www.directivepublications.org

The Annals of Internal Medicine

ISSN 3064-6650

Review Article

Anti-Ri Paraneoplastic Neurological Syndrome in Breast Cancer: An a Typical Case and Contemporary Review.

Fernando Gonzalez Trujillo [1]*, Tomas de los Milagros Rengifo Sandoval [2], Karol Yulissa Cano García [3].

Affiliation:

[1] *MD Clinical Neurology, Neuro-Oncology, MSc in Neuroscience, Clínica de Occidente, S.A., Centro Integral de Cáncer, Santiago de Cali, Colombia;* ***Email:*** *fernando.gonzaleztrujillo@gmail.com*

[2] *MD Coloproctology Assistant. Clínica de Occidente, S.A., Santiago de Cali, Colombia;* ***Email:*** *tomasrengifo93@gmail.com*

[3] *MD Emergency Medicine, MD Neurology-Neurosurgery Assistant, Clinica de Occidente, S.A., Santiago de Cali, Colombia;* ***Email:*** *karolcanog.24@gmail.com*

Abstract

Background: Paraneoplastic neurological syndromes (PNS) associated with anti-Ri antibodies are characterized by a low prevalence and are frequently overlooked. They are typically associated with breast tumors and exhibit significant variability in clinical presentation.
Objective: To describe an atypical case of anti-Ri PNS in a patient with breast cancer and to review the current literature on its clinical spectrum, diagnostic challenges, and therapeutic approaches.
Methods: We present a case study of a patient with breast carcinoma associated with anti-Ri antibodies. A narrative review of the literature was conducted using PubMed and Scopus (2000-2024) with the terms “anti-Ri,” “paraneoplastic neurological syndrome,” and “breast cancer.”
Results: Symptoms partially improved with immunotherapy but persisted despite oncologic treatment. Fewer cases of anti-Ri PNS were identified in the literature, with extrapyramidal involvement reported in only a minority, and with limited response to immunomodulatory therapy.
Limitations: This is a single case report with a narrative review; potential selection bias in the literature cannot be excluded.
Conclusion: Anti-Ri PNs may present with atypical extrapyramidal features in patients with breast cancer. Early recognition is critical, as timely diagnosis may guide oncologic and immunologic management, although prognosis remains guarded.

Keywords: Paraneoplastic neurological syndrome; anti-Ri antibodies; central nervous system; cerebellar ataxia; autoimmune response.

INTRODUCTION

Paraneoplastic neurological syndromes (PNS) are rare but potentially devastating disorders that arise from immune-mediated responses to malignant tumors, even in the absence of metastasis (1). Syndromes associated with anti-Ri antibodies have received more attention recently due to their complex clinical manifestations and strong link with breast carcinoma. Anti-Ri antibodies, also known as type 2 anti-neuronal nuclear antibodies (ANNA-2), are a marker of the syndrome and are most associated with paraneoplastic cerebellar degeneration and opsoclonus-myoclonus syndrome (**Table 1**).

This table summarizes the clinical manifestations, tumor associations, and therapeutic strategies of anti-neuronal nuclear antibody type 2 (ANNA-2, Anti-Ri) paraneoplastic neurological syndrome. The syndrome predominantly affects cerebellar and brainstem structures and is characterized by opsoclonus-myoclonus, spasticity, and extrapyramidal features, including Parkinsonism and mandibular dystonia. Tumor screening is essential, as approximately 70% of cases are associated with breast or lung cancer. Management strategies include tumor removal, corticosteroids, plasma exchange, intravenous immunoglobulin (IVIG), and rituximab (8). Breast cancer is one of the most prevalent malignancies affecting women worldwide, and its association with anti-Ri PNS poses significant clinical challenges. Although the precise mechanism by which breast tumors trigger an autoimmune response remains unclear, few studies report a correlation between HER2 receptor expression and the presence of anti-Ri autoantibody in breast cancer patients (1,2).

