MILD COGNITIVE DISORDER AS CLINICAL MANIFESTATION OF PITUITARY STALK NEUROSARCOIDOSIS: CASE REPORT

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SUMMARY – A case is presented of a 59-year-old male patient with a 5-year history of sarcoidosis. In the last half a year, deterioration of his intellectual abilities was noticed. Psychological testing detected a mild cognitive disorder. Laboratory diagnostics found a decreased level of testosterone and magnetic resonance imaging showed pituitary stalk neurosarcoidosis without any other pathomorphological substrate of cognitive impairment. This case indicates that neurosarcoidosis should be considered as a possible cause of mild cognitive disorder and, consequently, included in the International Classification of Mental and Behavioural Disorders.

Key words: Neurosarcoidosis; Pituitary gland; Cognition disorders

Introduction

Sarcoidosis is a multisystemic granulomatous disease of unknown etiology. In 90% of patients, the disease affects the lungs, most commonly as bilateral lymphadenopathy. Sarcoidosis can also involve other organs such as lymph nodes, eyes, skin, heart, muscles, liver and brain¹⁻¹⁴.

Neurosarcoidosis (central and peripheral) can be found in 5%-15% of patients with sarcoidosis, whereas central neurosarcoidosis occurs in about 5% of patients. Diagnosis of neurosarcoidosis is based on the clinical picture of sarcoidosis in other parts of the body (lung, liver, etc.), magnetic resonance imaging (MRI) of the brain, finding of angiotensin converting enzyme (ACE) in serum and liquor, and x-ray, but definitive diagnosis can only be made after biopsyl⁴⁻²⁰.

Previous research has shown that MRI helps in differentiating sarcoidosis from other brain diseases

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such as vasculitis, neoplasms (glioma, meningeoma, metastases) and other granulomatous diseases. It is important to distinguish neurosarcoidosis from non-neoplastic diseases of the central nervous system, such as multiple sclerosis, stroke, pyogenic abscess, toxoplasmosis, tuberculosis, cysticercosis, fungal infections, syphilis, Behcet disease, radiation necrosis, venous thrombosis, but also manifestations of systemic lupus erythematosus, Henoch-Schönlein purpura, polyarteritis nodosa, Wegener's granulomatosis, etc.²⁰⁻²³. In this case report, we describe a patient with pituitary stalk neurosarcoidosis, which presented itself as a mild cognitive disorder.

Case Report

A 59-year-old man diagnosed with lung sarcoidosis five years before was treated with corticosteroids during the first year and a half after the diagnosis, but in the last three and a half years he received no therapy. During the last 6 months, his family members and friends noticed that his thoughts were slowed down and that sometimes he had difficulties in remembering things. Therefore, he was referred for neurologi-

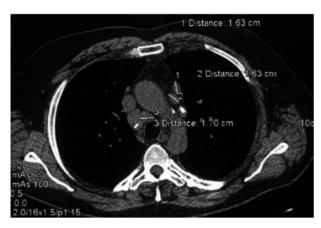


Fig. 1. Axial chest computed tomography without contrast media administration showed enlarged hilar and mediastinal lymph nodes. Peribronchovascular and interlobar septa are thick with nodal lesions connected to the pleura, corresponding to the lymphoid type of distribution in parenchymal sarcoidosis. There are signs of fibrosis within the lower lung lobes (not seen in these images).

cal, neuroradiological and hormonal examination, as well as psychological testing. On clinical neurological examination, no motor deficit was found. Given the existing underlying disease, further diagnostic procedures were undertaken. Physical examination detected no enlarged lymph nodes, ACE in serum and liquor was increased; serum ACE was 127 IU/L (reference range: 8-51 IU/L) and ACE in the liquor finding was positive; computerized tomography (CT) of the chest showed lesions characteristic of sarcoidosis: bilateral

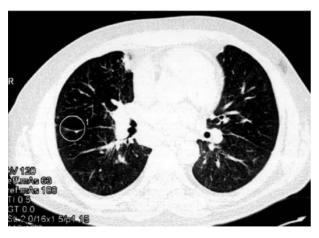


Fig. 2. Lung computerized tomography shows bilateral lymphadenopathy.

