. Currently, there is no information on the pathogenetic correlation between TNO-α and DD. This can be an area of studies as a marker of this disease. The patient in this case study had a high CRP level, which normalised with the therapy; however, not all patients with DD have elevated CRP values [16].

Therefore, DD can be diagnosed in obese patients suffering from chronic pain in the adipose tissue, provided any other aetiology of changes is ruled out.

Table 6. *Diagnostic criteria for Weber-Christian panniculitis [25]*

Criteria		What the patient had
fever 38–39°C	1	–
the presence of dense painful formations mainly on the trunk, buttocks, thighs and limbs	1	+
joint pain	1	+
joint swelling	1	–
fatigue, weakness	1	+
headache	1	–
nausea	1	–
diarrhea	1	–

Diagnosis: triad — the presence of painful subcutaneous formations, fever, constant recurrence of these symptoms

Conclusion

Thus, a combination of panniculitis syndrome and significant asthenia and neuropathic pain is a specific manifestation of DD. DD should be differentiated from rheumatologic conditions, sarcoidosis, panniculitis of other origin. Awareness of healthcare providers of DD will ensure timely diagnosis and patient referral to a specialist for titration of therapy.

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Яровой М.Д.: написание статьи, анализ клинического случая, обзор литературы, перевод на английский язык

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Author Contribution:

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

Marshala S.N.: case study selection, selection and processing of the visual materials, text editing.

Iarovoi M.D.: article writing, literature review, case study analysis, translation into English

Semeniakin I.V.: text editing Reznik E.V.: article writing, text editing

Reznik E.V. — idea, leadership, work organization, edition

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