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А.Ю. Черников*, С.А. Кабанова, А.В. Дьяков

Кафедра клинической иммунологии, аллергологии и фтизиопульмонологии ФГБОУ ВО Курский государственный медицинский университет Минздрава РФ, Курск, Россия

КЛИНИЧЕСКИЙ СЛУЧАЙ ГЕНЕРАЛИЗОВАННОГО САРКОИДОЗА С ПРЕИМУЩЕСТВЕННЫМ ПОРАЖЕНИЕМ СПИННОГО МОЗГА

A.Y. Chernikov*, S.A. Kabanova, A.V. Dyakov

Department of Clinical Immunology, Allergology and Phthisiopulmonology, Kursk State Medical University of the Ministry of Health of the Russian Federation, Kursk, Russia

A Clinical Case of Generalized Sarcoidosis with a Predominant Lesion of the Spinal Cord

Резюме

Саркоидоз, как системный эпителиоидно-клеточный гранулематоз, может сопровождаться поражением не только внутригрудных лимфатических узлов и лёгких, но и других органов, в частности, центральной нервной системы и периферических лимфатических узлов. В спектре экстраторакальных поражений саркоидоз спинного мозга встречается лишь в 6-8 % случаев всех поражений мозга. Представленный клинический пример иллюстрирует поражение спинного мозга на уровне грудного отдела, хотя в литературе чаще описывается поражение шейного отдела. Заболевание сопровождалось саркоидозом внутригрудных лимфатических узлов с быстрой спонтанной регрессией и саркоидозом надключичного лимфатического узла. Диагноз был подтвержден после биопсии периферического лимфоузла. Саркоидоз спинного мозга у данного пациента характеризовался быстрой регрессией на фоне парентерального введения дексаметазона в течение 14 дней с последующим переводом на таблетированные формы преднизолона. Положительная динамика саркоидоза спинного мозга опровергла предположение о наличии саркоидной реакции в лимфатических узлах на фоне опухоли спинного мозга. Использование курса реабилитационных методик способствовало восстановлению работоспособности.

Ключевые слова: *генерализованный саркоидоз, саркоидоз спинного мозга, саркоидоз центральной нервной системы, реабилитация.*

Конфликт интересов

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

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*Контакты: Александр Юрьевич Черников, e-mail: ale-cherny@yandex.ru
*Contacts: Alexander Y. Chernikov, e-mail: ale-cherny@yandex.ru
ORCID ID: https://orcid.org/0000-0002-2045-9607

Abstract

Sarcoidosis, as systemic epithelioid cell granulomatosis, can be accompanied by damage not only to the intrathoracic lymph nodes and lungs, but also to other organs, in particular, the central nervous system and peripheral lymph nodes. In the spectrum of extrathoracic lesions, spinal cord sarcoidosis occurs only in 6-8 % of cases of all brain lesions. The presented clinical example illustrates the lesion of the spinal cord at the level of the thoracic region, although the literature more often describes the lesion of the cervical region. The disease was accompanied by sarcoidosis of the intrathoracic lymph nodes with rapid spontaneous regression and sarcoidosis of the supraclavicular lymph node. The diagnosis was confirmed after a peripheral lymph node biopsy. Spinal cord sarcoidosis in this patient was characterized by rapid regression against the background of parenteral administration of dexamethasone for 14 days, followed by transfer to tablet forms of prednisone. The positive dynamics of spinal cord sarcoidosis refuted the assumption of the presence of a sarcoid reaction in the lymph nodes against the background of a spinal cord tumor. The use of a course of rehabilitation techniques contributed to the recovery of working capacity.

Keywords: *generalized sarcoidosis, spinal cord sarcoidosis, central nervous system sarcoidosis, rehabilitation.*

Conflict of interests

The authors declare no conflict of interests

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ACE — angiotensin-converting enzyme, ALT — alanine aminotransferase, APTT — activated partial thromboplastin time, AST — aspartate aminotransferase, BMI — body mass index, BP — blood pressure, CD — cluster of WBC differentiation, CEA — carcinoembryonic antigen, CT — computed tomography, ENMG — electroneuromyography, ESR — erythrocyte sedimentation rate, FDG — fluorodeoxyglucose, HIV — human immunodeficiency virus, HR — heart rate, INR — international normalized ratio, L — lumbar, Max — maximum, MRI — magnetic resonance imaging, PSA — prostate specific antigen, PET — positron emission tomography, RF — respiratory failure, RR — respiratory rate, RV — reference values, S — sacral, SUV — standardized uptake value, T3 — triiodothyronine, T4 — thyroxine, Th — thoracic, TSH — thyroid stimulating hormone

