Acta Biomed 2023; Vol. 94, N. 6: e2023258 DOI: 10.23750/abm.v94i6.15234

Case report

Anti-recoverin antibodies in a cerebellar syndrome without retinal involvement

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Abstract. Anti-recoverin antibodies are typically found in cancer-associated retinopathy or autoimmune diseases. We present a case of anti-recoverin positive cerebellar syndrome without any signs of malignancy or retinopathy. The patient was treated with steroids and intravenous immunoglobulins, improving cognitive and motor symptoms. (www.actabiomedica.it)

Key words: recoverin, cerebellitis, anti-recoverin antibodies, dizziness, ataxia, dysarthria

Introduction

Recoverins are calcium-binding proteins primarily expressed in retinal rod photoreceptors and primarily involved in phototransduction. Anti-recoverin antibodies have been found to be linked to cancer-associated retinopathy (CAR) (1, 2) and autoimmune retinal degeneration (3). While there have been a few reports linking anti-recoverin antibody positivity to extra-ocular pathologies, such as LES (4) or Covid-19 (5), the underlying pathophysiology has not been fully elucidated, as recoverin appears to be exclusively located in the retina and pineal gland (6). We present a case of anti-recoverin-positive cerebellitis without ocular involvement.

Case description

A 57-year-old male with a history of drug addiction, uncontrolled hypertension, and obesity, presented to our hospital with a subacute onset of dizziness, ataxia, and a feeling of light-headedness. He had previously been treated for labyrinthitis with levosulpiride and diclofenac, but his symptoms persisted and, in addition, he developed dysarthria and dysphagia. Neurological evaluation revealed a mild left VII cranial nerve deficit, dysarthria with intelligible speech, left lower limb hyposthenia (MRC 4/5), bilateral dysmetria and dyssynergia with left prevalence at upper and lower limbs, severe ataxia with multidirectional oscillations and widened base in orthostatic position, requiring bilateral support. Radiological examinations were free from alterations. Diagnostic hypotheses included acute polyradiculoneuritis with prevalent involvement of the cranial nerves (Miller-Fisher syndrome) and rhombencephalitis. Despite negative results from brain MRI, electroneurography, and blink reflex test, a diagnostic lumbar puncture revealed mild protein elevation (51 mg/dL) and 28 leukocytes/mm^3 (predominantly lymphocytes) with no evidence of viral or bacterial infection. Viral and bacterial serological tests showed only positivity for anti-HBcAg antibodies. After ruling out infectious causes, we treated the patient with intravenous immunoglobulins (7) for five days, resulting in a regression of the left VII cranial nerve deficit. Subsequent tests, including chest-abdomen CT and thyroid and testicles ultrasound, as well as neoplastic

serological markers, were negative. When the oligoclonal bands were finally available (>6 exclusively liquor bands, type 2 interpretative criterion), high-dose steroid therapy (1g methylprednisolone iv for five days) was initiated, resulting in an improved trunk control and ataxia. The rheumatological panel showed positivity for anti-ENA antibodies (SSA, Pm/Scl-100), p-ANCA, granular ANA+ with 1:320 dilution, anti-cardiolipin IgG, and slight positivity for anti-recoverin antibodies. Due to this dysimmune process, a post-hospital salivary gland biopsy was planned, also considering the maternal familiarity with Sjogren's disease. An ophthalmologist examination did not reveal any ocular involvement. We re-evaluated the patient several months after discharge and after intensive physiotherapy. The dizziness and the feeling of light-headedness had completely disappeared, while ataxia was still detectable but improved in the last 20-30 days after i.v. immunoglobulins.

Discussion

The absence of classical associated conditions such as retinopathy or cancer made diagnosis and treatment more challenging. Moreover, while the patient had multiple comorbidities contributing to his overall medical complexity, we have ruled out any other possible condition that could have caused such symptoms. A critical limitation of this case report is the lack of definitive evidence linking anti-recoverin antibodies to cerebellitis. While the presence of anti-recoverin antibodies in the patient's serum and the clinical improvement with immunomodulatory therapy support this hypothesis, we have not found these antibodies in CSF. However, the clinical improvement after immunomodulatory therapy highlights the importance of considering autoimmune etiologies in patients with cerebellar syndromes.

Conclusion

In conclusion, we present a case of anti-recoverin positive cerebellitis without retinopathy or neoplasia. While the underlying mechanism is still unclear, we believe that anti-recoverin antibodies may have affected the patient's symptoms. Further research is needed to elucidate the pathophysiology of this condition and establish definitive diagnostic and therapeutic guidelines.

Funding: This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Ethic Committee: The patient gave informed consent in the use of his personal data for research and teaching purposes.

Conflict of Interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors Contribution: Collecting data, writing and original draft preparation MM; Review and editing GB, LZ.

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Received: 28 August 2023
Accepted: 28 September 2023
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