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Case report

FDG PET findings leading to diagnosis of neurosarcoidosis

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1. Introduction

Sarcoidosis is a multisystemic inflammatory disease of unknown aetiology characterized by the accumulation of non-caseating epithelioid granulomas in involved organs. The most common locations are the lung, the skin and the eye [1]. The nervous system is estimated to be involved in 5–15% [1]. Clinical diagnosis of neurosarcoidosis is often delayed due to its heterogenous manifestations. Cranial neuropathies, basal meningitis, hypophyseal and hypothalamic disorders are common. Spinal cord involvement, radiculopathy, peripheral neuropathy, isolated cauda-equina-syndrome and myopathy can also occur [1]. We present a patient with findings of [^{18}F]-fluorodeoxyglucose (F18-FDG) imaging which were the indication of discovering neurosarcoidosis and of initiating appropriate therapy.

2. Case report

A 37-year old woman complained of back pain, distal paraesthesia in all extremities, a feeling of pressure belt-shaped around her abdomen as well as facial palsy, unsteadiness and hearing impairment for five weeks. Still as an outpatient cerebral and lumbar MRI were found unsuspicious.

The first CSF analysis showed a slight lymphocytic pleocytosis (44 cells/μl) with elevated protein level (989 mg/l). Initially neuroborreliosis was suspected, and treatment with ceftriaxone was initiated.

Despite antibiotics the symptoms worsened, and the patient was referred to our neurological department. On admission the patient was fully orientated, the cognitive abilities were normal and there was no meningism. The cranial nerves VII (left > right) and VIII were affected as bilateral hearing was impaired. The patellar tendon and abdominal reflexes were missing bilaterally. A belt-shaped sensory disorder was found affecting the lower thoracic and upper lumbar roots (Th10-L2). Pyramidal signs were not present. Bladder and bowel function were normal. The patient was afebrile and the vital signs were normal.

The routine laboratory results were within normal range and the first interpretation of the chest X-ray was judged to be normal.

Motor conduction velocity and compound muscle action potential of both median, ulnar, peroneal and tibial nerves were normal. The distal motor latency of the peroneal nerve was slightly prolonged bilaterally (5.6 and 5.4 ms; normal value <5 ms). F-waves of both tibial and median nerves were normal (shortest latency). The sensory conduction velocity and sensory action potential amplitude were not analysed.

The second CSF analysis revealed a normal cell count (4/μl) with highly elevated protein level (1360 mg/l). This finding was interpreted as a cytoalbuminic dissociation. As the patient had been deteriorated, despite a sufficient treatment of antibiotics, the diagnosis of neuroborreliosis was abandoned, and a Guillain–Barré