Introduction

Sarcoidosis, as a systemic epithelioid granulomatous disease, may not be limited to only the respiratory system; it may be localized in other organs, such as skin, subcutaneous tissue, peripheral lymph nodes, bones, joints, kidneys, myocardium, eye, or nervous system. Extrathoracic signs of sarcoidosis generally include skin manifestations, however, the lesions of central nervous system and peripheral nerves are found in 5–10 % of cases [1]. Among the patients with a generalized process with the nervous system involvement the predominating group are both middle-aged female patients aged 35–60 with stage 2 or 3 pulmonary sarcoidosis, often with the disease relapse, and young male patients up to 35, with new onset stage 1 or 2 pulmonary sarcoidosis. In such cases, other localizations of the disease are often found only in cervical or submandibular peripheral lymph nodes [1]. Most case reports on central nervous system sarcoidosis describe predominant brain lesion, however, changes in spinal cord are found in 6-8 % of patients [2]. At the same time, there are no descriptions of a generalized damage to the central nervous system due to sarcoidosis. According to the literature sources, clinically intact lesions of the nervous system were detected during autopsy in 15–25 % of patients with generalized sarcoidosis. 10–20 % of patients probably had isolated neurosarcoidosis, however, in 84 % of cases, neurosarcoidosis was the first sign of the disease with the subsequent development of symptoms typical of a generalized process [3]. Sarcoidosis of the spinal cord is considered to be a rare disease. N. Soni (2019), using the sample of 18 individuals, concludes that the detection of this pathological condition is more often accompanied by the changes found in cervical region (up to 76 %); lepto- and pachymeningeal lesions are found in 61 % of patients; intramedullary lesions — in 46 % [4]. The clinical signs of spinal cord sarcoidosis depend on the localization and may develop gradually, for example, with radiculomyelopathy. The damages to the spinal cord membranes cause, first of all, hyperalgesia, radicular syndrome, followed by anesthesia, and pareses. Localized or diffuse intramedullary process results in the signs of motor and sensory dysfunction, as well as symptoms of compression that require differential diagnosis with tumors. In vast majority of cases, no spinal lesions are found in spinal cord sarcoidosis. Some researchers specified an increased ratio of CD4/CD8 and interleukin-6 in the cerebrospinal fluid of patients with neurosarcoidosis compared with the other inflammatory diseases of central nervous system. The analysis of angiotensin-converting enzyme level in the cerebrospinal fluid in neurosarcoidosis patients does not allow interpreting the increase in this parameter as a reliable diagnostic criterion due to its low sensitivity and specificity [5]. The increased level of angiotensin-converting enzyme in peripheral blood, CD4/CD8 ratio in bronchoalveolar lavage and peripheral blood is of great importance for the diagnosis of generalized sarcoidosis [1].

Isolated spinal cord sarcoidosis requires histologically verified diagnosis, although the literature sources describe the single cases of direct intravital spinal cord biopsy [5]. According to the criteria developed by J.P. Zajicek (1999), when all other causes of damage to the central nervous system are excluded, neurosarcoidosis with its corresponding clinical signs and examination results can be described as possible (no histological confirmation), probable (with histological confirmation of systemic sarcoidosis), or definitive (with histologically confirmed damage to the nervous system due to sarcoidosis). Descriptions of definitive spinal cord sarcoidosis in the literature are rare. Most works include the analysis of probable sarcoidosis damage to spinal cord. Thus, in the study by N. Soni (2019), only 5 patients out of 18 had a definitive diagnosis, and 8 patients had a probable one. It should be mentioned that there is no description of the cases of discrepancy in diagnosis or medical error in probable spinal cord sarcoidosis [4].

The management of spinal cord sarcoidosis involves methylprednisolone for the first 3-5 days at a dose of 1 g (preferably parenterally); then the patient is switched to oral drug at an initial dose of 1 mg/kg of body weight with gradual dose tapering associated with clinical improvement within 12 months. Glucocorticosteroid therapy is effective in most patients, however, in cases of intolerance and ineffectiveness, methotrexate or azathioprine can be prescribed, as well as tumor necrosis factor-α inhibitors [3]. Complete regression of tomographic changes in the spinal cord associated with resolution of clinical symptoms of the disease occurs rarely. In most cases, several residual focal changes with sharp outlines persist. The possibility of spinal cord sarcoidosis relapse is significantly reduced if there is small amount of residual changes and long basic treatment course [5].

Sarcoidosis is a systemic granulomatous disease, therefore, the first symptoms of the disease in any organ may cause the patient to visit a relevant medical specialist, however, the complete examination and diagnosis justification with the choice of adequate therapeutic approach requires the cooperation of all involved specialists [1]. Focus on a narrow clinical issue can lead to a chronic course of the disease or to irreversible consequences of other initially asymptomatic manifestations. We would like to present the following case report.

