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TOPICAL REVIEW

Clinical manifestations of neurosarcoidosis

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Abstract: Sarcoidosis is a chronic disease of unknown aetiology. Neurosarcoidosis is registered in 5 % of patients with sarcoidosis. Clinical manifestations of sarcoidosis are numerous and diverse. Manifestation of Neurosarcoidosis includes partial- and grand-mal seizures, low-grade fever, headache, increased intracranial pressure, visual disturbances, diabetes insipidus, amenorrhea- galacterorrhea syndrome and pituitary failure, hypogonadotropic hypogonadism, hyperprolactinemia, unilateral and bilateral facial palsy, infiltration of meninges (aseptic meningitis) and nerve roots, leptominingitis, pachymeningitis with cranial neuropathies, pseudotumor, mild cognitive disorder, psychosis, delirium, dementia, disorientation, amnesia, progressive visual deterioration and proptosis, axonal polyneuropathies, mononeuropathies, chronic polyradiculoneuritis, peripheral neuropathy, cranial nerve abnormalities, radiculopathies, peripheral neuropathy, mononeuritis multiplex, progressive numbness and deep sensation disturbance in bilateral lower extremities, hemiplegia, hyperreflexia with pathological reflexes and hypesthesia, upward gaze palsy, spinal cord compression, dysarthria, dysphagia, weakness, episodes of blurred vision, diplopia, intracerebral hemorrhage, neuro-ophthalmic manifestations, intranuclear ophthalmoplegia, dysorientation, vasculitis presenting with strokes, intracranial hypothalamic lesion, paresthesis, hemiparesis, myelopathy in the cervico-thoracic region, lumbar pain, sensory level and inability of lateral gaze *(Tab. 2, Ref. 60).* Full Text (Free, PDF) *www.bmj.sk.*
Key words: sarcoidosis, neurosarcoidosis, hydrocephalus and intracranial hemorrhage.

Sarcoidosis is an inflammatory chronic disease of unknown aetiology characterised by non-caseating epithelioid granulomas, presence of T lymphocytes, mononuclear phagocytes, and non-caseating epithelioid granulomas in the tissues (1, 2). The organs most often involved are the lungs, but any other organ may be affected as well, with or without concomitant lung involvement. Yet, the lungs are involved in about 90 % of patients. The typical appearance is bilateral hilar lymphadenopathy with or without lung parenchymal infiltrates, but a wide range of chest radiographic patterns may be observed. In cases of liver involvement, cirrhosis and portal hypertension develop, occurring in fewer than 1 % of cases (3–5).

Many non-neoplastic neurological disease can mimic brain neoplasms on neuroimaging or on histological examination, including multiple sclerosis, stroke, pyogenic abscess, toxoplasmosis, tuberculosis, cysticercosis, fungal infections, syphilis, sarcoidosis, Behcet disease, radiation necrosis, venous thrombosis, and others (6). The assessment of pathological an final diagnosis is based on particularly important new diagnostic techniques, such as diffusion-weighted MRI, perfusion-weighted MRI, magnetic resonance spectroscopy, single-photon emission tomography, and positron emission tomography, but also histological examination including immunohistochemistry and molecular genetics, as well as laboratory diagnosis based on serum and cerebrospinal liquor examination (6–10).

According to numerous researches, neurosarcoidosis (central and peripheral) is found in about 5–15 % patients with systemic sarcoidosis (11, 12).

The subjective of the study is to review the possible clinical manifestations of neurosarcoidosis.

Review of literature

We performed a systematic review of peer-reviewed publications identified through MEDLINE databases (searched through September 2008). The search terms were neurosarcoidosis and sarcoidosis, and the search was limited to clinical trials and articles in English. The search was extended by review of bibliographies from pertinent original reports of data and review articles. Unpublished trials and data presented only in abstract form were not included. The authors report numerous different clinical manifestations of neurosarcoidosis (Tab. 1).

The authors present numerous localisations of brain and spinal cord lesions in patients with neurosarcoidosis. Particula neurosarcoidosis diagnoses are based upon pathohistological findings in biopsy samples. The probable diagnosis is based upon clinical signs, laboratory diagnosis and magnetic resonance (MRI) findings. MRI of brain and spinal cord show nu-

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Tab. 1. Clinical manifestations of neurosarcoidosis.

