Case Report

Spinal Paradox in Suspected Thoracolumbar Metastatic Epidural Spinal Cord Compression from Recurrent HER2-Positive Breast Cancer

Bima D. Alparisi,[1*] Sucipto Sucipto,[2] Yossi Maryanti,[2] Daril A. Rasyid,[2] Novia Aiko,[2] Riki Sukiandra,[2] Desby Juananda,[2] Agus T. Joko,[2] M. Ihsan[3]

[1]Clinical Clerkship, RSUD Arifin Achmad, Riau University, Pekanbaru, Indonesia
[2]Department of Neurology, RSUD Arifin Achmad, Riau University, Pekanbaru, Indonesia
[3]Medical Education Program, Riau University, Pekanbaru, Indonesia

*****Corresponding author: bimadiokta21@gmail.com
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Abstract

Metastatic epidural spinal cord compression is a neurological emergency. Thoracolumbar involvement can produce overlapping cord and nerve-root findings, complicating localisation when degenerative lumbar disease coexists. This report describes the clinical-radiological correlation of mixed upper and lower motor neuron signs in a patient with suspected thoracolumbar metastatic epidural spinal cord compression due to recurrent HER2-positive breast cancer. A 56-year-old woman with recurrent HER2-positive left breast carcinoma developed progressive back pain, severe paraparesis, asymmetric sensory impairment, and bladder–bowel dysfunction. Examination demonstrated predominantly flaccid weakness, diminished reflexes, and bilateral extensor plantar responses. MRI showed L1 vertebral collapse and T12–L2 epidural extension compressing the distal spinal cord and conus medullaris, alongside L5–S1 disc protrusion. Mastectomy-bed ultrasonography was classified as BI-RADS 5, and biopsy reportedly supported locoregional recurrence. Spinal metastatic involvement remained clinically and radiologically suspected. Anatomical findings suggested combined spinal cord and nerve-root involvement. However, a patient-specific pathophysiological mechanism could not be confirmed. Treatment included corticosteroids, zoledronic acid, supportive care, and rehabilitation. Partial motor recovery and improvement in bladder and bowel control were reported, although objective follow-up measurements were unavailable. This case highlights the importance of clinical-radiological correlation in patients presenting with mixed upper and lower motor neuron signs. Progressive weakness and sphincter dysfunction in breast cancer survivors should prompt urgent spinal imaging and multidisciplinary evaluation, even when flaccid weakness predominates.
Keywords: *breast carcer, HER2-positive, metastatic epidural spinal cord compression, conus medullaris, paraparesis.*

Paradoks Spinal Pada Kompresi Medula Spinalis Epidural Metastatik Torakolumbal Pada Kanker Payudara HER2-Positif Rekuren

Abstrak

Kompresi medula spinalis epidural metastatik merupakan kegawatdaruratan neurologis. Keterlibatan torakolumbal dapat menimbulkan kombinasi gangguan medula dan akar saraf yang menyulitkan lokalisasi lesi, terutama bila disertai penyakit degeneratif lumbal. Laporan kasus ini mendeskripsikan korelasi klinis-radiologis tanda neuron motorik atas dan bawah yang muncul bersamaan pada pasien dengan dugaan kompresi medula spinalis epidural metastatik torakolumbal akibat kanker payudara HER2-positif rekuren. Perempuan berusia 56 tahun dengan karsinoma payudara kiri rekuren HER2-positif mengalami nyeri punggung progresif, paraparesis berat, gangguan sensorik asimetris, serta disfungsi kandung kemih dan usus. Pemeriksaan menunjukkan kelemahan dominan flaksid, penurunan refleks, dan respons plantar ekstensor bilateral. MRI memperlihatkan kolaps vertebra L1 serta perluasan epidural T12–L2 yang menekan medula spinalis distal dan konus medularis, disertai protrusi diskus L5–S1. Ultrasonografi daerah mastektomi dikategorikan sebagai BI-RADS 5, sedangkan biopsi dilaporkan mendukung kekambuhan lokoregional. Metastatis spinal merupakan dugaan klinis dan radiologis. Temuan anatomis mendukung kombinasi keterlibatan medula spinalis dan akar saraf, namun tidak dapat membuktikan mekanisme patofisiologis spesifik pada pasien ini. Penatalaksanaan mencakup kortikosteroid, asam zoledronat, perawatan suportif, dan rehabilitasi. Perbaikan motorik parsial serta peningkatan kontrol berkemih dan defekasi dilaporkan meskipun luaran tindak lanjut objektif tidak tersedia. Laporan ini menekankan pentingnya korelasi klinis-radiologis pada pasien dengan kombinasi tanda neuron motorik atas dan bawah. Kelemahan progresif dan disfungsi sfingter pada penyintas kanker payudara harus mendorong dilakukannya pencitraan spinal segera dan evaluasi multidisiplin, meskipun kelemahan flaksid lebih dominan.
Kata kunci*: karsinoma payudara, HER2-positif, kompresi medula spinalis epidural metastatik, konus medularis, paraparesis.*

