Case Report

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Radiology Section

Leptomeningeal Carcinomatosis with Brain and Spinal Metastasis from Triple-negative Breast Cancer: A Case Report

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ABSTRACT

Leptomeningeal Carcinomatosis (LMC) is an uncommon but devastating complication of metastatic breast cancer, particularly associated with Triple-Negative Breast Cancer (TNBC). The authors report a 46-year-old female with previously treated TNBC and a parenchymal brain metastasis who presented with subacute, progressive bilateral lower-limb weakness and gait impairment. Contrast-Enhanced Magnetic Resonance Imaging (CE-MRI) of the brain and spine demonstrated diffuse leptomeningeal enhancement over the cerebellar hemispheres, spinal cord, and cauda equina, along with post-radiotherapy white-matter changes and hydrocephalus. Cerebrospinal Fluid (CSF) cytology confirmed the presence of malignant cells, establishing the diagnosis of LMC. Given her poor performance status and extensive intracranial and spinal disease burden, she was managed with palliative intent, including corticosteroids, analgesics, antiemetics, and best supportive care, but experienced progressive neurological decline. The present case highlights the close temporal relationship between brain metastases and LMC in TNBC, underscores the diagnostic value of CE-MRI with post-contrast Fluid Attenuated Inversion Recovery (FLAIR) and Three-Dimensional (3D) T1-weighted sequences combined with CSF cytology, and emphasises the need for heightened clinical suspicion and optimised palliative strategies in patients with advanced TNBC who develop new neurological symptoms.

Keywords: Breast neoplasms, Cerebrospinal fluid, Hydrocephalus, Magnetic resonance imaging

CASE REPORT

A 46-year-old female presented with progressive weakness of both lower limbs over the past six weeks and was unable to walk without support at the time of presentation. There was no history of seizures, loss of consciousness, or recent head trauma. The patient had no history of breast, ovarian, or other malignancies in first-degree relatives at an early age; however, no genetic testing was performed. The patient had no history of addiction.

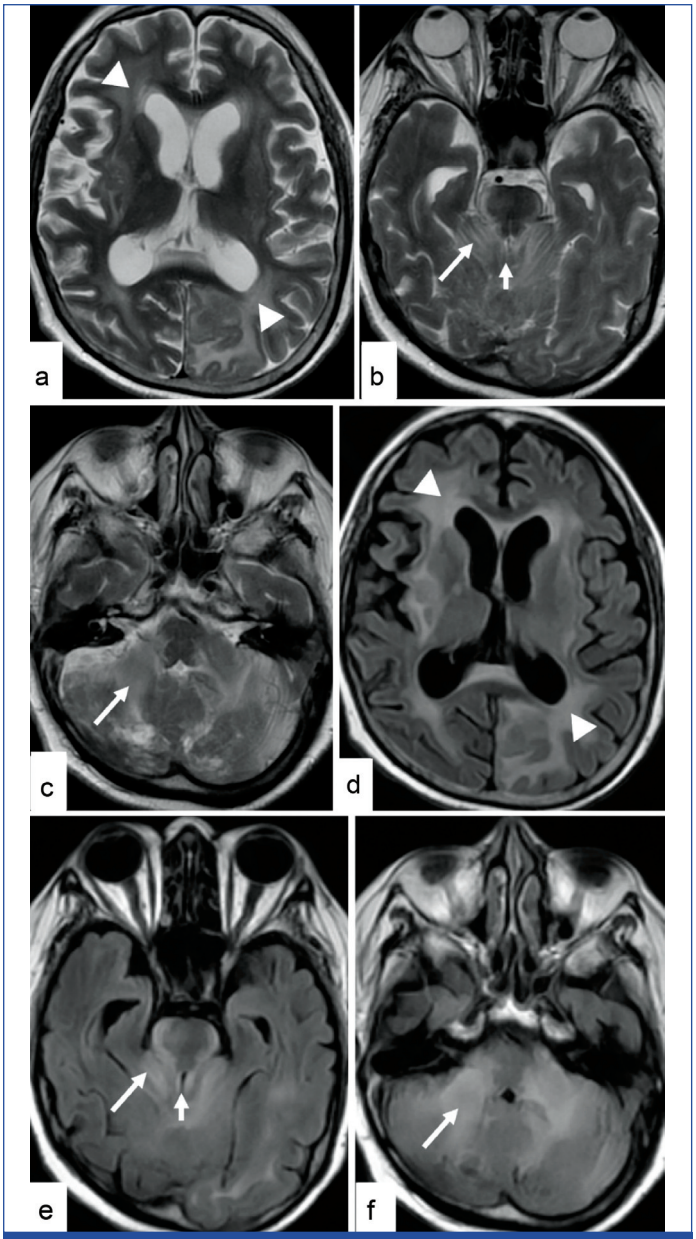
On clinical examination, the patient was conscious, oriented, and following verbal commands. There was generalised weakness, more pronounced in the lower limbs, with impaired gait requiring assistance. On physical examination, symmetric proximal and distal weakness was noted in both lower limbs, Medical Research Council grade 3/5, with preserved upper-limb strength. Deep tendon reflexes were brisk in the lower limbs, with preserved or slightly increased knee and ankle jerks. Plantar responses were extensor. Cerebellar testing revealed impaired coordination. Her functional status at presentation corresponded to an Eastern Cooperative Oncology Group performance status of 3, consistent with progressive difficulty ambulating and inability to walk without support [1]. Cranial nerve examination was normal.