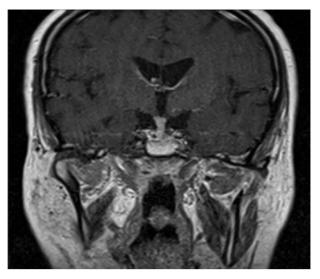


Fig. 3. Brain magnetic resonance image: coronal post contrast T1 weighted image demonstrating abnormally thickened pituitary stalk.

enlarged hilar lymph nodes, enlarged hilar and mediastinal lymph nodes with peribronchovascular thickening and thickening of interlobular septa with nodal lesions connected to the pleura, consistent with the

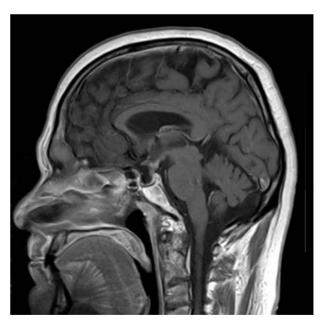


Fig. 4. Brain magnetic resonance image: on the sagittal post contrast T1 weighted image, abnormally thickened pituitary stalk is confirmed, with very tiny hypophysis pushed down and posteriorly by a mild suprasellar cerebrospinal fluid herniation.

lymphoid type of distribution in parenchymal sarcoidosis (Figs. 1 and 2).

Signs of initial fibrosis were found in the lower lung lobes (not shown in the images). CT of the neck was normal and showed no enlarged lymph nodes. Brain MRI revealed pituitary stalk neurosarcoidosis (Figs. 3 and 4).

Hormonal examination showed normal levels of T3, T4, TSH and growth hormone, whereas serum testosterone concentration was lowered (Table 1). Psychological examination detected psychomotor deterioration, memory losses and learning difficulties. Psychological testing showed a decrease in the efficacy in the tests of learning and remembering, as well as some cognitive deterioration. Visual-motor coordination and constructional functions were normal. Scores on the tests for solving complex visual-spatial tasks were within the acceptable limits, but with certain deviations indicative of cerebral dysfunction. Most prominent failures were detected in the tests of acquiring new verbal content, while memory functions were moderately affected. Verbal fluency tested by naming familiar objects was also subnormal. The speed of visual content processing was mildly to moderately decreased. Non-verbal memory was also somewhat below the average. Flexibility of thinking was reduced (Table 2). The above findings were indicative of a mild cognitive disorder. Six months after the therapeutic treatment, the findings partly normalized, and cognitive functions showed somewhat lesser deviations. Serum ACE was mildly raised and serum testosterone was mildly lowered. Neuropsychological tests showed signs of cognitive function improvements consistent with mild cognitive disorder.

Table 1. Hormone levels in the male patient with neurosarcoidosis

Serum hormone level	Serum hormone reference range		
T3 2.1 mmol/L	1.3-3.6 mmol/L		
T4 98 mmol/L	58-161 mmol/L		
TSH 3.2 mIU/L	0.3-3.6 mIU/L		
Growth hormone 4.8 mIU/L	<10 mIU/L		
Testosterone 2.8 nmol/L	4.6-21.7 nmol/L		

Table 2. Neuropsychological tests in the male patient with neurosarcoidosis

Neuropsychological test Test result			
Memory losses			
Learning difficulties			
Test of learning and remembering	Decreased		
Visual-motor coordination	Normal		
Constructional functions	Normal		
Complex visual-spatial tasks	Subnormal		
New verbal content	Prominent failures		
Memory functions	Moderately affected		
Verbal fluency tested	Subnormal		
Visual content processing	Decreased		
Non-verbal memory	Somewhat below the average		
Flexibility of thinking	Reduced		

