CASE REPORT

Diagnostics of Incomplete Brown-Sequard Syndrome caused by Meningococcal Myelitis

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Abstract

We are reviewing a 20-year old girl with the history of acute meningococcal sepsa with meningitis and thoracic myelitis resulting from an early embolia into the spinal cord, which is a very rare complication of meningococcemia. The ipsilateral loss of sensation indicates lesion of the posterior ascendent fiber tracts in the spinal cord, witout contralateral motoricity deficite, this indicating the incomplete Brown-Sequard syndrome. Somatosensory-evoked potentials (SSEP) of the n. tibialis show milder impediments of conductivity by the thoracic segment at left. Magnetic resonance (MRI) of the thoracic spine shows lesion of the posterior ascendent fiber tracts *(Fig. 2, Ref. 17)*.
Key words: Brown-Sequard syndrome, incomplete, SSEP, hypesthesia.

Brown-Sequard syndrome is an incomplete spinal cord lesion characterized by a clinical picture reflecting hemisection of the spinal cord in the cervical or thoracic region. Brown-Sequired syndrome may be the result of penetrating injury to the spine, extramedullary spinal neoplasm, cervical disc herniation, epidural hematoma, acute subdural haematoma, cervical stenosis, spontaneous spinal epidural hematoma, posterior spinal corde infarction and so on. The incomplete form of the Brown-Sequard syndrome registers omnly some of the described symptoms, depending on location of the spinal cord lesion (1–7). Somatosensory-evoked potential (SSEP) is biopotential through the spinal chord, whose image, in the form of waves, has its quantitive and qualitative characteristics. Changes in latency and amplitude of particular evoked potentials indicate a pathological change (8, 9). MRI in sagital, transverse and coronary cross-sections clearly shows pathological processes and has an important role in solving of the diagnostic dilemmas and monitoring the development of the disease (10).

Case report

The 20-year old girl was hospitalised for a sudden attack of shivers, body temperature risen up to 39.8 °C, vomiting, intensive headache and pains all over the body. She reported pains in her left lumbar region and parestesias down the left leg. After a few hours, petechiasis appeared. The patient was adynamic, prostrated, somnolent, dehidrated, of severe general status, with petechiasis all over the body. The meningitic syndrome is completely positive, no motoricity defects were noticed. The diagnostic treatment included liquor analysis, liquor culture, haemoculture, blood tests. Meningococcal myelitis was diagnosed. Subsequent liquor and blood culture findings proved this was N. meningitidis gr B. The patient was administered adequate antibiotic therapy right away. On the second day of the dicease, the body temperature fell, the general condition improved, rush ceased appearing and petechias gradually paled. There was still present intensive headache, the patient complained of paind in the back and under the left rib arch. As from the third day, she was afebrile, the headache was significantly lesser, there was no motoricity deficit, however the patient had hypesthesia and parestesias from the inferior thoracic segment left down to the medial line and down the left leg. The middle and the lower skin abdominal reflexes at left were absent. Somatosensory-evoked potentials (SSEP) were performed by Medelec Synergy – Oxford Instruments apparatus. SSEP of the nervus medianus were

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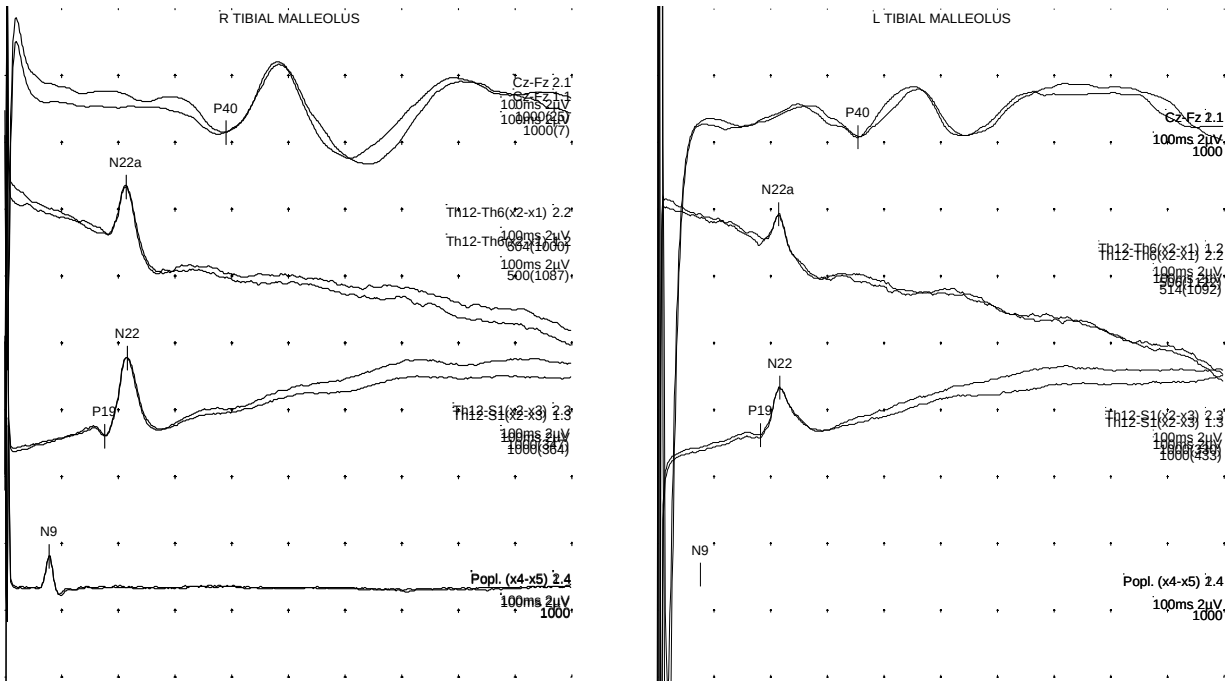