The patient's oncological history was significant for advanced TNBC with documented parenchymal brain metastases (Tumour 3, Node 2 and Metastasis 1 (T3N2M1) at initial diagnosis) four years ago. Histopathology with immunohistochemistry showed Estrogen Receptor (ER) negativity (<1%), Progesterone Receptor (PR) negativity (<1%), Human Epidermal Growth Factor Receptor 2 (HER2) negativity (score 0/1+), with a Ki-67 proliferation index of 40%, consistent with TNBC. The patient received eight cycles of Neoadjuvant Chemotherapy (NACT) combining doxorubicin, cyclophosphamide, paclitaxel, and carboplatin, followed by a right modified radical mastectomy. Following surgery, the patient received adjuvant Radiotherapy (RT) to the chest wall at 26 Gy in five fractions, Whole-Brain Radiotherapy (WBRT) to a total dose of 20 Gy in 10 fractions, and eight cycles of capecitabine as systemic therapy for brain metastasis. Records of prior brain imaging documenting metastasis were unavailable to the patient. A timeline of treatment and imaging findings is provided in [Table/Fig-1].

Date/Year	Event	Details
Dec 2022	Completion of neoadjuvant chemotherapy	Eight cycles of NACT (doxorubicin, cyclophosphamide, paclitaxel, and carboplatin) completed; immunohistochemistry confirmed TNBC, grade III
Jan 2023	Surgery	Right modified radical mastectomy
Jul 2023-Jan 2024	Adjuvant radiotherapy	26 Gy in 5 fractions to the chest wall
Feb 2025	MRI	MRI report showed a brain lesion consistent with parenchymal metastasis
Feb 2025	WBRT	Whole Brain Radiotherapy (WBRT) 20 Gy in 10 fractions for brain metastasis
Apr 2025	Post-WBRT Chemotherapy	Eight cycles of capecitabine as systemic therapy for brain metastasis.
Late 2025	Presentation with LMC	Developed a 6 week history of progressive bilateral lower limb weakness and inability to walk without support
		CE-MRI brain and spine revealed disseminated brain and spinal metastases with Leptomeningeal Carcinomatosis (LMC)

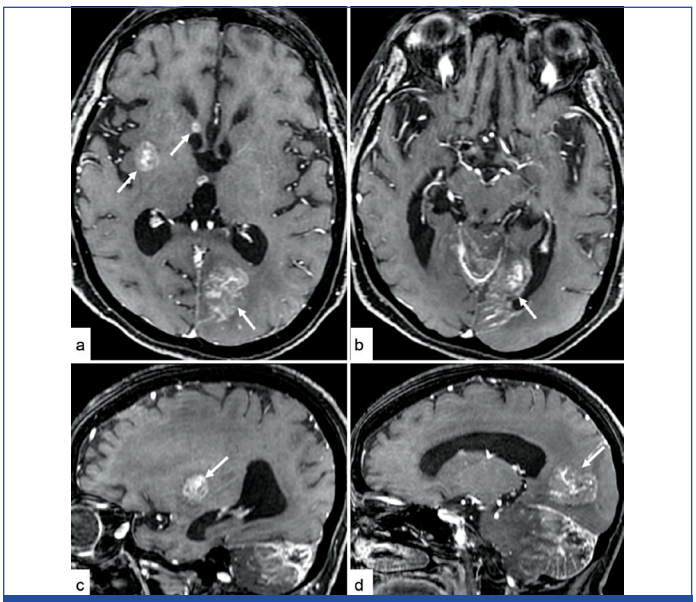
[Table/Fig-1]: Summary of key events.