***Corresponding Author:** **Fernando Gonzalez Trujillo,** MD Clinical Neurology, Neuro-Oncology, MSc in Neuroscience, Clínica de Occidente, S.A., Centro Integral de Cáncer, Santiago de Cali, Colombia. **Email:** fernando.gonzaleztrujillo@gmail.com.
Received: 05-November-2025, Manuscript No. TAOIM - 5230 **; Editor Assigned:** 08-November-2025 **; Reviewed:** 26-November-2025, QC No. TAOIM - 5230 **; Published:** 17-December-2025, **DOI:** 10.52338/taoim.2025.5230.
Citation: Fernando Gonzalez Trujillo. Anti-Ri Paraneoplastic Neurological Syndrome in Breast Cancer: An A typical Case and Contemporary Review. The Annals of Internal Medicine. 2025 December; 14(1). doi: 10.52338/taoim.2025.5230.
Copyright © 2025 **Fernando Gonzalez Trujillo.** This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Table 1. Clinical Features of Anti-Ri (ANNA-2) PNS

Category	Details
Antibody	Anti-neuronal nuclear antibody type 2 (ANNA-2, Anti-Ri).
Symptoms	Nystagmus, ataxia, opsoclonus-myoclonus (predominantly cerebellar in 90% of cases), spasticity, brainstem signs, Parkinsonism, mandibular dystonia, and slowly progressive gait disturbance.
Associated Tumors	Breast cancer (70% of cases), lung cancer.
Treatment	Tumor removal, corticosteroids, plasma exchange, intravenous immunoglobulin, and intravenous rituximab.

The primary objective of this study is to describe an atypical case of anti-Ri PNS in a patient with breast cancer and to review the current literature on its clinical spectrum, diagnostic challenges, and therapeutic approaches. The clinical case presented highlights an atypical manifestation, as the patient did not exhibit the classic features typically associated with anti-Ri–positive PNS. In addition, this review examines diagnostic challenges and therapeutic approaches to improve recognition and management of neurological complications in patients with breast cancer. Multidisciplinary evaluation and individualized treatment are crucial for optimizing outcomes and preserving quality of life.

METHODS

Case report

A 65-year-old woman with a history of breast carcinoma diagnosed in 2011 (stage IIA luminal, HER2-negative, Ki-67 proliferation index 10%) developed metastatic disease involving the skin, subpleural thoracic region, lymph nodes, and mediastinum. In February 2018, she was admitted to the emergency department after several months of progressive gait and coordination decline, which had advanced to complete immobility and dependence for all activities of daily living.

On admission, she appeared emaciated but was cognitively intact. Voluntary movements were severely impaired, and she was unable to reposition herself in bed. Neurological examination revealed both horizontal and vertical nystagmus, marked extrapyramidal rigidity, severe spastic quadriparesis, spontaneous clonus, hyperactive deep tendon reflexes (including a brisk masseter reflex), and muscle atrophy most pronounced in the intrinsic hand muscles.

From a clinical perspective, the presentation suggested a combination of pyramidal and extrapyramidal syndromes, with suspicion of stiff-person syndrome (SPS). The patient provided prior imaging performed one year before admission, including a cervical MRI, which showed bone marrow involvement of the vertebral bodies and degenerative changes in the left posterolateral elements of C2 with contrast enhancement, as well as degenerative changes at C3 and C4.

At the institution, updated imaging included brain MRI and complete spinal MRI with gadolinium, both of which were unremarkable. Further studies included electromyography and nerve conduction tests of all four limbs, which showed no evidence of myopathy, polyneuropathy, or motor neuron disease. Cerebrospinal fluid analysis demonstrated normal cell counts and cytochemical parameters, and cytology was negative.

Screening tests excluded metabolic, infectious, and autoimmune causes, and no relevant findings were identified. Given the patient's history of breast cancer, serological testing for paraneoplastic autoimmunity was performed and returned positive for anti-Ri onconeural antibodies, confirming the diagnosis of paraneoplastic neurological syndrome.