Fig. 1. SSEP of the n. tibialis – decreased amplitude of the thoracic and cortical evoked responses at left, normal findings at right.

of adequate amplitude and latency. SSEP of the nervus tibialis were obtained by stimulating both nervi tibiali behind the medial maleolus. The neurograms were detected in the popliteal fosse, the spinograms at S1, Th12, Th 6, and cortical responses above the leg sensory region. The n. tibialis stimulations produced evoked responses at all levels, with lower amplitudes of the thoracic and cortical responses at left. Thefindings indicate milder impediments in biopotential conductivity through the inferior thoracic segment at left (Fig. 1).

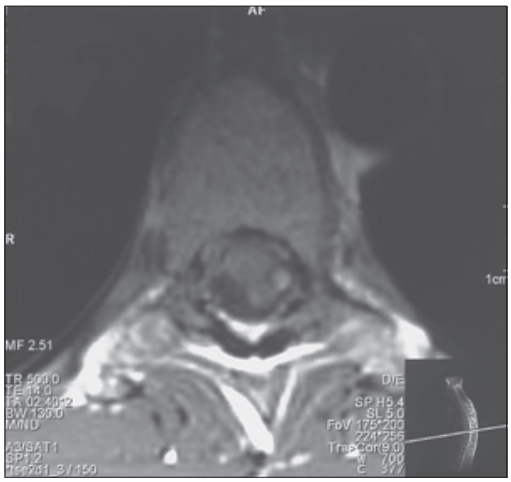

Fig. 2. MRI of the thoracic spine with contrast agent applied, transversal cross-section through the Th 5–Th 6 intervertebral space, post-contrast – posterior intramedullar process with peripheral opacity of the left side of the medula spinalis.

The MRI of the thoracic spine, made by Shimatzu EPIOS5 0.5 T, sagital and transverse cross-sections in standard presentation techniques. All cross-sections were repeated after application of the intravenous (i.v.) paramagnetic contrast agent. Series of sagital an transverse MRI cross-sections through the thoracic spinal canal segment shows vertebrae bodies of normal height, form and structure, with shallower Schmorl's herniae at the inferior thoracic vertebrae surfaces. Postcontrast sagital cross-sections made at the Th 5–Th 6 intervertebral space level, positioned intermedularly, marginally, posteriorly, make evident a spindle-shaped opacification zone. Transversal cross-section show a left posterior opacification zone (Fig. 2).

The patient has the history of an acute meningococcal sepsa with meningitis and thoracic myelitis resulting from an earlier embolia into the spinal cord, which is a very rare complication of meningococcemia. The ipsilateral loss of sensation indicates lesion of the posterior ascendent tracts in the spinal cord, incomplete Brown-Sequard syndrome. The Brown-Sequard syndrome has been described following injuries to the spinal cord, more often in the standard form and much more rarely as the incomplete form. Spinal injuries and degenerative changes make the most common causes of the Brown-Sequard syndrome, other causes being exceptionally rare (11–13). Viral and bacterial myelitises are no rarity, but no case of the incomplete Brown-Sequard syndrome caused by meningococcal myelitis has been described (14). Besides neurologic symptoms as basic signs of a spinal cord disease, besides nontypical disease signs, of significant help are SSEP since they confirm and localise the pathological process. Furthermore, intensity of the change may indicate thedegree of damages of the somatic sensation. This method

may be used in monitoring the evoked potential and its changes (12, 15). In our patient, SSEP of the n. tibialis showed milder sensory path conductivity impediments through the thoracic segment at left, which again indicated further required diagnostic treatments. MRI is a highly specific and sensitive method of imaging of the spinal cord pathological processes, including myelitis. This is the imaging method of choice in cases of the Brown-Sequard syndrome (16, 17). From the nonspecific neurological symptoms and SSEP findings, we assessed needs for further diagnostic differentiation, wherefore MRI of the thoracic spinal cord segment was made. MRI of the thoracic spine with application of contrast agent showed lesion that is well opacified with the contrast agent, this corresponding to lesion of posterior ascendent fiber tracts in the spinal cord.

To conclude with, the meningococcal myelitis is a possible complication of the meningococcal meningitis and the meningococcal sepsa. In cases of the suspected Brown-Sequard syndrome, the diagnostic treatment is to be completed. Evoked potentials indicate a possible pathological process by chanes of latency and/or amplitudes of the evoked response of a given spinal cord segment. This enables more precise assessment of location and gravity of the process, as well as establishing of indications for an MRI. This is the method of choice in diagnostics of the incomplete Brown-Sequard syndrome.

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