To evaluate the cause of her progressive neurological decline, a CE-MRI of the brain and spine was performed. Imaging revealed confluent areas of T2- and FLAIR-hyperintense signal abnormality in the white matter of the cerebrum and cerebellum, consistent with post-RT changes, causing effacement of the fourth ventricle and upstream hydrocephalus [Table/Fig-2]. In the post-contrast sequence, there was a cerebral parenchymal metastasis and

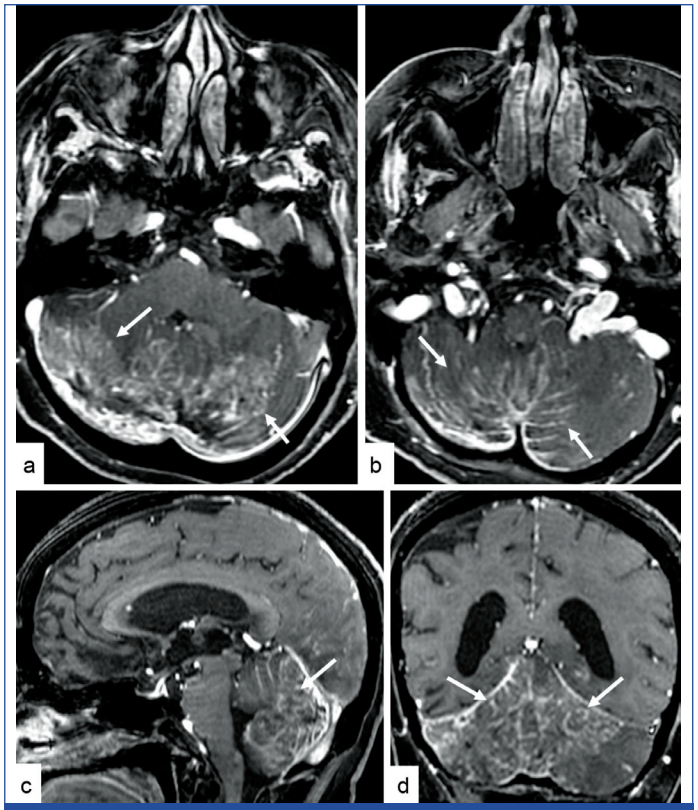


[Table/Fig-2]: (a-c) MRI brain axial T2 and (d-f) FLAIR images showing confluent hyperintense signal changes in the brain parenchyma (white arrows and arrowheads), causing partial effacement of the fourth ventricle (small white arrow).

diffuse leptomeningeal enhancement in the cerebellar hemispheres, representing LMC [Table/Fig-3,4].



[Table/Fig-3]: (a,b) MRI brain postcontrast axial and (c,d) sagittal images showing enhancing lesions in the cerebral hemispheres (white arrows).



[Table/Fig-4]: (a,b) MRI Brain postcontrast axial and (c,d) sagittal images showing diffuse leptomeningeal enhancement in cerebellum and vermis (white arrows).

Imaging of the spine also showed enhancing lesions along the spinal cord surface and the cauda equina nerve roots, indicating dissemination into the CSF [Table/Fig-5]. Spinal metastases also correlated with the patient's lower-limb weakness. CSF analysis showed malignant cells on cytology, supporting the diagnosis of LMC and correlating with the imaging findings. Based on the imaging findings, CSF analysis, and clinical background, a diagnosis of disseminated cerebral metastases with associated LMC secondary to TNBC was made. In this patient, brain metastases developed approximately two years after the initial breast cancer surgery and completion of neoadjuvant chemotherapy. In contrast, LMC occurred approximately eight to ten months after the first radiologic detection of brain metastasis.

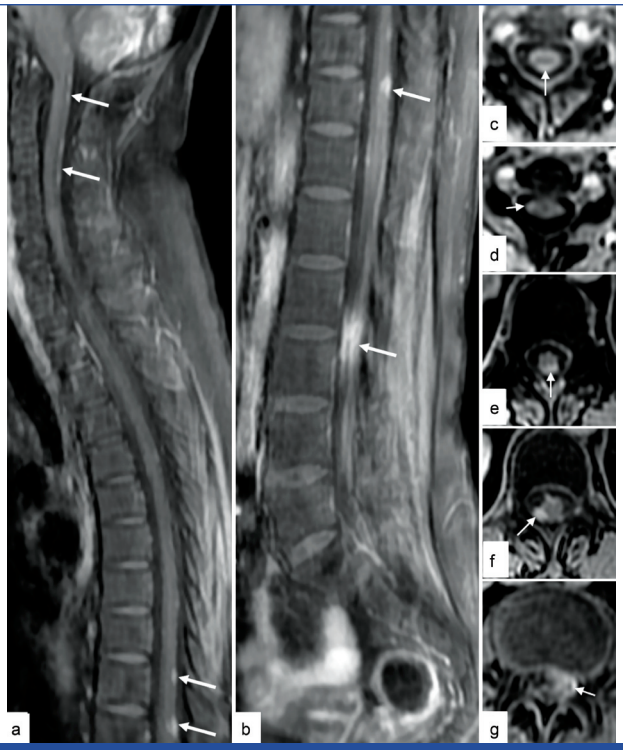
Given her poor performance status and extensive disease burden, treatment was palliative, with supportive care to maintain quality of life. After the diagnosis of LMC, the patient was managed with palliative intent, including corticosteroids, opioids, adjuvant analgesics, antiemetics, and best supportive care. Despite transient symptomatic relief, she experienced progressive neurological decline, with worsening lower-limb weakness, increasing dependency in activities of daily living, and gradual deterioration in performance status. Subsequently, the patient was lost to follow-up and had no further hospital visits.