Case report

Patient S., male, 31, presented with complains of general weakness, lack of sensitivity in lower limbs, no movements in lower limbs, no urge to urinate or defecate.

The patient is a coach for a children's football team. After a back injury in February 2021, he began to notice progressive muscle weakness, then — decreased sensitivity in lower limbs starting from the feet. In April, he was unable to move, with a complete loss of sensitivity in lower limbs, and pelvic organ dysfunction. According to the patient, no previous history of tuberculosis, HIV, hepatites, syphilis, oncological diseases. No past surgical interventions. The patient does not drink alcohol, denies smoking and substance use. About 20 years ago, the patient's mother had stage 2 pulmonary sarcoidosis and was treated with corticosteroids; there are residual changes in her lungs of the type of the areas of pulmonary fibrosis on both sides.

Physical examination of the patient revealed the general condition of moderate severity. The patient is oriented in space and time. Skin and visible mucous membranes are normally colored, no rash. Subcutaneous tissue is developed normally. BMI 18.2 kg/m^2. No peripheral edema. Peripheral lymph nodes are not palpable. Thyroid gland is not enlarged. No pathological changes found on palpation, percussion and auscultation. RR 16/min. BP 120/75 mm Hg. HR 76 bpm. Urination is spontaneous, painless, uncontrolled. Stool is regular, formed, uncontrolled.

Neurological status: fully conscious, cooperated, oriented. No meningeal signs. Palpebral fissures and pupils are symmetrical, direct and consensual pupillary reflexes are brisk. Eyeball movements are in full, painless. No nystagmus or diplopia. Sensitivity of face is remained. Tenderness of thoracic spinous processes during percussion. Tendon reflexes are brisk with the increased reflexogenous zones of lower limbs. Lower limb paraparesis up to 3 points. Heel-to-shin test is performed with significant dysmetria. Sensitive ataxia. The patient moves in a wheelchair.

Complete blood count: RBC 4.47×10^{12}/L (RV 4.28-5.78×10^{12}/L), hemoglobin 136 g/L (RV 130-170 g/L), WBC 12.2×10^9/L (RV 3.9-10.9×10^9/L), stab neutrophils 1 % (RV 1-5 %), segmented neutrophils 80 % (RV 40-70 %), lymphocytes 16 % (RV 20-45 %), monocytes 3 % (RV 3-8 %), ESR 3 mm/h (RV 2-16 mm/h).

Urinalysis: specific gravity 1015 g/L (RV 1015-1025 g/L), pH 6.5 (RV 5-9), protein 0 (RV 0-0.033 g/L), glucose 0 (RV 0-0.8 mmol/L), WBC 2-3 PFV (RV 0-6 PFV), RBC not found (RV 0-2 PFV), oxalates (RV none).

Biochemical assay: AST 23.5 U/L (RV 0-37 U/L), ALT 11.5 U/L (RV 0-45 U/L), creatinine 79.3 μmol/L (RV 62-115 μmol/L), bilirubin 16.2-13.3-2.9 μmol/L (RV 3.4-20.1 μmol/L, 2.4-12.2 μmol/L, 1-7.9 μmol/L), uric acid 3.86 μmol/L (PV 210-420 μmol/L), seromucoid 0.255 U (RV 0.13-0.2 U), C-reactive protein (CRP) 10.21 mg/L (RV 0-5 mg/L), glucose 4.49 mmol/L (RV 3.3-5.5 mmol/L), potassium 3.61 mmol/L (RV 3.5-5.5 mmol/L), sodium 140.8 mmol/L (RV 136-145 mmol/L), chlorine 100.8 mmol/L (RV 98-107 mmol/L), calcium 2.45 mmol/L (RV 2.25-3.0 mmol/L), ACE 14.6 ACE unit (RV 20-70 ACE unit).

TSH 0.548 μIU/L (RV 0.4-4 μIU/L), T3 4.82 pmol/L (RV 3.1-6.8 pmol/L), T4 0.54 ng/dL (RV 0.89-1.76 ng/dL).

APTT 34.5 s (RV 24.6-31.2 s), antithrombin III 100 % (RV 75-125 %), D-dimer 315.0 ng/mL (RV < 440 ng/mL),

INR 1.41 s (RV 0.85-1.15 s), Quick prothrombin 49.2 % (RV 70-130 %), thrombin time 17.2 s (RV 15.8-24.9 s), fibrinogen 4.0 g/L (RV 1.7-4.2 g/L).

