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IMAGES IN CLINICAL NEUROLOGY

From oncology to neurology: A decade-long journey to paraneoplastic cerebellar degeneration

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Paraneoplastic cerebellar degeneration (PCD) is a rare autoimmune disorder characterized by progressive ataxia due to the destruction of cerebellar Purkinje cells.[1] It is a nonmetastatic systemic complication of cancer. In addition to ataxia, it may cause symptoms such as vertigo, nausea, and nystagmus.[2,3]

Symptoms may appear before, simultaneously with, or during the follow-up after a cancer diagnosis. Although most commonly associated with breast, gynecological, and small cell lung cancers, PCD can occur with all types of malignancies.[4] Antibodies generated against tumor antigens may cross-react with Purkinje cells and other cerebellar neurons, leading to the manifestation of symptoms.[5] Herein, we presented a female patient diagnosed with PCD following the diagnosis and treatment of breast cancer, accompanied by progressive cerebellar degeneration and ataxia. Magnetic resonance imaging (MRI) findings were discussed in light of current literature.

A 59-year-old female patient who underwent segmental mastectomy and axillary lymph node dissection due to Grade 3 intraductal carcinoma in two foci in the right breast (ER+, PR+, HER2–) was admitted to the neurology clinic. Pathological staging revealed pT1N0M0, corresponding to Stage 1. Adjuvant chemotherapy was not administered, as the patient received surgery, postoperative radiotherapy, and hormonal therapy. The neurological history was notable only for a meningioma and a millimetric arteriovenous malformation. No recurrence or systemic metastasis was detected during long-term follow-up. Written informed consent was obtained from the patient.

No neurological findings were observed in the early postoperative period; however, in the fourth postoperative year, the patient developed a headache and ataxia. These neurological symptoms did not progress significantly.

Postoperative and one-year follow-up MRI for meningioma showed no pathological findings other than a stable residual meningioma (Figure 1a). Follow-up MRI at three, five, and 10 years (Figures 1b, c and d) revealed bilateral, slowly progressive, asymmetric FLAIR (fluid-attenuated inversion recovery) hyperintensities in the cerebellar vermis without significant diffusion restriction. Perfusion MRI demonstrated hypoperfusion (Figure 1e), while magnetic resonance spectroscopy showed no choline peak (Figure 1f). Notably, FLAIR hyperintense changes in the cerebellar vermis were observed in the third postoperative year, preceding the onset of clinical symptoms, which occurred in the fourth year. Upon the detection of abnormal MRI findings, differential diagnoses, including demyelinating diseases, autoimmune conditions (e.g., SLE and Behçet's disease), atypical CNS infections, and metabolic disorders (e.g., Wernicke's encephalopathy, B12 deficiency, and celiac disease), were considered. All relevant investigations yielded negative results. The patient was diagnosed with PCD and placed