Consequently, targeted therapy was initiated with plasmapheresis (8 exchanges) and intravenous immunoglobulin (0.4 g/kg/day for 5 days, administered in two cycles). The patient demonstrated gradual and sustained improvement in motor function, ultimately regaining the ability to ambulate independently with the aid of a walker. She was discharged in stable condition and continued routine follow-up in outpatient neurology consultations.

The patient's clinical course was characterized by fluctuations, alternating between periods of stability and relapse, with partial improvement following serial intravenous immunoglobulin infusions. This treatment approach allowed her to maintain a degree of autonomy in daily activities, with family support for approximately two years.

Her final hospitalization was triggered by fever and hypoactive delirium, with suspected pulmonary sepsis as the primary focus. The patient subsequently developed acute ventilatory failure requiring orotracheal intubation and transfer to the intensive care unit. During hospitalization, she also developed a urinary tract infection (as indicated by urine culture positive for multisensitive Escherichia coli), which was treated with targeted antibiotics. Despite these interventions, her condition deteriorated to septic shock and multiorgan failure. Hemofiltration and high-dose vasopressor support were required, but clinical stability could not be achieved, and the patient ultimately passed away.

Table 2 presents the relevant laboratory results associated with the diagnosis of breast carcinoma and confirming the presence of anti-Ri paraneoplastic neurological syndrome.

Table 2. Relevant laboratory and paraclinical results

Test	Report summary
Right breast gland biopsy and IHC	Histological Type: Invasive carcinoma of unspecified type (invasive ductal carcinoma without further specification). Estrogen Receptors: Positive -Percentage of Positive Cells: 100% -Intensity: Moderate -External Control: Present Reactivity: Present Progesterone Receptors: Negative -External Control: Present -Reactivity: Present Her2: (2+) -Percentage of Cells with Moderate Positivity: 85% Cell Proliferation Index (Ki-67): 23% P63 and Heavy Chain Myosin: Negative
EMG – limb study	Motor and sensory nerve conduction within normal ranges. Needle electrode study: no abnormalities. The right upper limb muscles were not evaluated due to prior lymph node dissection.
CSF analysis	Leukocytes: 0.0 / mm³ (neutrophils 0%, lymphocytes 0%, monocytes 0%). Red blood cells: 0.0 / mm³; morphology: normomorphic. India ink: negative. Gram stain: no microorganisms observed. Glucose: 57 mg/dL; protein: 49.1 mg/dL; LDH: 23 U/L. Culture: negative after 72 hours. Overall: normal.
Non-treponemal VDRL (serum)	Non-reactive.
Chloride	101.8 mEq/L (normal)
Potassium	4.27 mEq/L (normal)
Sodium	141.0 mEq/L (normal)
Creatinine	0.78 mg/dL (normal)
Hemogram	Leukocytes 7490 Neutrophiles: 4120 Lymphocytes. 1930 Hemoglobin: 14.6 Plaquettes 278.000 Normal
Folic acid (folate)	21.2 ng/dl (abnormal, elevated, nonspecific)
Vitamin B12	630 pg/ml (normal)
Anti-neuronal autoantibody profile	Anti-Hu Negative Anti-Yo: Negative *Anti Ri - Positive Anti Amphiphysin: Negative Anti-CV2: Negative Anti-Ma2: Negative
Cervical spine MRI (with and without contrast)	No evidence of tumor or compressive lesions at the cervicomedullary junction.
Thoracic spine MRI (with and without contrast)	No evidence of tumor or compressive lesions at the cervicomedullary junction.
Brain MRI (with and without contrast)	No demonstrable tumor lesions.

Abbreviations: IHC: immunohistochemistry; EMG: electromyography; CSF: cerebrospinal fluid; MRI: magnetic resonance imaging.