Immunohematology. Antigen of the Kell (K) system not found (RV negative); antigens of the Rh system (C, c, E, e) C-E+c+e+ (RV negative); antibodies to RBC antigens not found.

Tumor markers. PSA: total 1.41 ng/mL (RV < 4 ng/mL), free (free PSA) 0.141 ng/mL (RV 10 % of total value); CEA 0.5 ng/mL (RV < 5 ng/mL).

Intradermal test with recombinant tuberculosis allergen — negative.

Ultrasound examination: signs of reactive lymphadenopathy of the neck on the right side. Abdominal organs without pathological findings. Small diffuse changes in thyroid parenchyma. No thrombosis of the veins of lower limbs.

Whole body 18-FDG PET/CT (Figure 1): in the right palatine tonsil, there is pathological metabolic activity, with SUV max 5.3, with no reliably detectable changes related to contrast enhancement; in a single right hypervascular supraclavicular lymph node, there is pathological metabolic activity, with SUV max 6.2, in size; there is pathological metabolic activity, with SUV max 24.0,

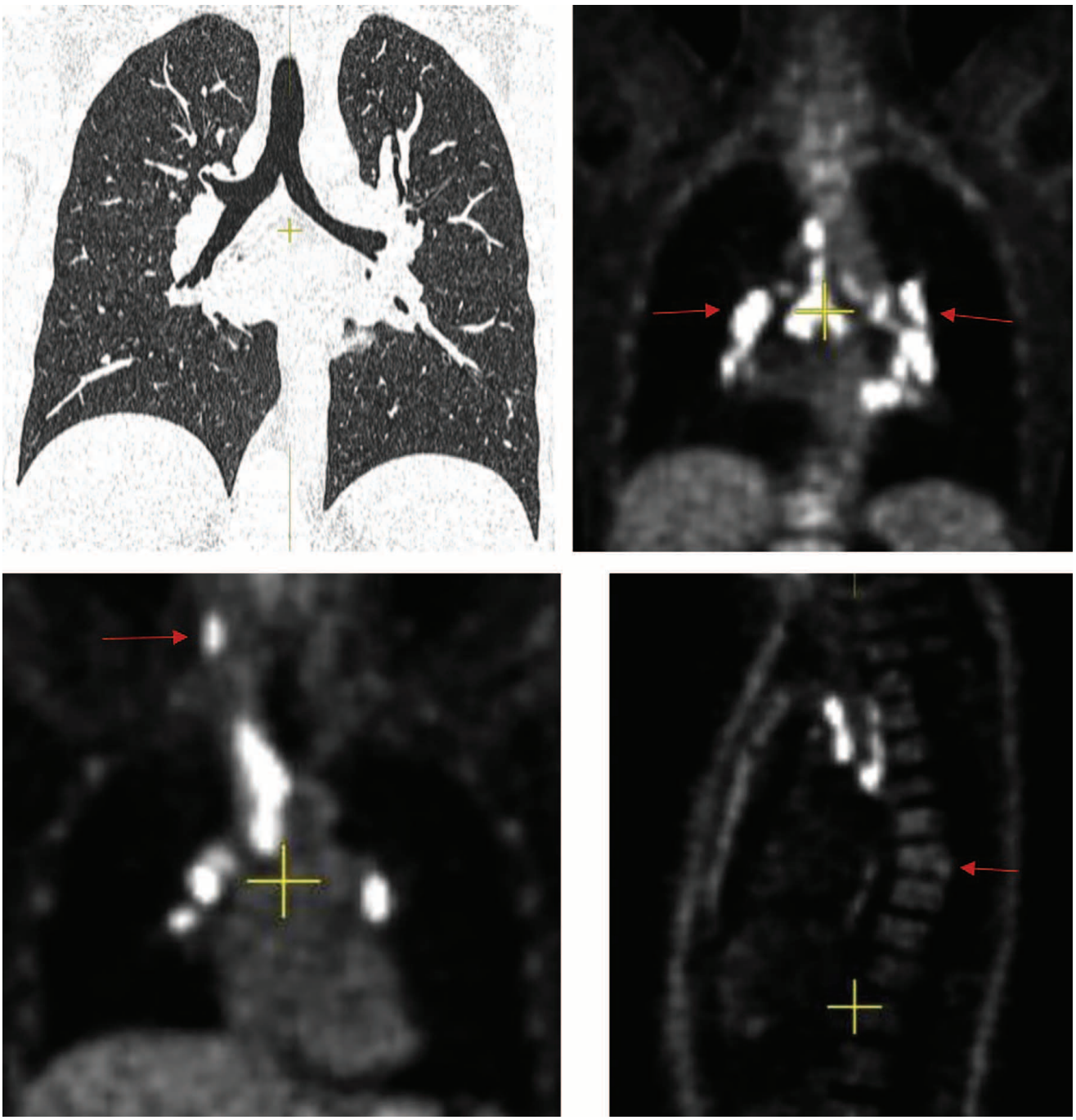

Figure 1. *PET/CT scan with 18-FDG of the chest of patient C. Pathological metabolic activity of SUV in enlarged intrathoracic lymph nodes, in enlarged right supraclavicular lymph node and in the spinal cord*