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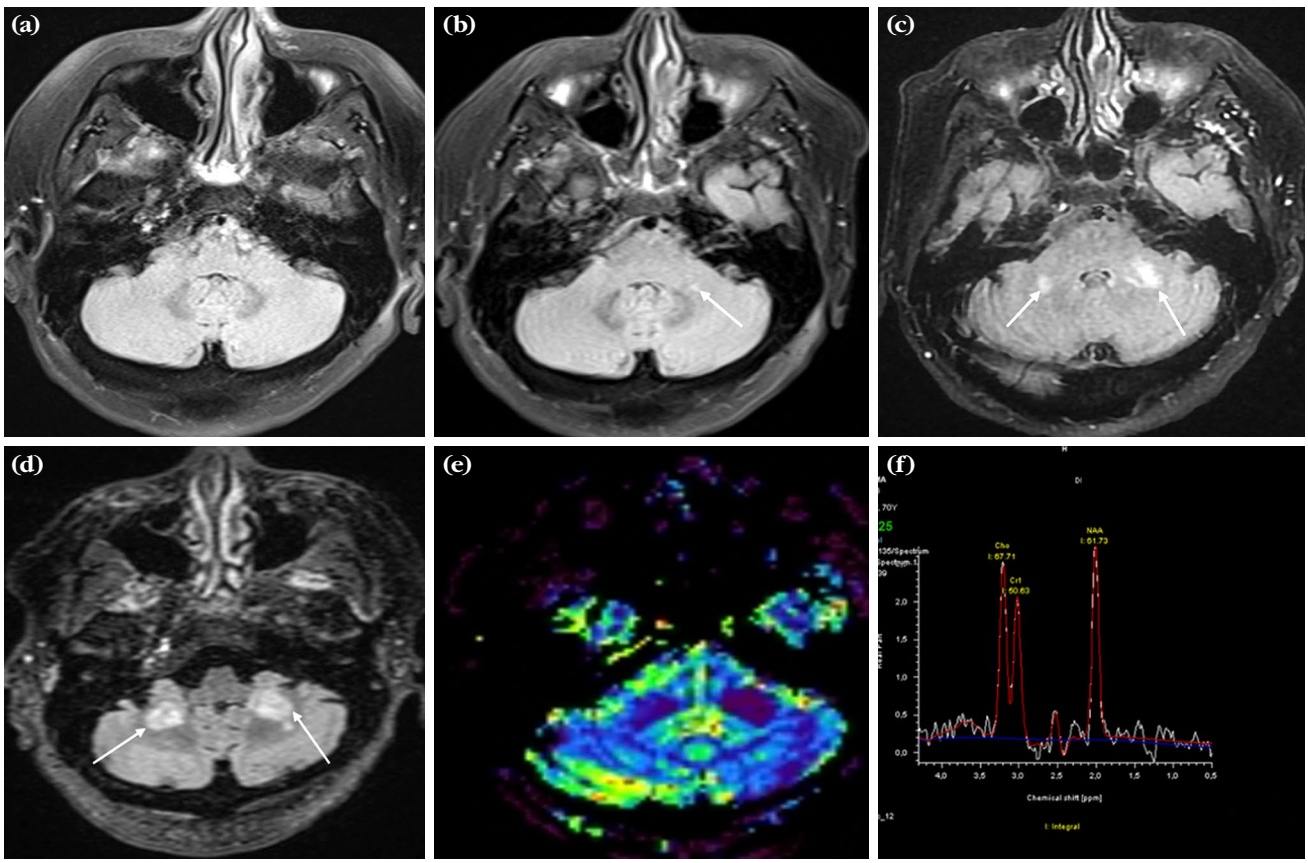

Figure 1. **(a)** No pathological findings in the cerebellar vermis in the first postoperative year. Follow-up MRIs at **(b)** three, **(c)** five, and **(d)** 10 years reveal slowly progressive, bilateral FLAIR hyperintensities in the cerebellar vermis. **(e)** Hypoperfused areas on perfusion MRI. **(f)** Magnetic resonance spectroscopy demonstrates no significant choline peak.
MRI, magnetic resonance imaging; FLAIR: Fluid-attenuated inversion recovery.

under regular MRI surveillance. Paraneoplastic antibody testing was not performed in this patient, which limited diagnostic certainty. No immunomodulatory therapies (e.g., corticosteroids, intravenous immunoglobulin, or plasmapheresis) were administered, as the patient's neurological symptoms remained relatively stable and did not warrant such interventions.

Paraneoplastic cerebellar degeneration occurs in approximately 1 to 3% of cancer patients.[6] Diagnosis is often challenging, and the syndrome typically responds poorly to treatment. It is more common in middle-aged female patients. The present case involved a middle-aged female patient with breast cancer, placing her in a high-risk group for PCD.

Ataxia is the most common presenting symptom.[7,8] Some patients may test positive for anti-Yo antibodies, although about 40% are seronegative. Diagnosis is therefore largely clinical and supported by radiological findings. There is no significant prognostic difference between seropositive and seronegative patients.[9]

Imaging findings in PCD are known to evolve with disease stage. In the acute or subacute phase, increased T2/FLAIR signal changes may predominate in the cerebellar hemispheres, whereas in the chronic phase, these hyperintensities often decrease and are replaced by cerebellar atrophy.[10,11] In our case, serial MRI demonstrated slowly progressive FLAIR hyperintensities localized to the cerebellar vermis over several years, without marked hemispheric involvement. This pattern is more consistent with the chronic-phase spectrum of PCD, in which persistent but evolving signal abnormalities and gradual atrophic changes may be observed. Magnetic resonance imaging is also useful for excluding other metabolic, structural, or demyelinating processes. In some cases, neurological symptoms precede cancer diagnosis,

and positron emission tomography-computed tomography may be used to identify the primary malignancy.