Literature review

Autoimmune phenomena in cancer can trigger both humoral and cellular immune responses. In humoral immunity, plasma cells produce anti-neuronal autoantibodies that target cell-surface antigens, whereas cellular immunity primarily targets intracellular antigens. These mechanisms form the pathophysiological basis of paraneoplastic neurological syndromes (PNS). Certain tumors may generate specific antibodies that not only facilitate tumor localization but may also help assess the clinical response to targeted oncological treatment (1,2).

PNS are reported in cancer patients at a low incidence, with reviewed data indicating a frequency of approximately 0.01%.

Clinically, these syndromes often manifest as a monophasic condition, characterized by temporary stability followed by progressive deterioration without relapses. The response to available treatments, such as corticosteroids, plasmapheresis, intravenous immunoglobulin, and immunosuppressants, is generally limited (2).

The diagnosis of PNS is based on the following criteria (1,2):

1. Evidence of the central or peripheral nervous system involvement, which may present a variety of clinical manifestations without a distinct topographic pattern.
2. Establishment of a causal relationship between the neurological syndrome and an underlying malignancy.
3. Identification of an immune-mediated cause, as demonstrated by the presence of anti-neuronal antibodies.

Anti-Ri paraneoplastic neurological syndrome occurs predominantly in women (2), although sporadic cases have been reported in men (3). One documented case described infiltrating ductal breast carcinoma with an atypical neurological presentation characterized by complete horizontal ophthalmoplegia, sensory symptoms due to involvement of the left trigeminal nerve, and truncal ataxia. Brain MRI demonstrated a hyperintense lesion in the pontine tegmentum without gadolinium enhancement (3).

PNS have been associated with various malignancies, including breast cancer, small-cell lung carcinoma, neuroblastoma, gastric adenocarcinoma, renal cell carcinoma, and cervical cancer (2-12). Regarding the tumor subtype that triggers PNS, immunohistochemical profiling has not demonstrated a consistent correlation between tumor markers and the specific neurological phenotype. For instance, HER2-positive breast cancer has been more frequently associated with anti-Yo antibodies and paraneoplastic cerebellar degeneration, followed by opsoclonus-myoclonus syndrome and the presence of anti-Ri antibodies (5).

The humoral autoimmune origin explains the pathophysiology of opsoclonus-myoclonus syndrome, in which the fastigial nucleus of the cerebellum is the primary target of antibody-mediated attack, leading to the GABAergic inhibition of omnipause neurons that control saccadic eye movements, resulting in opsoclonus (12).

The pathological mechanism in PNS is associated with anti-Ri antibodies that target NOVA1 and NOVA2 antigens, which are widely expressed throughout the nervous system (4,5). Reports indicate that up to 70% of patients with breast cancer and opsoclonus-myoclonus syndrome express anti-Ri (ANNA-2) antibodies (12).

Clinically, the syndrome presents with variable multisystem dysfunction and a subacute or chronically progressive course. There may be periods of symptom resolution, remission, and exacerbation, which can resemble non-paraneoplastic conditions such as neurodegenerative or inflammatory disorders. This overlap frequently delays diagnosis and obscures the underlying neoplastic etiology (4-6).

The neurological presentation of this condition is characterized by opsoclonus-myoclonus signs, downbeat nystagmus, and ataxia (10). However, other distinct clinical syndromes have also been reported, including cases of autoimmune encephalitis without evidence of neoplasia, in which multiple autoantibodies are expressed, such as anti-Ri, anti-LGI1, anti-AMPAR2, and anti-CENP-A/B (2-4, 11).

Opsoclonus refers to brief, involuntary, rapid eye movements that occur conjugately, while myoclonus involves involuntary muscle contractions that can be focal or multifocal throughout the body (3-6). It should be noted that opsoclonus is not exclusively a paraneoplastic manifestation; it may also occur in association with other neurological conditions, such as viral infections, hyperosmolar syndromes, and metabolic abnormalities (6, 13).

The combination of opsoclonus-myoclonus and ataxia has also been reported in association with other autoantibodies, including anti-Yo and anti-Hu, indicating that this clinical presentation is not exclusive to anti-Ri antibodies (5, 13).