in paratracheal, bifurcation, bronchopulmonary, tracheobronchial lymph nodes that are enlarged from 15 to 22 mm; in spinal cord at Th8 endplate level, there is a focus of increased metabolic activity, with SUV max 3.1, 13x8 mm in size. Conclusion: findings suggestive of metabolically active neoplastic tissue in pathologically enlarged intrathoracic lymph nodes and right supraclavicular lymph node; the focus of increased metabolic activity in the spinal cord at Th8 endplate level requires additional examination.

MRI of thoracic spine with intravenous contrast enhancement (Fig. 2) in T1, T2, DWI, STIR, FAT SAT modes: degenerative changes and intervertebral disc height loss. The height of vertebral bodies is not changed. Bone endplates are sclerotic and visualized irregularly. Multiple Schmorl's nodes in Th7–Th12. Thoracic kyphosis is preserved. No listhesis of vertebral bodies was found.**1**Spondylosis changes are visible at the levels of Th7-Th12. Spinal canal is not narrowed. An intramedullary zone with altered MR signal was found, 18*5 mm in size (in sagittal plane). Spinal subarachnoid spaces are not deformed, not narrowed. After i/v contrast enhancement, its weak uptake by the pathological focus was observed. In DWI mode, the focus has a hyperintense signal. The roots of cauda equina are not deformed, not displaced. Conclusion: MRI signs of an intramedullary focal lesion at Th8-9 level.

ENMG of lower limbs: impaired function of impulse conduction along the left peroneal nerve and the left sural nerve. The lesion is of axonal-demyelinating type. There are signs of reinnervation along the sciatic nerve at the proximal level.

Considering the results obtained, an opinion was given about the need for the lymph node biopsy. However, during the examination of the patient, there was a significant regression of intrathoracic lymphadenopathy. An excisional biopsy of the right supraclavicular jugular lymph node was performed. Microscopic description: sections with the fragments of a lymph node with an inapparent structure pattern due to granulomatous inflammatory process. Cellular composition of the granuloma is represented by epithelioid cells and single multinucleated giant cells. The granuloma has quite sharp contours, with lymphocytic infiltration along the periphery. A developing reticular stroma of the granuloma with sharp contours was found; this fact (along with the absence of caseosis) allows speaking on a sarcoid granuloma. Conclusion: sarcoidosis of a lymph node.

After a multidisciplinary team meeting, the following diagnosis was established:

Generalized sarcoidosis: Sarcoidosis of intrathoracic lymph nodes and lungs, stage 1, active phase. Sarcoidosis of spinal cord, active phase. Sarcoidosis of peripheral lymph nodes, active phase. RF0. Central lower paraparesis. Dysfunction of pelvic organs. Lumbosacral spine osteochondrosis. Herniated discs L4-S1.

Considering the recommendations of all involved specialists, the following treatment was prescribed: dexamethasone 4 mg i/m 2 times a day; thioctacid 600 mg