Study author(s)	Symptoms
Favre J et al[13]	partial seizure
Tamagno G, Murialdo G[11]	low-grade fever, headache, visual disturbances, amenorrhea-galactorrhea syndrome and pituitary failure due to an infiltrative lesion involving the hypothalamus and thepituitary stalk
Moore FG et al[14]	bilateral facial palsy, infiltration of meninges and nerve roots
Shin JH et al[15]	central diabetes insipidus
Prüter C et al[16]	mild cognitive disorder
Friedman SH, Gould DJ[17]	psychosis, delirium, dementia
Ozerova LV et al[18]	fever, headache, thirst
Kodama M et al[19]	dementia
Tobias S et al[20]	progressive visual deterioration and proptosis
Murialdo G, Tamagno G[21]	amenorrhea-galactorrhea syndrome with hypogonadotropic hypogonadism
Vannemreddy PS et al[22]	intractable headache
Ferriby D et al[23]	axonal polyneuropathies, mononeuropathies, chronic polyradiculoneuritis
Said G et al[24]	facial diplegia, peripheral neuropathy
Teirstein A[25]	cranial nerve abnormalities, leptomeningitis, seizures, mass lesion of brain and spinal cord, pituitary dysfunction, neuropathies
Kidd D et Beynon HL[26]	pachymeningitis with cranial neuropathies, hydrocephalus, encephalopathy and hypothalamic dysfunction, radiculopathies, peripheral neuropathy, mononeuritis multiplex
Kort L et al[27]	pseudotumour
Mahadewa TG et al[28]	progressive numbness and deep sensation disturbance in bilateral lower extremities
Nakagaki H et al[29]	hemiplegia, hyperreflexia with pathological reflexes and hypesthesia
Hamada H et al[30]	upward gaze palsy
Tabuena RP et al[31]	diabetes insipidus, hypogonadism, hyperprolactinemia
Spencer TS et al[32]	seizures, spinal cord compression, increased intracranial pressure
Trabelsi L et al[33]	amenorrhea, galactorrhea
Nishie M et al[34]	dysarthria, dysphagia, weakness, multiple cranial nerve palsy
Karouache A et al[35]	peripheral neuropathy, meningitis
Sponsler JL et al[36]	partial seizures
Miscusi M et al[37]	acute intracranial hypertension
Yamaguchi S et al[12]	intracerebral hemorrhage
Huang EH et al[38]	neuro-ophthalmic manifestations
Jain V et al[39]	bilateral facial paralysis
Maeda K et al[40]	dysorientation, amnesia, dementia
Brisman JL et al[41]	vasculitis presenting with strokes
Schlienger JL et al[42]	diabetes insipidus, gonadotropic deficiency
Baussart B et al[43]	aseptic meningitis, intracranial hypothalamic lesion
Hodge MH et al[44]	paresthesias, dysarthria, right facial droop, hemiparesis
Rózsa A et al[45]	myelopathy in the cervico-thoraccic region, internuclear ophthalmoplegia
Bihan H et al[46]	diabetes insipidus, hyperprolactinemia, gonadotropin deficiency, hyperprolactinemia
Perdigao S et al[47]	lumbar pain, gait and sphincter disturbances, spastic paraparesis, hyperreflexia, sensory level, episodes of blurred vision
Tumilán LM et al[48]	delirium, seizures
Westhout FD, Linskey ME[49]	papilledema, diplopia, headache, psychotic symptoms
Walid MS et al[50]	incapability of lateral gaze

Tab. 2. Localisation of neurosarcoidosis lesions shown by MRI.