Magnetic resonance imaging (MRI) is the preferred method for evaluating paraneoplastic neurological syndromes. However, published reports indicate that only about half of patients with this syndrome show abnormalities on MRI. These abnormalities are typically found in areas such as the insular cortex, pons, or cerebral white matter (1-3, 6). Moreover, in patients positive for anti-Ri antibodies, brain MRI findings are frequently normal (3).

In the field of nuclear medicine, positron emission tomography using fluorodeoxyglucose (FDG-PET) is recommended for whole-body screening to identify hidden neoplasms (11).

Lumbar puncture, along with cerebrospinal fluid (CSF) analysis, often shows abnormalities in most patients, including elevated immunoglobulin levels, an increased leukocyte count, and higher protein concentrations (2-6).

The detection of anti-neuronal Ri antibodies is conducted on serum samples using immunoblot assays with recombinant antigens and immunohistochemistry (ELISA). This antibody is valuable not only for diagnostic purposes but also serves as a marker of treatment response and disease stability. Additionally, the monitoring and quantification of circulating autoantibodies can help assess the potential for tumor reactivation and the progression of PNS (7).

The treatment approach for PNS primarily focuses on tumor resection. Multidisciplinary cancer teams adhere to evidence-based guidelines regarding surgical indications, which often exclude surgery for patients with paraneoplastic involvement. However, in the presence of PNS, tumor removal is essential to reduce the autoimmune response driven by circulating autoantibodies. In such cases, neoadjuvant chemotherapy should be avoided, particularly in patients with localized

breast cancer who would otherwise undergo such treatment prior to resection (8,9).

Following tumor removal, specific adjunctive treatment includes immunosuppressive chemotherapy. Therapeutic options for PNS comprise immunosuppressive agents such as cyclophosphamide, corticosteroids (e.g., methylprednisolone 1 g/day intravenously for 5 days), and immunomodulatory therapies such as plasmapheresis and intravenous immunoglobulin IgG (400 mg/kg/day for 5 days) [2,3,5,8-11].

It is typically administered in 2 intravenous infusions of 1 g each, separated by 15 days, in combination with standard regimens (11).

RESULTS AND DISCUSSION

PNS include paraneoplastic cerebellar degeneration, opsoclonus-myoclonus syndrome, stiff-person syndrome, paraneoplastic neuropathy, and paraneoplastic encephalomyelitis. These syndromes are characterized by distinct antibody profiles, including anti-Hu, anti-Yo, anti-CV2, anti-Ma2, anti-amphiphysin, and anti-Ri (4). Among these, anti-Ri antibodies are most frequently associated with paraneoplastic cerebellar degeneration and opsoclonus-myoclonus syndrome.

The present case of anti-Ri-positive PNS is notable for its predominance in female patients and its association with HER2-positive ductal breast carcinoma, one of the tumor types most often linked to this antibody. The patient presented with nystagmus and gait disturbances and demonstrated meaningful improvement after immunomodulatory therapy, regaining functional independence with the aid of a walker.

The clinical course was characterized by alternating periods of stability and relapse, an uncommon pattern infrequently reported in the literature that requires repeated cycles of immunomodulatory treatment to maintain functional stability. Although serum and neuroimaging studies were inconclusive, the diagnosis was established based on positive serum titers of anti-Ri antibodies, consistent with prior findings.

A noteworthy aspect of this case is the atypical presentation, which is not commonly reported as a classical manifestation of anti-Ri syndrome. The patient exhibited severe spastic quadriparesis, clonus, and rigidity, resulting in significant functional limitations. The combination of extrapyramidal and pyramidal symptoms left her unable to turn in bed and rendered her entirely dependent on family members for self-care. Although this clinical phenotype has been observed in patients with stiff-person syndrome, this diagnosis was ruled out due to negative anti-amphiphysin antibody results. An atypical feature of this case was the presentation with severe spastic quadriparesis, clonus, and rigidity, leading to profound functional impairment. The coexistence of extrapyramidal and pyramidal signs left the patient unable to turn in bed and entirely dependent on caregivers for basic activities. Although such a phenotype has been observed in patients with stiff-person syndrome, this diagnosis was excluded by negative anti-amphiphysin antibody testing.