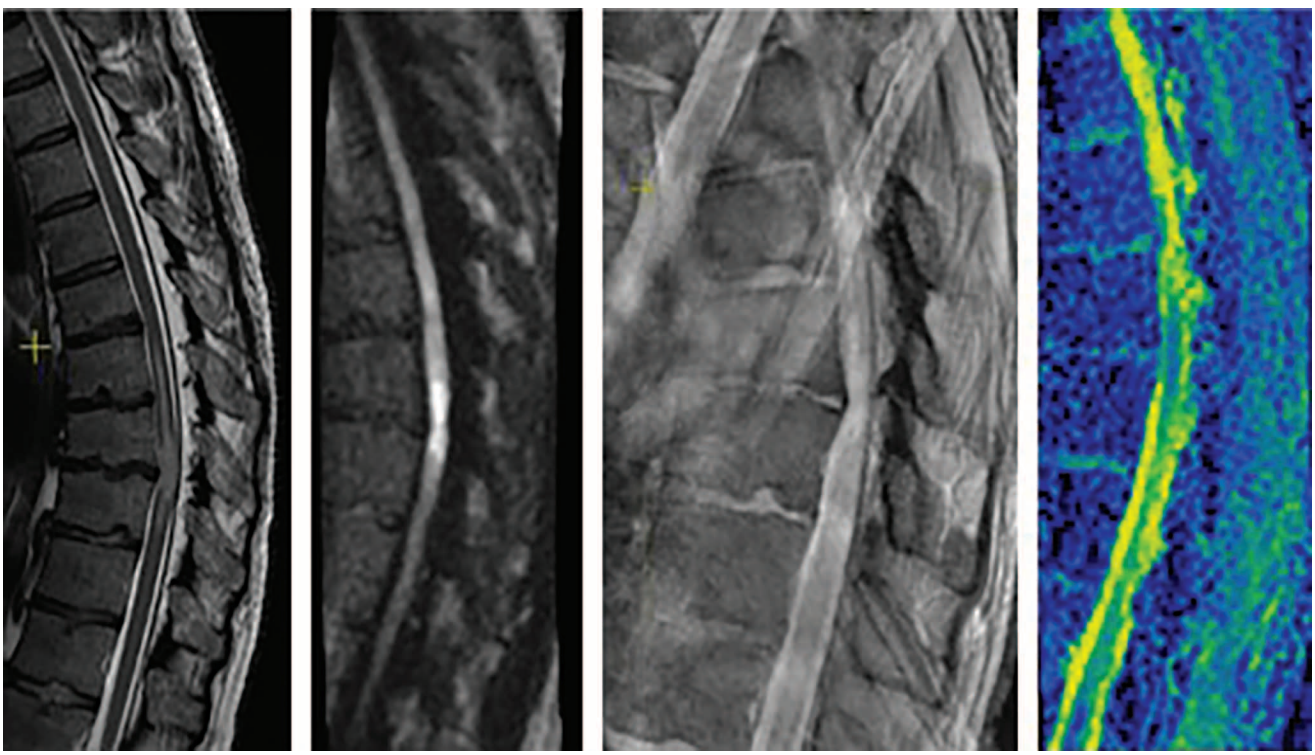

Figure 2. *MRT of the thoracic spine with intravenous contrast, performed in different modes. Signs of intramedullary focal formation in the spinal cord at the level of the Th8-9 vertebrae*

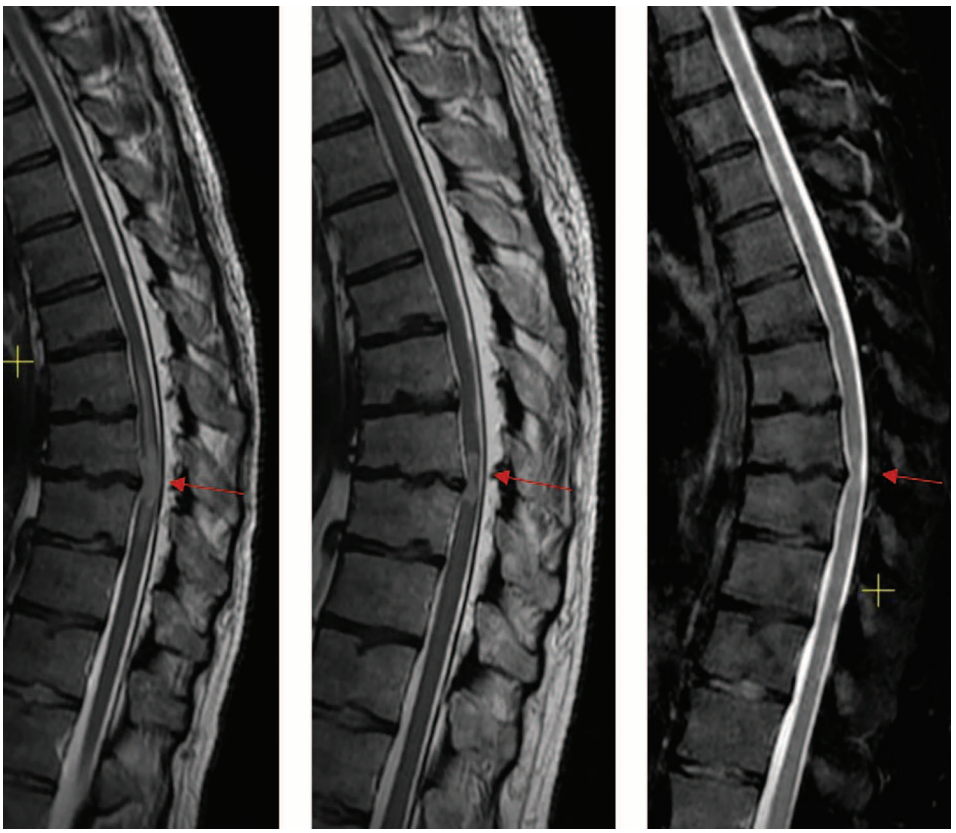

Figure 3. *Control MRT of the thoracic spine with intravenous contrast after 2 months and after 10 months of corticosteroid therapy. There is a decrease in the size of the intramedullary focus in the spinal cord at the level of the Th8-9 vertebrae*

orally once a day; actovegin 800 mg orally; omeprazole 20 mg orally; ipidacrine hydrochloride 20 mg 2 times a day.