Discussion

A number of clinical manifestations of neurosarcoidosis have been described14-22. Primarily, one should differentiate between central and peripheral neurosarcoidosis. Peripheral neurosarcoidosis presents itself as polyneuropathy, myopathy, multiple mononeuritis, and also affects peripheral and cranial nerve roots. Central neurosarcoidosis involves multiple or solitary supra- and infratentorial brain lesions, mostly in the hypothalamus and/or pituitary gland, pituitary stalk, basal ganglia, multiple lacunar infarctions, fourth ventricles, inflammatory lesions in the brain and spinal cord, dural and leptomeningeal enhancement, intracranial hemorrhage, pituitary tuberculoma, amygdale, anterior hippocampus, mesial temporal neocortex, medullary involvement, deep white matter, cerebral aqueduct with hydrocephalus, pons, spinal mass lesion, temporodorsal in the hemisphere and cranial nerves^{17,19,24-54}. 'Aqueductal stenosis' is commonly associated with psychosis and delirium in patients with neurosarcoidosis^{19,55}.

Pituitary gland involvement caused by granulomatous changes in the hypothalamo-hypophyseal system results in hormonal disbalance, which is responsible for diabetes insipidus as well as for amenorrheagalactorrhea with hypogonadotropic hypogonadism^{16,26,34,36,38,42,44,49,50}.

Brain MRI reveals changes in the pituitary gland and especially in the pituitary stalk that is often thickened, which is quite characteristic of neurosarcoidosis. However, thickening of the pituitary stalk can occur in other diseases such as Langerhans cell histiocytosis and pituitary hypophysitis^{26,34,44}.

This case report describes a man with visible changes in pituitary stalk, as detected by MRI, accompanied by the lowered level of testosterone and impairment of cognitive function, i.e. mild cognitive disorder consisting of concentration difficulties and some degree of memory and psychomotor deterioration.

The lowered level of testosterone can be explained by the pituitary stalk damage.

The new, 10th, revision of the International Classification of Mental and Behavioural Disorders (ICD-10) contains mild cognitive disorders⁵⁶. Mild cognitive disorder is diagnosed on the basis of neuroradiological and laboratory findings, and psychological testing, after all other possible causes have been ruled out.

Neuropsychological impairments (psychomotor slowing, weakened memory, etc.) in mild cognitive disorder are not confirmed by morphological substrates in the brain detected by the neuroradiological diagnostics (CT and MRI). Definitive diagnosis of neurosarcoidosis in our patient was made on the basis of lung x-ray, ACE in serum and liquor, neuroradiological examination and psychological testing which, together with clinical symptoms, indicated mild cognitive disorder. In our patient, neurosarcoidosis of the pituitary stalk could explain the lowered level of testosterone, but could not be associated with cognitive impairments. In the case presented, we found no morphological substrate that could explain the mild cognitive disorder. Prüter et al. have previously described a mild cognitive disorder with no clear morphological substrate found on MRI in a woman with sarcoidosis, which they explained by meningitis caused by neurosarcoidosis⁵⁷. In our case, there were no signs of meningitis or any other inflammatory process in the

central nervous system during the course of illness. Prüter *et al.* state that neurosarcoidosis in most cases occurs as a subacute form of sarcoidosis⁵⁷. Obviously, in our case neurosarcoidosis developed in a subacute phase, when bilateral hilar lymphadenopathy was in regression.

In the male patient, pituitary stalk neurosarcoidosis resulted in a lowered level of testosterone, whereas hyperprolactinemia and amenorrhea are described in female patients^{26,44,58,59}. Similar to the previously described case⁵⁷, mild cognitive disorder found in our patient did not have a clear morphological substrate. Studies have shown that in most of the patients with mild cognitive disorder caused by other etiological factors there is no clear pathomorphological substrate detectable by standard neuroradiological diagnostics. An exception is MRI 3T, which allows recognizing some differences in comparison to control group^{60,61}.