DISCUSSION

TNBC is associated with early relapse, a high proliferative index, and an increased risk of Central Nervous System (CNS) dissemination [2]. Although breast cancer is the most common non-haematologic tumour with leptomeningeal spread, the incidence remains low at only 1% to 5% [3]. LMC is a rare but devastating late manifestation of metastatic breast disease, with an incidence ranging from 0.8% to 6.6% in clinical reports and up to 16% in autopsy series [4].

In a retrospective analysis of 45 patients with TNBC and Leptomeningeal Metastasis (LM), LM was diagnosed by CSF cytology in 41 patients and by MRI in 24 patients [5].

The patient in the present case developed progressive weakness and difficulty walking, symptoms consistent with spinal and leptomeningeal involvement. A case by Satani N et al., describes that a woman in her early 50s with breast cancer underwent



[Table/Fig-5]: (a,b) MRI spine postcontrast sagittal and (c-g) axial images showing nodular enhancement over the surface of the entire spinal cord as well as nerve roots (thick and thin white arrows). CSF analysis revealed malignant cells.

breast-conserving surgery and radiotherapy. Three years later, she developed lung and bone metastases and received bevacizumab plus paclitaxel chemotherapy. After six months, she developed cognitive decline and memory impairment. MRI showed multiple cortical and subcortical diffusion-restricted lesions without gadolinium enhancement, initially suggesting Trousseau's syndrome. Follow-up MRI demonstrated minimal lesion enlargement, while CSF cytology was negative for malignancy. Open biopsy revealed tumor cells morphologically similar to the primary breast cancer spreading along the Virchow-Robin spaces, confirming LMC [4].

CE-MRI has become the imaging modality of choice, revealing leptomeningeal enhancement, nodular dural deposits, involvement of the cranial and spinal nerves, and hydrocephalus [6].

Treatment of LMC is primarily palliative, aiming to preserve neurological function and improve quality of life [2,7]. Therapeutic options include systemic or intrathecal chemotherapy, used alone or in combination with radiotherapy [2]. WBRT remains a cornerstone for patients with multiple brain metastases or symptomatic LMC [2]. Focal radiotherapy is reserved for localised symptomatic lesions, cranial nerve palsies, cauda equina involvement, or tumoural obstruction of CSF pathways to achieve local symptom control. However, survival is generally not significantly prolonged [2].

Prognosis depends upon the patient's age, performance status, histological subtype of tumour, cranial nerve involvement, high CNS disease load, metastasis, particularly to the lungs, prior treatment, intensity of current therapy, CSF obstruction, and findings of high protein and low glucose [7].

The patient in the present case, with heavily pretreated TNBC, multiple parenchymal and spinal metastases, and leptomeningeal spread, represents the aggressive clinical course typical of this entity. The present case underscores the importance of early recognition of neurological symptoms in advanced breast cancer patients, particularly those with TNBC and prior CNS involvement. It also highlights the limited efficacy of current therapeutic options in LMC. Newer approaches to LM include CSF circulating tumour cell assays, CSF cell-free Deoxyribonucleic Acid analysed by Next-Generation Sequencing and standardised European Association of Neuro-Oncology-European Society for Medical Oncology response frameworks for improved diagnosis and follow-up [8]. On the therapeutic side, modern CNS-active systemic therapies, antibody-drug conjugates, immunotherapy in selected patients, and biomarker-guided treatment strategies are emerging. However, evidence remains limited and largely subtype-specific [9].

CONCLUSION(S)

TNBC is highly aggressive and carries a strong tendency for CNS spread. Early recognition of new neurological symptoms, timely imaging, and a multidisciplinary approach are essential for enhancing supportive care and maintaining quality of life in these patients. MRI and CSF analysis are necessary for diagnosis, though sensitivity limitations require consideration of brain biopsy and repeated testing, especially in TNBC.

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