14 days after the start of dexamethasone, positive changed in clinical presentation were observed: gradual restoration of sensitivity and movements in lower limbs. The patient was recommended to switch to oral prednisolone at a daily dose of 40 mg (30 mg in the morning and 10 mg in the afternoon). After 60 days of treatment with corticosteroids, complete restoration of sensitivity of lower extremities was achieved, as well as restoration of the pelvic organs function; the patient could move unassisted, first — using walking aids, later — without them. Control MRI of thoracic spine with intravenous contrast enhancement after 2 months and after 10 months of treatment with corticosteroids revealed decreased in size intramedullary lesion in spinal cord at Th8-9 level and the regression of perifocal edema (Fig. 3).

Adverse effects of treatment with corticosteroids included moon face, 5 kg weight gain, and acne.

Subsequently, a gradual dose tapering of prednisolone (by 5 mg per month) and two rehabilitation courses were performed (therapeutic exercises according to an individual method for lower paraparesis, 10 procedures; therapeutic swimming using a lift, 10 procedures; a course of mechanotherapy with Ortorent, Corvit, Detensor devices, 10 procedures; whirlpool pine bath for lower limbs, 10 procedures; programmable multi-channel electrical stimulation of the muscles of lower limbs, 5 procedures). The patient was fully recovered and returned to work. No relapse of sarcoidosis was observed.

Discussion

This case report confirms that sarcoidosis with a predominant involvement of intrathoracic lymph nodes can be asymptomatic, especially in young male patients, and tends to spontaneously regress. However, without timely diagnosis and adequate treatment, the extrathoracic signs of the process often tend to progress. One example is the lesions of central nervous system. Some researchers who dealt with the cases of isolated spinal cord sarcoidosis could observe early rapid regression of changes in intrathoracic lymph nodes at the time of the manifestation of cerebral symptoms. In such situations, the search for changes in peripheral lymph nodes is reasonable, as the process there tends to regress more slowly, as well as the analysis of ACE level — although in our case this parameter was within normal values — and blood CRP.

The clinical hypothesis about the possibility of a sarcoid reaction in connection with a tumor was confirmed by many reports on the possibility of sarcoidosis changes in lymph nodes combined with a local neoplastic process, or of their development that precedes a tumor. However, sarcoid reaction in tissues and lymph nodes is always localized and affects only one anatomical group of lymph nodes that is on the path of lymph outflow from the corresponding organ. As a rule, sarcoid reaction has no tendency to spontaneous regression. Our patient demonstrated generalized involvement of intrathoracic lymph nodes in combination with enlarged supraclavicular node, with a tendency to regression.

To confirm sarcoidosis, a tissue biopsy is reasonable in such cases, although the selection of an organ for biopsy in generalized process can be difficult. In our case, it was a peripheral lymph node. In many case reports on spinal cord sarcoidosis, researchers considered the results of a biopsy of a peripheral lymph node, so, the diagnosis was probable; while no cases of diagnostic errors were described. Direct biopsy of spinal cord is rare and is performed only in the absence of typical sarcoid lesions in any other organs. Histological results in our case report confirmed the presence of distinct non-caseous epithelioid cell granulomas with moderate lymphocytic infiltration. Although in some cases the histological conclusion is not always so unambiguous, there may be cases of obtaining a result of present lymphocytic infiltration with no typical granulomas in the early stages of the process. In such situations, a repeat biopsy is required.

However, is the following combination possible: sarcoidosis of intrathoracic and peripheral lymph nodes with a neoplastic lesion of spinal cord? We have found no such cases in the literature sources; sarcoidosis is usually combined with tumors of breast, thyroid gland, uterus and ovaries, or lungs. The patient was diagnosed with systemic inflammatory response in the presence of a normal level of tumor markers according to laboratory tests, as well as with maximum pathological metabolic activity of SUV 24.0 in enlarged intrathoracic and peripheral lymph nodes, and pathological metabolic activity of SUV 3.1 in spinal cord at Th8 endplate level. Considering the histological results of peripheral lymph node biopsy, the diagnosis of generalized sarcoidosis with a predominant lesion of spinal cord became probable, and the approach with a course of intensive corticosteroid therapy with a subsequent assessment of changes seemed to be adequate.