In conclusion, pituitary stalk neurosarcoidosis in men can present itself with a low level of testosterone, but also as a mild cognitive disorder. Since this is the second case of mild cognitive disorder reported in patients with neurosarcoidosis, we suggest that neurosarcoidosis should be considered as a possible cause of mild cognitive disorder and, consequently, included in the International Classification of Mental and Behavioural Disorders.

References

- NUNES H, BRILLET PY, VALEYRE D, BRAUNER MW, WELLS AU. Imaging in sarcoidosis. Semin Respir Crit Care Med 2007;8:102-20.
- 2. RICCI A, MRIOTTA S, SALTINI C, FALASCA C, GIOVAGNOLI MR, MANNINO F, GRATUABI P, SCIACCHITANO S, AMENTA F. Neurotrophin system activation in bronchoalveolar lavage fluid immune cells in pulmonary sarcoidosis. Sarcoidosis Vasc Diffuse Lung Dis 2005;22:186-94.
- GUPTA S, FAUGHNAN ME, PROD'HOMME GJ, HWANG DM, MUNOZ DG, KOPPLIN P. Sarcoidosis complicated by cirrhosis and hepatopulmonary syndrome. Can Respir J 2008;15:124-6.
- YASUTAKE H, SEINO Y, KASHIWAGI M, HONMA H, MATSUZAKI T, TAKANO T. Detection of cardiac sarcoidosis using cardiac markers and myocardial integrated backscatter. Int J Cardiol 2005;102:259-68.
- DATE T, SHINOZAKI T, YAMAKAWA M, TANIGU-CHI I, SUDA A, HARA H, YAMANE T, KOMUKAI K, SUGIMOTO K, MOCHIZUKI S. Elevated plasma brain

- natriuretic peptide level in cardiac sarcoidosis patients with preserved ejection fraction. Cardiology 2007;207:277-80.
- 6. TERISTEIN A. Neuromuscular sarcoidosis. Semin Respir Crit Care Med 2002;23:505-12.
- HANDA T, NAGAI S, ITO I, SHIGEMATSU M, HAMADA K, KITAICI M, OHTA K, IZUMI T, MISHI-MA M. Multiple bone fractures found in a young sarcoidosis patient with long stable disease. Intern Med 2005;44:1269-75.
- KANEMITSU S, MIYAKE Y, OKABE M. Surgical removal of a left ventricular thrombus associated with cardiac sarcoidosis. Interact Cardiovasc Thorac Surg 2008;7:333-5.
- 9. GOTTSCHALK A, DANZ B, VÖLK M. Infratentorial progressive multifocal leukoencephalopathy in a patient with pulmonary sarcoidosis. Rofo 2005;177:1583-5.
- VÖLKER HU, KRAFT K, ARNOLD E, STEINHOFF S, KOLIOS G, SOMMER S. Progressive multifocal leukoencephalopathy developing in advanced pulmonary sarcoidosis. Clin Neurol Neurosurg 2007;109:624-30.
- BYARD RW, MANTON N, TSOKOS M. Sarcoidosis and mechanisms of unexpected death. J Forensic Sci 2008;53:460-4.
- YAMAGUCHI S, KURODA S, KOBAYASHI H, MAR-UICHI K, KUBOTA K, ITO T, IWASAKI Y. CNS sarcoidosis presenting with intracerebral hemorrhage: a case report. No Shinkei Geka 2006;34:839-42.
- 13. SCHELHORN J, SMESNY U, FITZEK C, BRODHUN M, WITTE OW, TERBORG C. Differential diagnosis of solitary neurosarcoidosis. Nervenarzt 2005;76:984-7.
- TSAO CY, LO WD, RUSIN JA, HENWOOD MJ, DOUE DR. Isolated neurosarcoidosis presenting as headache and multiple brain and spinal cord lesion mimicking central nervous system metastases. Brain Dev 2007;29:514-8.
- 15. SPONSLER JL, WERZ MA, MACIUNAS R, COHEN M. Neurosarcoidosis presenting with simple partial seizures and solitary enhancing mass: case reports and review of the literature. Epilepsy Behav 2005;6:623-30.
- 16. TAMAGNO G, MURIALDO G. Amenorrhea-galactorrhea syndrome as an uncommon manifestation of isolated neurosarcoidosis. Ann Ital Med Int 2001;16:260-6.
- 17. MISCUSI M, POLLI FM, MISORI P, DELFINI R. Ghost lesions in patient with cerebral-isolated neurosarcoidosis. A case report. J Neurosurg Sci 2006;50:17-20.
- 18. IIZUKA T, SAKAI F. Neurosarcoidosis. Nippon Rinsho 2002;60:1785-93.
- MARANGONI S, ARAGENTIERO V, TAVOLATO B. Neurosarcoidosis. Clinical description of 7 cases with a proposal for a new diagnostic strategy. J Neurol 2006;153:488-95.
- 20. OMURO AM, LEITE CC, MOKHTARI K, DELATTRE JY. Pitfalls in the diagnosis of brain tumours. Lancet Neurol 2006;5:937-48.

