Short Report

An Acute Cerebellar Syndrome. Who Is Involved?

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Summary

Case Report

We report the case of a 79 year-old bipolar patient treated with Lithium who developed an acute cerebellar syndrome following a Legionellosis episode. Clinical evolution, biological exams, structural and functional imaging were all favoring a post-infectious disease.

Discussion

Cerebellar disorders in Legionsaire’s disease are considered an inflammatory process although pathophysiology remains poorly understood. Granting the recent studies on the role of lithium as neuroprotective in neuroinflammation, we empirically hypothesized that lithium might have played a beneficial role in this case.

Conclusion

This case shows that FDG PET/CT can help confirm the diagnosis of post-Legionella cerebellitis and should be easily prescribed in this indication. Furthermore, we suspect that Lithium might have a neuroprotective effect in this post-infectious phenomena.

Subarachnoid haemorrhage (SAH) accounts for approximately only 5% of strokes [1], yet occurs at a young age leading to lengthy burden. SAH outcomes research has rarely examined psychological outcomes, despite their prevalence and substantial contribution to recovery [2], and potentially to functional outcomes.

Keywords: Cerebellar Disorders; Legionellosis; Cerebellar Neurodegeneration; Acute Ataxia; Neu-roinflammation

He received noninvasive ventilation and high flow oxygen therapy. The *Legionella pneumophila* urinary antigens assay came back positive. The antibiotherapy was eventually changed to Levofloxacin 500mg 2/day. The evolution was favorable and he was transferred to a traditional Pneumology unit.

Fifteen days after the initial fever, the patient developed a severe cerebellar syndrome overnight. There was neither confusion, nor tremor, nor oculomotor disturbances, nor fever. Blood tests showed decreased inflammatory markers. Head-CT showed no sign of hemorrhage. Lithium blood concentration was below therapeutic range and was highly monitored. The patient was transferred to our Neurology Department. At the admission, he showed a severe cerebellar syndrome (cerebellar ataxia and dysarthria). The severity was 31 on the Scale for Assessment and Rating of Ataxia (SARA) [1]. He was conscious and oriented. Biological screening was normal. Brain Magnetic Resonance Imaging (MRI) showed diffused encephalic atrophy, but neither significant hypersignal on Diffusion, FLAIR, T2*, Time Of Flight weighted images, nor Gadolinium contrast lesions on the initial MRI, nor on the 4th week control. CSF analysis was normal. Onconeuronal antibodies were negatives. EEG showed diffuse slow waves predominantly in anterior regions compatible with encephalopathy.

Finally, a FDG PET/CT showed notable hypometabolism of both cerebellar hemispheres, respecting the vermis, compatible with a post-infectious disease (Figure 1). We confirmed the diagnosis of acute cerebellitis secondary to *Legionella pneumophila*. One month after the onset, the patient was still dysarthric but intelligible.
was not reported in any Lithium induced cerebellar syndrome published cases [3]. Our patient’s brain MRI did not show any specific lesions, nor signs of Gay-et-Wernicke encephalopathy, nor Acute Disseminating Encephalomyelitis (ADEM) which latter have been associated with Legionella Encephalopathy in numerous recent case reports [4]. Other key elements were the absence of meningitis arguments which excluded a direct bacterial aggression and the PET-FDG/CT outcome which showed non-systemic focal hypometabolism defects compatible with post-infectious sequels. Functional imaging is more sensitive than structural MRI when it comes to inflammatory lesions. [5,6] In FDG-PET/CT, hypermetabolic changes occur in acute inflammatory lesions and hypometabolic changes in inflammatory sequels [7]. Although we cannot confirm post-Legionella cerebellitis based on the unique FDG PET/CT, the body of arguments from clinic to functional and structural imaging asserts our hypothesis.

He showed significant clinical improvements and could circulate with a walker. SARA was then 16. The patient was eventually discharged and successfully returned home.

Discussion

Legionellosis is an unfrequent but grave infectious disease with high morbidity and mortality. Neuro-logical symptoms such as confusion, disorientation, stupor and coma are common extrapulmonary signs in this disease (up to 40-50%) [2]. Cerebellar dysfunction associated with Legionellosis’ incidence has been estimated to 3.7% among one of the earliest cohorts of Legionnaire’s disease patients [2].

