***Case report***

**Brain MRI in Gougerot–Sjögren syndrome: A Case Report with diagnostic approach and imaging review**

**ABSTRACT**

We report here the case of a 35-year-old patient who was followed for Gougerot's disease and who had headaches for several weeks.

Gougerot-Sjögren's disease is a systemic autoimmune disease characterized by impaired specific glands (notably the lachrymal and salivary glands), which presents various systemic symptoms that may affect multiple organs. Besides, this condition may develop neurological symptoms. An MRI of the brain is therefore useful in these instances to evaluate dual pathology.

**Key words:**

MRI, Sjögren's syndrome, Aseptic meningitis, T2 hypersignal.

**INTRODUCTION**

Le syndrome de Sjögren primitif est une maladie rare, dont la prévalence est estimée à environ 10 cas pour 10 000 habitants selon les données épidémiologiques récentes [1]. Les femmes étant atteintes dix fois plus souvent que les hommes, avec une apparition de la maladie généralement autour de l’âge de 50 ans ; toutefois, il peut se manifester beaucoup plus tôt, entre 20 et 30 ans, ces formes précoces étant souvent cliniquement plus sévères [2].

Sjögren syndrome (SGS) is characterized by lymphocyte infiltration of exocrine glands, including the salivary and lacrimal glands, resulting in dry eye. Although often seen as a restricted disorder, SGS can also bring systemic implications, involving various organs, including the central nervous system.[3].

Here we present a rare case of a 35-year-old female patient followed for Gougerot-Sjögren syndrome and the role of MRI in the diagnosis.

**CASE PRESENTATION**

A 35-year-old woman with a known history of primary Sjögren’s syndrome was referred to the neurology department for evaluation of persistent headaches that had progressively worsened over the preceding six weeks. The headaches were described as diffuse, constant, and of moderate intensity, with no response to standard analgesic therapy. There was no associated photophobia, nausea, vomiting, or visual disturbances. The patient denied any recent infections, trauma, or exposure to toxic substances. There was no history of seizures or focal neurological deficits.

Her past medical history included a diagnosis of Sjögren’s syndrome made two years earlier, based on sicca symptoms, positive anti-SSA (Ro) and anti-SSB (La) antibodies, and labial salivary gland biopsy demonstrating lymphocytic infiltration. She had been managed with hydroxychloroquine and symptomatic therapy.

On clinical examination, the patient was alert, oriented, afebrile, and hemodynamically stable. General and systemic physical examinations were unremarkable. A detailed neurological examination revealed no focal deficits, cranial nerve abnormalities, or cerebellar signs.

Laboratory investigations showed a normal complete blood count. Inflammatory markers revealed a mildly elevated erythrocyte sedimentation rate (ESR: 35 mm/hr), while C-reactive protein (CRP) was within normal limits. Lumbar puncture yielded clear cerebrospinal fluid (CSF) with mild lymphocytic pleocytosis (12 cells/mm³), elevated protein concentration (65 mg/dL), normal glucose levels.

Brain magnetic resonance imaging (MRI) was performed using T1, T2, FLAIR, diffusion-weighted imaging (DWI), and post-contrast T1 sequences. MRI demonstrated multiple bilateral hyperintense lesions in the periventricular and subcortical white matter on T2 and FLAIR sequences. Diffuse leptomeningeal thickening and enhancement were observed on post-contrast T1 images, suggestive of aseptic meningitis. There was no evidence of hydrocephalus, mass effect, or restricted diffusion.

**The patient underwent a brain MRI (Fig. 1):**

A close up of a brain scan

AI-generated content may be incorrect. A close up of a scan

AI-generated content may be incorrect.

B

A

**FLAIR**

**Diffusion**

**T2**

A computer screen shot of a brain

AI-generated content may be incorrect.A close up of a brain scan

AI-generated content may be incorrect.

D

C

**T1 c+**

**T2**

**Fig.1:** Axial FLAIR (A), Axial diffusion (B), Coronal T2 (C), Axial T1 post-contrast (D): The images reveal the presence of white matter lesions, characterized by hyperintensities on T2 and FLAIR sequences, as well as thickening and enhancement of the meninges, described as hyperintensities on T2.

Based on the clinical context and imaging findings, a diagnosis of central nervous system (CNS) involvement secondary to primary Sjögren’s syndrome was established. The patient was initiated on high-dose corticosteroid therapy (intravenous methylprednisolone 1 g/day for 5 days), followed by a tapering regimen of oral corticosteroids. She exhibited rapid symptomatic improvement and was discharged on maintenance immunosuppressive therapy with azathioprine.