Study author(s)	MRI of brain/spinal cord
Moore FG et al[14]	mininges and lumbar nerve roots
Kodama M et al[19]	multiple lacunar infarctions
Murialdo G, Tamagno G[21]	hypotalamus and/or pituitary gland
Mahadewa TG et al[28]	medulla oblongata
Nakagaki H et al[29]	parietooccipital lobes
Hamada H et al[30]	third and fourth ventricles
Tabuena RP et al[31]	pituitary stalk
Spencer TS et al[32]	inflammatory lesion in the brain and spinal cord, leptomeningeal enhancement, hydrocephalus, intracranial hemorrhage
Trabelsi L et al[33]	pituitary tuberculoma
Sponsler JL et al[36]	amygdala, anterior hippocampus, mesial temporal neocortex
Miscusi M et al[37]	fourth ventricule lesion
Baussart B et al[43]	hypotalamic lesion, aseptic meningitis
Hodge MH et al[44]	white matter, basal ganglica infarction, dural and leptomeingeal enhancement
Rózsa A et al[45]	spinal cord
Bihan H et al[46]	hypothalamic-pituitary, parenchymal brain and spinal cord
Perdigao S et al[47]	medullary involvement
Westhout FD, Linskey ME[49]	deep white mater, cerebral aqueduct, diffuse meningeal enhancement
Walid MS et al[50]	pons, fourth ventricle
Byard RW et al[51]	hypothalamic
Marangoni S et al[52]	white mater lesions, spinal mass lesion
Suzuki M et al[53]	pituitary gland
Takano K[54]	hypothalamus, pituitary gland
Bruns F et al[55]	temporodorsal in the hemisphere, basal ganglia
Fels C et al[56]	periventricular and white matter lesions, multiple or solitary supra- and infratentorial brain lesions, leptomeningeal enhancement, involvement of brain nerves and intramedullar lesions
Iizuka T, Sakai F[57]	hypothalamus, pituitary gland, leptomeninges, cranial nerves
Hesselmann V et al[58]	fourth ventricle
Ketonen L et al[59]	ventricular enlargement, diffuse periventricular white matter changes

merous neurosarcoidosis lesions in numerous clinical manifestations (Tab. 2).

Discussion

The cause of appearance of sarcoidosis and manifestation of neurosarcoidosis are unclear. Central and peripheral neurosarcoidosis incidence varies significantly from one research to another, and is deemed to be from 5 to 15 %. The central sarcoidosis incidence is about 5 % (11, 47, 60). The clinical manifestations of neurosarcoidosis are numerous, including cranial neuropathy, facial palsy, optic nerve or other cranial nerve involvement, peripheral neuropathy, or manifestations of the central nervous system affecting the hypothalamus, pituitary gland, cerebral cortex, cerebellum, meninges and spinal cord, etc. (13–50, 60).

The variety of possible manifestations of neurosarcoidosis significantly hinder the Assessment of diagnosis and differential

diagnosis. Therefore, in addition to sound knowledge of its clinical manifestations, knowledge of additional diagnostic procedures is of importance.

Presently, the diagnosis is based on neuroradiological and laboratory tests. Magnetic resonance imaging (MRI) of the brain is presently the basic neuroradiological diagnostic method for central, but also for spinal neurosarcoidosis (44, 60). MRI represents the key method in localising the brain and spinal cord lesions: hypothalamus and/or pituitary gland, pituitary tuberculoma, pituitary stalk, cranial nerves, amygdalae, anterior hippocampus, medial temporal neocortex, deep white matter, basal ganglion infarction, spinal cord, medullary involvement, medulla oblongata, lumbar nerve roots, multiple lacunar infarctions, parietooccipital lobes, third and fourth ventricles, cerebral aqueduct, pons, temporodorsal in the hemisphere, periventricular and white matter lesions, multiple or solitary supra- and infratentorial brain lesions, inflammatory lesion in the brain and spinal cord, dural and leptomeningeal enhacement, hydrocephalus and intracranial hemorrhage.

Additional diagnostic methods include x-rays of the bones, angiotensin converting enzyme (ACE) in the serum and the cerebrospinal liquor, cerebrospinal liquor treatment and the Kveim test (19, 60). Sometimes, when possible and required, in order to reach an utmost precise diagnosis, the positron emission tomography (PET) scan is used (36).

In certain cases the final diagnosis is made by biopsy (43, 47).

According to data stated above, conclusion can be made that in addition to laboratory diagnosis, sound knowledge of clinical manifestations of neurosarcoidosis forms the basis for further differential diagnostic evaluation of potential neurosarcoidosis.

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