In our case, the difficulty was to determinate the origin of the acute cerebellar syndrome. We ruled out lithium toxicity since the patient clinically improved and never showed any toxicity symptoms while treated with Lithium Carbonate. This aspect was not reported in any Lithium induced cerebellar syndrome published cases [3]. Our patient’s brain MRI did not show any specific lesions, nor signs of Gay-et-Wernicke encephalopathy, nor Acute Disseminating Encephalomyelitis (ADEM) which latter have been associated with Legionella Encephalopathy in numerous recent case reports [4]. Other key elements were the absence of meningitis arguments which excluded a direct bacterial aggression and the PET-FDG/CT outcome which showed non-systemic focal hypometabolism defects compatible with post-infectious sequels. Functional imaging is more sensitive than structural MRI when it comes to inflammatory lesions. [5,6] In FDG-PET/CT, hypermetabolic changes occur in acute inflammatory lesions and hypometabolic changes in inflammatory sequels [7]. Although we cannot confirm post-Legionella cerebellitis based on the unique FDG PET/CT, the body of arguments from clinic to functional and structural imaging asserts our hypothesis.
Physiopathology of cerebellar dysfunction in Legionnaire’s disease remains unknown. Most patients with cerebellar syndrome associated with Legionellosis suffer severe dysarthria and ataxia sequelae with modest clinical improvements. [8] Since normal CSF, normal brain imaging (CT and MRI) and slow-waving encephalopathy signs on EEG are usually found, it has been postulated that an indirect inflammatory reaction involving Lipopolysaccharide (LPS) endotoxin may be the reason of this rare affection [9,10]. LPS is the major immunodominant antigen of Legionella pneumophila and all Legionella species. [11] Activation of microglia precedes and triggers neuronal death. [12] Recent studies have shown that lithium can be neuroprotective by reducing microglial migration, hence, inflammation induced neurotoxicity. [13] Dong et al. showed that lithium reduces lipopolysaccharide-induced microglial activation via inhibition of toll-like receptor 4 (TLR4) expression by activating a specific intracellular pathway. [14] Surprisingly, our 79 year old patient showed significant clinical improvements of his cerebellar symptoms with moderate sequelae. We hypothesized that lithium might have played an anti-inflammatory action in this case by reducing neurotoxicity in this acute cerebellitis post-Legionellosis. However further experimental studies should confirm this hypothesis in order to elucidate eventually new therapeutic solutions for this highly disabling affection.

Conclusion

This case report is, to our knowledge, the first to show that FDG PET/CT can help confirm the diagnosis of post-Legionella cerebellitis and should be easily prescribed in this indication. Furthermore, we suspect that Lithium might have a neuroprotective effect in this post-infectious phenomena.

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Competing Interests Statement

There are no competing interest to declare.

Contributorship Statement

Salimata Gassama is a medical intern who wrote and designed the submitted article, acquired and interpreted data, participated in the patient’s medical care during the entire hospitalisation in Neurology until he was discharged.

Dr. Damien Fetter is a senior Neurologist who supervised the patient’s medical care during the entire hospitalisation in Neurology until he was discharged and revised the article critically for important intellectual content.

Prof. Xavier Monnet is an ICU Professor who revised the article critically and significantly, and contributed in the conception of the short report, especially on the critical care and pneumology aspects.

Dr. Bertrand Bourre, is a senior Neurologist specialised in Neuroinflammation who revised the article critically, especially on the neuroinflammation aspect of this report.

Dr. Mathieu Chastan is a Senior Nuclear Medicine radiologist who interpreted the patients FDG-PET/CT and revised the article critically.

Prof. Didier Hannequin is a Neurology Professor who revised the article critically.

Prof. Didier Hannequin is a Neurology Professor, head of the department of the neurology department in Rouen who contributed in the design of the work and revised the article critically.

Dr. Nicolas Mirlink is a Senior Neurologist who participated in the patient’s medical care during the entire hos-pitalisation in Neurology until he was discharged, critically revised the article and gave the final approval of the version published.

References