Table 3 summarizes previously reported cases of anti-Ri–associated syndromes with atypical clinical features, compiled from multiple research groups.

Table 3. Published cases of anti-Ri-associated syndromes

Case study	Journal/date	Underlying malignancy
Pearls & Oysters: gait instability, jaw dystonia, and horizontal diplopia in a woman with anti-Ri antibodies and breast cancer. Kim H. et al.	Neurology 2022	Breast cancer
Anti-Ri-associated paraneoplastic ophthalmoplegia ataxia syndrome in a woman with breast cancer: a case report and review of the literature. Sena G. et al.	J Med Case Rep. 2020	Breast cancer
Anti-Ri-associated paraneoplastic neurological syndrome: initial symptom of breast cancer with HER2 overexpression and treatment by dual HER2 blockade. Olmez O. F. et al.	J Oncol Pharm Pract. 2019	Breast cancer
Anti-Ri antibody-associated small cell lung carcinoma. O'Leary CG. et al.	Ir J Med Sci. 2017	SCLC
Anti-Ri-associated paraneoplastic cerebellar degeneration. Report of a case and revision of the literature. Mancuso M. et al.	Arch Ital Biol 2011 SCLC	SCLC
Paraneoplastic jaw dystonia and laryngospasm with antineuronal nuclear autoantibody type 2 (anti-Ri). Pittock SJ. et al.	Arch Neurol. 2010	Breast cancer, sweat gland angiosarcoma, uterine cervical carcinoma
Anti-Ri antibody-associated paraneoplastic syndrome in a man with breast cancer showing a reversible pontine lesion on MRI. Kim H. et al.	J Clin Neurol. 2009	Breast cancer

Abbreviations: SCLC: Small-cell lung carcinoma.

This clinical case is notable due to the onset of PNS, which occurred seven years after the patient was initially diagnosed with breast cancer. Remarkably, the patient survived more than two years with a favorable functional outcome. This highlights the importance of early recognition of PNS, as prompt intervention can substantially improve functional status.

PNS may indicate the presence of an undiagnosed malignancy, which emphasizes its diagnostic value. Therefore, optimal management requires a dual therapeutic strategy that addresses both the underlying tumor and the paraneoplastic manifestations to maximize patient outcomes.

Limitations

This is a single case report with a narrative review; potential selection bias in the literature cannot be excluded.

CONCLUSIONS

Anti-Ri–associated paraneoplastic neurological syndromes (PNS) are typically characterized by ocular symptoms and signs, often accompanied by ataxia. However, it is crucial to consider the atypical variants that have been documented.

These syndromes are associated with a range of malignancies that should be considered in the diagnostic process, including but not limited to breast cancer, small-cell lung carcinoma, neuroblastoma, gastric adenocarcinoma, renal cell carcinoma, and cervical cancer. The presence of autoantibodies is crucial to guiding the search for the underlying tumor and to monitoring treatment response and disease course.

Surgical removal of the neoplasm is essential for the subsequent management of the PNS, complemented by immunosuppressive and immunomodulatory therapies. Multidisciplinary care is essential to address both oncologic and neurologic aspects. Although therapeutic success is often limited, timely recognition and intervention can improve functional outcomes, enhance quality of life, and, in some cases, prolong survival.

Acknowledgments

The authors thank Dr. Jorge Karim Assis, Director of the Department of Research and Clinical Education at Clínica de Occidente S.A., and his colleagues for providing access to the institutional medical records system necessary for this study.