In all cases of spinal cord sarcoidosis that are described in the literature sources, the effectiveness of parenteral methylprednisolone or prednisolone during the first month was mentioned, followed by oral agents for a period of at least 12 months. Our case is interesting due to the fact that we used dexamethasone injections for 14 days, and then prednisolone in tablets at an initial dose of 40 mg; such treatment resulted in the rapid regression of clinical and radiological signs with no pronounced adverse effects.

Present-day treatment programs for sarcoidosis of all localizations include rehabilitation procedures. In the above clinical case, medical rehabilitation courses including mechanotherapy, balneotherapy, electrical stimulation, and exercise therapy led to a rapid recovery of the patient's ability to work.

The case of our patient is also interesting due to his history with the indication of past sarcoidosis in his mother. There are reports on cases of familial sarcoidosis in the literature sources. In our practice, we have observed the cases of sarcoidosis in siblings, a mother and a son, a mother and a daughter. The disease process commenced in relatives at different times and was characterized by no uniformity of clinical signs or course. In the described case, the patient's mother had pulmonary sarcoidosis with pronounced fibrotic changes at the age of 38. Her son fell ill under the age of 35; the process was characterized mainly by extrathoracic manifestations.

Conclusion

Generalized sarcoidosis may initially be represented by stage 1 or 2 pulmonary sarcoidosis, however, later on, it is often accompanied by the clinical signs of extrathoracic disease localizations. Systemic granulomatous lesions may be localized in spinal cord. The procedure for confirming the diagnosis of spinal cord sarcoidosis is demonstrated in Figure 4.

Regardless of the main symptoms, the correct diagnosis and adequate treatment require a comprehensive examination, with consideration of the recommendations of involved specialists and biopsy results. Corticosteroids are effective in the management of spinal cord sarcoidosis; the treatment takes many months and the initial use of parenteral drugs.

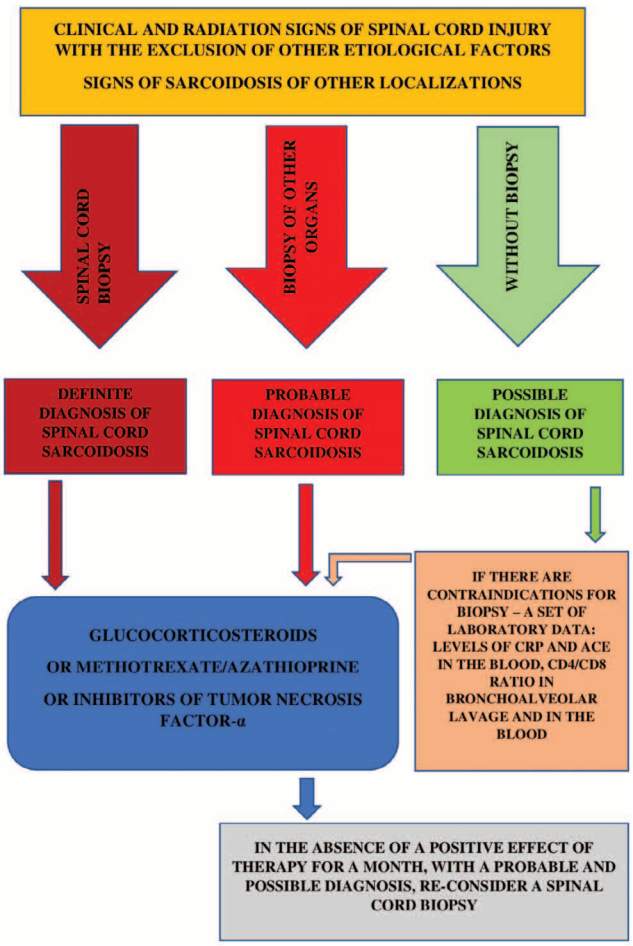

Figure 4. *Algorithm for confirming the diagnosis of spinal cord sarcoidosis*

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Черников А.Ю. (ORCID ID: https://orcid.org/0000-0002-2045-9607): разработка концепции и дизайна, обоснование и написание рукописи

Кабанова С.А.: сбор, анализ и интерпретация данных

Дьяков А.В.: проверка критически важного интеллектуального содержания и окончательное утверждение для публикации рукописи

Author Contribution:

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

Chernikov A.Y. (ORCID ID: https://orcid.org/0000-0002-2045-9607): contribution to the development of the concept and design, justification and writing of the manuscript

Kabanova S.A.: contribution to the collection, analysis and interpretation of data

Dyakov A.V.: contribution to the verification of critical intellectual content and final approval for publication of the manuscript

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