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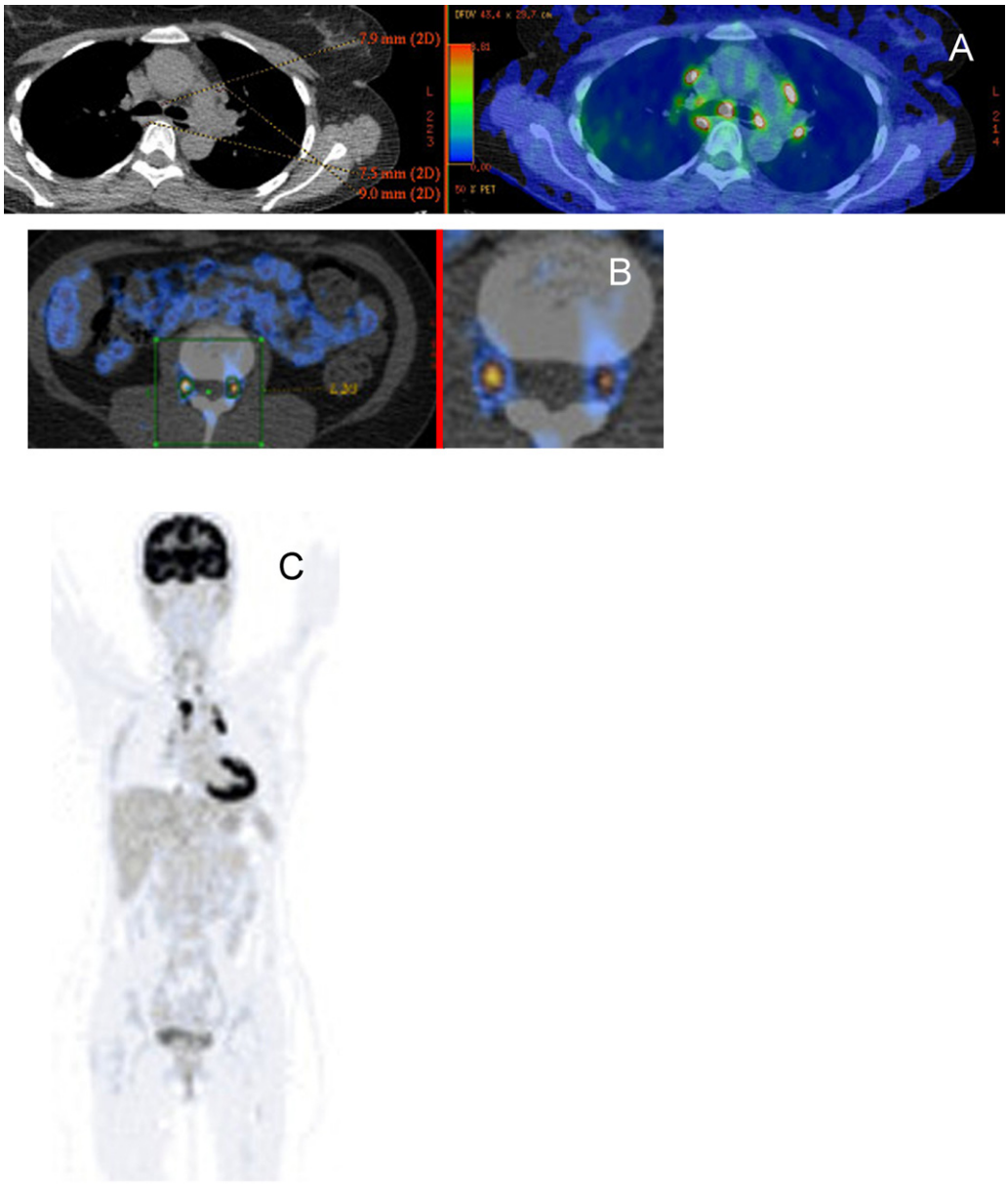

Fig. 1. Panel A shows hypermetabolic mediastinal and hilar foci in FDG PET/CT (right). In the CT scan the enlarged lymph nodes are marked with diameters between 7.5 and 9 mm (left). Panel B shows hypermetabolism at the lumbar spinal nerve roots indicating inflammatory changes (right; left in more accentuated figure). Panel C presents coronal maximum intensity projection image (MIP) demonstrating FDG uptake predominantly in the mediastinal lymph nodes. Normal physiologic intense FDG activity is seen in the brain, myocardium and to a less extent in the bladder.

syndrome was considered. Thus a treatment course of intravenous immunoglobulin (IVIG), 30 g per day for 5 days, was initiated. There was only a slight clinical improvement according to the patient. Seeking a tumour was ordered to rule out a paraneoplastic cause of the clinical syndrome. In order to use a very sensitive method for searching a malignant process of unknown origin, a F18-FDG PET was performed. The result provided the cue to sarcoidosis with an increased tracer-uptake in mediastinal lymphnodes (Fig. 1A) and additionally in lumbar spinal ganglia (Fig. 1B). The diameter of the enlarged lymph nodes amounted between 7.5 and 9 mm. An uptake at the level of the salivary glands was not detected (Fig. 1C). At second glance the X-ray showed a slightly enhanced mediastinum. A gynaecological examination was normal. After confirming the suspected diagnosis of neurosarcoidosis by following thoracic and abdominal CT-scanning as well as bronchoscopic biopsy (Fig. 2) a treatment regime with prednisolone (prednisolone 500 mg daily intravenously for five days, followed by prednisolone 1.5 mg/kg per day orally for five weeks and a consequent slow taper) was initiated, and symptoms were soon regressive. The results of all the supplementary examinations are summarized in Table 1 (Table 1). A follow-up after 3 months revealed a residual slight impairment of the facial muscles and a persisting hearing impairment. The sensory disturbances and the pain regressed completely. Meanwhile the patient is fully re-engaged in her profession. The steroid treatment was continued in lower dosages. Follow-up FDG PET had not been performed.

3. Discussion

Our case clearly indicates that diagnosing neurosarcoidosis is often delayed, as the clinical symptoms are not specific and mimicking other neurological diseases such as neuroborreliosis or Guillain–Barré syndrome [1].