REFERENCES

1. Braczkowski M, Soszyński D, Sierakowska A, Braczkowski R, Kufel K, Łabuz-Roszak B. Autoimmune encephalitis with antibodies: Anti-NMDAR, anti-AMPAR, anti-GQ1b, anti-DPPX, anti-CASPR2, anti-LGI1, anti-RI, anti-yo, anti-Hu, anti-CV2 and anti-GABAAR, in the course of psychoses, neoplastic diseases, and paraneoplastic syndromes. Diagnostics. 2023; 13(15): 2589. http://dx.doi.org/10.3390/diagnostics13152589

2. Freydl E, Tinchon A, Blauensteiner K, Oberndorfer S. Anti-Ri paraneoplastic neurological syndrome presenting with bilateral cranial nerve VI palsy and jaw dystonia-a distinctive syndrome within the anti-Ri spectrum? : Case report and literature review. Wien Med Wochenschr. 2024; 174(1–2): 16–21. http://dx.doi.org/10.1007/s10354-023-01006-8

3. Kim H, Lim Y, Kim K-K. Anti-ri-antibody-associated paraneoplastic syndrome in a man with breast cancer showing a reversible pontine lesion on MRI. J Clin Neurol. 2009; 5(3): 151–2. http://dx.doi.org/10.3988/jcn.2009.5.3.151

4. Sena G, Gallo G, Vescio G, Gambardella D, de Franciscis S, Renne M. Anti-Ri-associated paraneoplastic ophthalmoplegia-ataxia syndrome in a woman with breast cancer: a case report and review of the literature. J Med Case Rep. 2020; 14(1): 67. http://dx.doi.org/10.1186/s13256-020-02410-z

5. Olmez OF, Kinikoglu O, Yilmaz NH, Bilici A, Cubukcu E, Seker M, et al. Anti-Ri-associated paraneoplastic neurological syndrome: Initial symptom of breast cancer with HER2 overexpression and treatment by dual HER2 blockade. J Oncol Pharm Pract. 2019; 25(6): 1526–30. http://dx.doi.org/10.1177/1078155218792672.

6. Luque FA, Furneaux HM, Ferziger R, Rosenblum MK, Wray SH, Schold SC Jr, et al. Anti-Ri: an antibody associated with paraneoplastic opsoclonus and breast cancer. Ann Neurol. 1991; 29(3): 241–51. http://dx.doi.org/10.1002/ana.410290303.

7. Stich O, Rasiah C, Rauer S. Paraneoplastic antibody during follow-up of a patient with anti-Ri-associated paraneoplastic neurological syndrome. Acta Neurol Scand. 2009; 119(5): 338–40. http://dx.doi.org/10.1111/j.1600-0404.2008.01102.x.

8. Graber JJ. Paraneoplastic neurologic syndromes. Continuum (Minneap Minn). 2023; 29(6): 1779–808. http://dx.doi.org/10.1212/CON.0000000000001357.

9. Trujillo F, Medina YF, González PJP, Vega CZ, Gómez GM. The neurologies paraneoplasics syndromes to understand the immune response and paraneoplastic antibodies. Acta Neurol Colomb. 2011; 27(4): 237–42. https://www.actaneurologica.com/index.php/anc/article/view/1464.

10. Kim H-J, Kim H-J, Lee J-H, Kim J-S. Pearls & oy-sters: Vibration-induced downbeat nystagmus: A new cerebellar sign observed in paraneoplastic syndrome. Neurology. 2023; 100(1): 43–6. http://dx.doi.org/10.1212/WNL.0000000000201466.

11. Asbeutah S, Alhashime K, Alhamer M. A case report and literature review on positivity for multiple antibodies: Autoimmune encephalitis in focus. Cureus. 2023; 15(12): e50393. http://dx.doi.org/10.7759/cureus.50393.

12. Del Valle Pérez JA, Redondo Penas, Romero A, Segura T. Paraneoplastic opsoclous-myoclous syndrome in a patient with esophageal adenocarcinoma. Neurology. 2020; 35(9): 677–8.

13. Didelot A, Honnorat J. Les syndromes neurologiques paranéoplasiques. Rev Med Interne. 2011; 32(10): 605–11. http://dx.doi.org/10.1016/j.revmed.2010.09.007.