**DISCUSSION:**

The foundations of SS were established in the early 20th century. In 1925, Gougerot described a clinical picture associating dryness of the eyes, mouth, nose, and larynx, suggesting the disease’s systemic dimension [1]. In 1933, Henrik Sjögren expanded upon these observations, linking dryness with chronic arthritis in women, thereby confirming the autoimmune nature of SS [4].

The etiology of SS remains incompletely understood and differs from related autoimmune diseases such as systemic lupus erythematosus (SLE). Multiple factors contribute to its pathogenesis, including genetic, hormonal, immunologic, and viral components [5]. The disease is characterized by autoimmune infiltration of the exocrine glands, leading to their progressive destruction [6]. Autoantibodies targeting nuclear and glandular structures exacerbate tissue damage via chronic inflammation. Dendritic cells and macrophages play a central role in this immune dysregulation [6].

In addition to ocular and oral involvement, SS affects multiple organ systems, confirming its systemic nature. Joint manifestations such as pain, morning stiffness, and swelling are common. Cutaneous findings include dryness, rash, or vasculitis. Pulmonary involvement may present as recurrent infections, chronic cough, or dyspnea. Renal complications include glomerulonephritis and tubular disorders [7]. Vascular phenomena such as Raynaud’s syndrome or systemic vasculitis have also been described [8][9].

Sjögren’s syndrome may give rise to a wide spectrum of neurological symptoms. Lymphocytic infiltration of the nervous system, combined with the production of autoantibodies against neuronal antigens, leads to chronic inflammation, neuronal damage, and vascular injury, potentially resulting in infarctions or hemorrhages [6][7]. Clinically, neurological involvement may manifest as headaches, cognitive dysfunction, peripheral neuropathies, or dysautonomia.

Brain MRI is an essential and irreplaceable tool for investigating neurological manifestations in SS. It enables the detection of structural lesions not evident upon clinical examination and allows for the assessment of lesion extent and activity. T2-weighted and FLAIR sequences are particularly sensitive in detecting hyperintensities in the subcortical and periventricular white matter, often mimicking lesions observed in multiple sclerosis (MS) [10][11][12]. Gadolinium-enhanced T1-weighted imaging reveals areas of active inflammation, indicative of ongoing autoimmune processes.

Specific findings such as corpus callosum atrophy, basal ganglia abnormalities, and brainstem lesions have also been reported, suggesting widespread and deep central nervous system involvement. Assessment of cerebral atrophy, especially in the temporal and parietal lobes, offers insight into disease chronicity and severity [11]. In some cases, MRI demonstrates lymphocytic meningitis, visible as meningeal thickening and contrast enhancement, often associated with symptoms like persistent headaches or cognitive decline.

Spinal cord involvement, including transverse myelitis, typically appears as T2 hyperintensities in the cervical cord. These lesions can be distinguished from other etiologies based on their topography, longitudinal extent, and gadolinium enhancement profile [13]. Thus, MRI not only facilitates accurate diagnosis but also guides therapeutic decisions and longitudinal monitoring.

The presence of specific autoantibodies (anti-SSA, anti-SSB) and salivary gland biopsy confirming lymphocytic infiltration support the diagnosis. Cerebrospinal fluid (CSF) analysis frequently shows elevated IgG index and oligoclonal bands, also found in MS, necessitating a strict correlation with MRI findings [14].

In our case, a female patient was initially evaluated for chronic headaches. Brain MRI revealed aseptic meningitis and white matter hyperintensities, consistent with SS-related neurological involvement. This presentation warranted close radiological monitoring and regular neurocognitive testing to adjust therapeutic strategy.[15].

**CONCLUSION:**

Magnetic resonance imaging (MRI) plays a pivotal role in the identification and monitoring of neurological complications associated with Sjögren’s syndrome. Beyond detecting clinically silent abnormalities, MRI enables the differentiation of SS-related central nervous system lesions from other demyelinating disorders, particularly multiple sclerosis. Specific radiological findings, such as periventricular hyperintensities, corpus callosum atrophy, or meningeal enhancement, may serve as imaging biomarkers of disease activity or chronicity.

A multidisciplinary diagnostic approach integrating clinical, serological, and radiological data is essential to improve diagnostic accuracy, guide immunosuppressive therapy, and monitor disease progression. A deeper understanding of SS-specific imaging patterns could contribute to earlier diagnosis and more individualized treatment strategies.

**CONSENT**

All authors declare that ‘written informed consent was obtained from the patient family for publication of this case report and accompanying images’.

**ETHICAL APPROVAL**

All authors hereby declare that all experiments have been examined and approved by the

appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

**DISCLAIMER (ARTIFICIAL INTELLIGENCE)**

Author(s) hereby declares that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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