- HODGE MH, WILLIAMS RL, FUKUI MB. Neurosarcoidosis presenting as acute infarction on diffusion-weighted MR imaging: summary of radiologic findings. AJNR Am J Neuroradiol 2007;28:84-6.
- 22. VANNEMREDDY PS, NANDA A, REDDY PK, GONZALEZ E. Primary cerebral sarcoid granuloma: the importance of definitive diagnosis in the high-risk patient population. Clin Neurol Neurosurg 2002;104:289-92.
- DUZOVA A, BAKKALOGLU A. Central nervous system involvement in pediatric rheumatic disease: current concepts in treatment. Curr Pharm Des 2008;14:1295-301.
- 24. MOORE FG, ANDERMANN F, RICHARDSON J, TAMPIERI D, GIACCONE R. The role of MRI and nerve biopsy in the diagnosis of neurosarcoidosis. Can J Neurol Sci 2001;28:349-53.
- 25. KODAMA M, UMEGAKI H, MOGI N, IGUCHI A, TAKEDA. An elderly case with sarcoidosis whose symptom of dementia was effectively treated by steroid. Nippon Ronen Igakkai Zasshi 2002;39:648-53.
- MUIRALDO G, TAMAGNO G. Endocrine aspects of neurosarcoidosis. J Endocrinol Invest 2002;25:650-2.
- 27. FERRIBY D, STOJKOVIC T, De SEZE J, HURTEVENT JF, VERMERSCH P. Chronic polyradiculoneuritis disclosing sarcoidosis. Rev Neurol (Paris) 2002;158:357-60.
- 28. SAID G, LACROIX C, PLATÉ-BORDENEUVE V, Le PAGE L, PICO F, PRESLES O, SENANT J, REMY P, PONDEPIERRE P, MALLECOURT J. Nerve granulomas and vasculitis in sarcoid peripheral neuropathy: a clinicopathological study of 11 patients. Brain 2002;125(Pt 2):264-75.
- 29. KIDD D, BEYNON HL. The neurological complications of systemic sarcoidosis. Sarcoidosis Vasc Diffuse Lung Dis 2003;20:85-94.
- 30. KORT L, BONCOEUR MP, DELAGE-CORRE M, MOUFID A, DENES E, COURATIER P. Isolated neurosarcoidosis without systemic signs. Rev Neurol (Paris) 2003;159:455-7.
- MAHADEWA TG, NAKAGAWA H, WATABE T, IN-OUE T. Intramedullary neurosarcoidosis in the medulla oblongata: a case report. Surg Neurol 2004;61:283-7.
- 32. NAKAGAKI H, FURUYA J, NAGATA T, KOTORII S, NAGANO S, HIGASHINO T, YOSHIKAI S, NAKANISHI K, YAMADA T. An elder case of neurosarcoidosis associated with brain infarction. Rinsho Shinkeigaku 2004;44:81-5.
- 33. HAMADA H, HAYASHI N, KURIMOTO M, UME-MURA K, HIRASHIMA Y, ENDO S. Isolated third and fourth ventricles associated with neurosarcoidosis successfully treated by neuroendoscopy case report. Neurol Med Chir (Tokyo) 2004;44:435-7.
- 34. TABUENA RP, NAGAI S, HANDA T, SHIGEMAT-SU M, HAMADA K, ITO I, IZUMI T, MISHIMA M, SHARMA OP. Diabetes insipidus from neurosarcoidosis:

- long-term follow-up for more than eight years. Intern Med 2004:43:960-6.
- SPENCER TS, CAMPELLONE JV, MALDONADO I, HUANG N, USMANI Q, REGINATO AJ. Clinical and magnetic resonance imaging manifestations of neurosarcoidosis. Semin Arthritis Rheum 2005;34:649-61.
- 36. TRABELSI L, MAJDOUB-REKIK N, BOUAZIZ H, MNIF-FEKI M, HAMMEMI B, MAALOUL I, BEN JMAA M, ABID M. Pituitary tuberculosis: a case report. Ann Endocrinol (Paris) 2005;66:340-6.
- NISHIE M, MORI F, SUZUKI C, OGAWA M, KURA-HASHI K, KAIMORI M, WAKABAYASHI K. Disseminated intraparenchymal microgranulomas in the brainstem in central nervous system sarcoidosis. Neuropathology 2005;25:361-4.
- 38. KAROUACHE A, MOUNACH J, AZIZ N, BOURAZA A, SATTE A, OUHABI H, RAFIQ R, BOUTALEB N, MOSSADAQ R. A report of 9 cases of neurosarcoidosis. Rev Neurol (Paris) 2005;161:1091-101.
- 39. SPONSLER JL, WERZ MA, MACIUNAS R, COHEN M. Neurosarcoidosis presenting with simple partial seizures and solitary enhancing mass: case reports and review of the literature. Epilepsy Behav 2005;6:623-30.
- 40. MAEDA K, KITA Y, UEHARA S, YAMASAKI O, RI-KIMARU M, SAJI N, TABUTI M, FURUMOTO M. A case of isolated CNS sarcoidosis with diffuse confluent high intensity lesions at bilateral deep white matter. No To Shinkei 2006;58:605-10.
- 41. BRISMAN JL, HINDUJA A, McKINNEY JS, GER-HARDSTEIN B. Successful emergent angioplasty of neurosarcoid vasculitis presenting with strokes. Surg Neurol 2006;66:402-4.
- 42. SCHLIENGER JL, DELEMER B, VINZIO S. Impact of systemic disease on the pituitary gland. Ann Endocrionol (Paris) 2006;67:316-24.
- RÓZSA A, SZTANKANINECZ Y, GÁCS G, MAGYAR T. Uncommon manifestation of central nervous system sarcoidosis. Ideggyogy Sz 2007;60:46-50.
- 44. BIHAN H, CHRISTOZOVA V, DUMAS JL, JOMAA R, VALEYRE D, TAZI A, REACH G, KRIVITZKY A, COHEN R. Sarcoidosis: clinical, hormonal, and magnetic resonance imaging (MRI) manifestations of hypothalamic-pituitary disease in 9 patients and review of the literature. Medicine (Baltimore) 2007;86:259-68.
- PERDIGÃO S, ARANTES M, PINHEIRO L, COSTA M. Atypical presentation of intramedullary sarcoidosis: report of two cases. Rev Neurol 2007;45:406-8.
- 46. TUMIALÁN LM, GUPTA M, HUNTER S, TUMIA-LÁN L. A 55-year-old man with liver failure, delirium and seizures. Brain Pathol 2007;17:472-3.