In our case, MRI abnormalities preceded the clinical manifestation of ataxia by approximately one year, suggesting the presence of a subclinical phase of PCD. Such subclinical radiological changes was described in a previous report and may highlight the potential role of neuroimaging in detecting early disease activity before overt clinical deterioration.[9]

According to the recently updated Paraneoplastic Neurologic Syndrome (PNS)-Care criteria[12] our case reached a PNS-Care score of 6, fulfilling the category of a probable PNS rather than definite PNS, due to the absence of antibody testing and tumor antigen expression studies. This limitation has been explicitly acknowledged.

Diagnosis of PCD is often difficult and time-consuming. Treatment options are limited, and responses are generally poor. Although immunomodulatory treatments, including corticosteroids, IVIG, or plasmapheresis, have been attempted in PCD, clinical responses are generally poor.[1] In our case, such therapies were not initiated due to the relatively stable course and the limited evidence supporting their efficacy.

In oncology patients with neurological symptoms, particularly ataxia, PCD should be considered. If suspected, close clinical and radiological follow-up is essential, and the progressive nature and poor prognosis of the disease must be anticipated.

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REFERENCES

1. Greenlee JE. Treatment of paraneoplastic cerebellar degeneration. Curr Treat Options Neurol 2013;15:185-200. doi: 10.1007/s11940-012-0215-4.
2. Henson RA, Urich H. Cortical cerebellar degeneration. In: Henson RA, Urich H, editors. Cancer and the nervous system: the neurological manifestations of systemic malignant disease. Oxford: Blackwell Scientific Publications; 1982. p. 346-67.
3. Brain WR, Daniel PM, Greenfield JG. Subacute cortical cerebellar degeneration and its relation to carcinoma. J Neurol Neurosurg Psychiatry 1951;14:59-75. doi: 10.1136/jnnp.14.2.59.
4. Peterson K, Rosenblum MK, Kotanides H, Posner JB. Paraneoplastic cerebellar degeneration. I. A clinical analysis of 55 anti-Yo antibody-positive patients. Neurology 1992;42:1931-7. doi: 10.1212/wnl.42.10.1931.
5. Loehrer PA, Zieger L, Simon OJ. Update on paraneoplastic cerebellar degeneration. Brain Sci 2021;11:1414. doi: 10.3390/brainsci11111414.
6. Lorusso L, Hart IK, Giometto B, Pezzani R, Broome JC, Gritti D, et al. Immunological features of neurological paraneoplastic syndromes. Int J Immunopathol Pharmacol 2004;17:135-44. doi: 10.1177/039463200401700205.
7. Ducray F, Demarquay G, Graus F, Decullier E, Antoine JC, Giometto B, et al. Seronegative paraneoplastic cerebellar degeneration: The PNS Euronetwork experience. Eur J Neurol 2014;21:731-5. doi: 10.1111/ene.12368.
8. Yağcı M, Bir S, Haznedar R. Multiple myeloma and paraneoplastic cerebellar degeneration. Turk J Neurol 1999;5:68-9.
9. Masangkay N, Basu S, Moghbel M, Kwee T, Alavi A. Brain 18F-FDG-PET characteristics in patients with paraneoplastic neurological syndrome and its correlation with clinical and MRI findings. Nucl Med Commun 2014;35:1038-46. doi: 10.1097/MNM.0000000000000163.
10. Fu JB, Raj VS, Asher A, Lee J, Guo Y, Konzen BS, et al. Inpatient rehabilitation performance of patients with paraneoplastic cerebellar degeneration. Arch Phys Med Rehabil 2014;95:2496-9. doi: 10.1016/j.apmr.2014.07.003.
11. İlhan Y, Çagla Korkmaz M, Taşkıran Güzel E, Mammadova K, Sezgin Göks S, Tatlı AM, et al. Anti-yo associated paraneoplastic cerebellar degeneration in ovarian cancer: A rare case report. J Oncol Sci 2021;7:31-3.
12. Graus F, Vogrig A, Muñiz-Castrillo S, Antoine JG, Desestret V, Dubey D, et al. Updated diagnostic criteria for paraneoplastic neurologic syndromes. Neurol Neuroimmunol Neuroinflamm 2021;8:e1014. doi: 10.1212/NXI.0000000000001014.