- WESTHOUT FD, LINSKEY ME. Obstructive hydrocephalus and progressive psychosis: rare presentations of neurosarcoidosis. Surg Neurol 2008;69:288-92.
- WALID MS, AJJAN M, GRIGORIAN AN. Neurosarcoidosis the great mimicker. J Natl Med Assoc 2008;100:859-61.
- 49. SUZUKI M, MARSUI O, UEDA F, MATSUSHITA T, FUJINAGA Y, KOBAYASHI K, HORICHI Y, HAYASHI Y, TACHIBANA O, YAMASHITA J. Dynamic MR imaging for diagnosis of lesions adjacent to pituitary gland. Eur J Radiol 2005;53:159-67.
- 50. TAKANO K. Sarcoidosis of the hypothalamus and pituitary. Intern Med 2004;43:894-5.
- 51. BRUNS F, PRUEMER B, HAVERKAMP U, FISCH-EDICK AR. Neurosarcoidosis as an unusual indication for radiotherapy. Br J Radiol 2004;77:777-9.
- 52. FELS C, RIEGEL A, JAVAHERIPOUR-OTTO K, OBENAUER S. Neurosarcoidosis: findings in MRI. Clin Imaging 2004;28:166-9.
- 53. HESSELMANN V, WEDEKIND C, TERSEGGE K, SCHULTE O, VOGES J, KRUG B, LACKNER K. An isolated fourth ventricle in neurosarcoidosis: MRI findings. Eur Radiol 2002;12(Suppl 3):S1-3.
- KETONEN L, OKSANEN V, KUULIALA I. Preliminary experience of magnetic resonance imaging in neurosarcoidosis. Neuroradiology 1987;29:127-9.
- FRIEDMAN SH, GOULD DJ. Neurosarcoidosis presenting as psychosis and dementia: a case report. Int J Psychiatry Med 2002;32:401-3.
- World Health Organisation. The ICD-10 Classification of Mental and Behavioural Disorders. Clinical descriptions and diagnostic guidelines. Geneva: World Health Organization, 1992
- 57. PRÜTER C, KUNERT HJ, HOFF P. ICD-10 mild cognitive disorder following meningitis due to neurosarcoidosis. Psychopathology 2001;34:326-7.
- 58. OKSANEN V. Neurosarcoidosis. Sarcoidosis 1994;11:76-9.
- SABAAWI M, GUTIERREZ-NINEZ J, FRAGALA MR. Neurosarcoidosis presenting as schizophreniform disorder. Int J Psychiatry Med 1992;22:269-74.
- EGRHÁZI A, GLAUB T, BALLA P, BERECZ T, DE-GRELL I. P300 in mild cognitive impairment and in dementia. Psychiatr Hung 2008;23:349-57.
- 61. BOKDE AL, LOPERZ-BAVO P, BORN C, DONG W, MEINDL T, LEINSINGER G, TEIPEL SJ, FALTRACO F, REISER M, MÖLLER HJ, AMPEL H. Functional abnormalities of the visual processing system in subjects with mild cognitive impairment: an MRI study. Psychiatry Res 2008;163:248-59.

Sažetak

BLAŽI KOGNITIVNI POREMEĆAJ KAO KLINIČKA MANIFESTACIJA NEUROSARKOIDOZE LIJEVKA HIPOFIZE: PRIKAZ SLUČAJA

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Prikazuje se slučaj 59-godišnjeg bolesnika s 5-godišnjom poviješću bolesti sarkoidoze. U posljednjih pola godine primjetno je bilo slabljenje njegovih intelektualnih sposobnosti. Psihološkim testiranjem otkriven je blaži spoznajni poremećaj. Laboratorijskom dijagnostikom utvrđena je snižena razina testosterona, dok je magnetska rezonancija pokazala neurosarkoidozu lijevka hipofize bez ikakvog drugog patomorfološkog supstrata kognitivnog poremećaja. Ovaj slučaj pokazuje da neurosarkoidozu treba uzeti u obzir kao mogući uzrok blažeg spoznajnog poremećaja, pa bi je trebalo uvrstiti u Međunarodnu klasifikaciju psihičkih bolesti i bolesti ponašanja.

Ključne riječi: Neurosarkoidoza; Hipofiza; Kognitivni poremećaji