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Introduction

Breast cancer is the most frequently diagnosed malignancy among women and remains a leading cause of cancer-related mortality worldwide. Globally, it accounts for a substantial proportion of new cancer cases and continues to pose a major public health burden despite advances in early detection and systemic therapy.[1] Improvements in targeted therapy, hormonal treatment, and multimodal oncologic management have significantly increased long-term survival, resulting in a growing population of breast cancer survivors. However, prolonged survival has been accompanied by an increased incidence of late-onset distant metastases, particularly involving the skeletal system.[1,2] Bone metastasis represents a common and clinically significant complication of advanced breast cancer, with the spine being the most frequently affected skeletal site. Approximately 70% of patients with metastatic breast cancer develop spinal involvement during the course of their disease.[2] The thoracolumbar region is especially vulnerable due to its rich vascular supply, extensive venous plexus, and high bone marrow activity, which facilitate hematogenous dissemination and tumor cell seeding. Metastatic lesions typically originate within the vertebral body and extend posteriorly into the epidural space, leading to metastatic epidural spinal cord compression (MESCC).[2,3]

MESCC is a neurological emergency characterized by progressive axial back pain, motor weakness, sensory disturbances, and autonomic dysfunction. Among patients with breast cancer, the incidence of MESCC is estimated to range between 10% and 15%.[3] Delayed diagnosis can result in rapid and irreversible neurological deterioration, including permanent loss of ambulation and sphincter control. Consequently, early recognition and standardized management pathways are strongly emphasized in contemporary oncologic and neurological guidelines.[3,4] Although spinal metastases most commonly produce upper motor neuron (UMN) signs such as spastic paraparesis and hyperreflexia, lesions involving the thoracolumbar junction, conus medullaris, or cauda equina may present atypically. In these cases, lower motor neuron (LMN) type weakness, hyporeflexia, flaccidity, asymmetric sensory deficits, and neurogenic bladder or bowel dysfunction may predominate.[5] Such presentations can mimic peripheral neuropathy or lumbosacral plexopathy, frequently leading to diagnostic delay, particularly in patients who have completed oncologic treatment and are presumed to be in remission.[5,6]

This case report aims to highlight the importance of clinical-radiological correlation in patients presenting with mixed upper and lower motor neuron signs that complicate neurological localisation. The coexistence of suspected thoracolumbar metastatic epidural spinal cord compression and lumbar degenerative disease in this patient illustrates the diagnostic challenges encountered in evaluating progressive weakness and sphincter dysfunction in breast cancer survivors.

Case Description

A 56-year-old woman was diagnosed with left breast cancer in 2021 and underwent total mastectomy with axillary lymph node dissection. The primary tumour was reported as invasive breast cancer of no special type, Scarff–Bloom–Richardson grade 2, with an initial anatomical stage of pT2N1aM0 (stage IIB). Immunohistochemical assessment demonstrated oestrogen receptor (ER) positivity in 80% of tumour nuclei (Allred score, 7/8), progesterone receptor (PR) positivity in 60% (Allred score, 6/8), and HER2 overexpression (IHC 3+). Adjuvant treatment comprised six chemotherapy cycles described as an AC–T regimen, consisting of doxorubicin and cyclophosphamide followed by paclitaxel, intravenous trastuzumab for 18 cycles over approximately one year, and irradiation of the left chest wall and regional lymph nodes with 50 Gy in 25 fractions. Trastuzumab treatment was completed in early 2023. Targeted ultrasonography performed on 30 December 2024 identified multiple irregular hypoechoic lesions in the superomedial portion of the left mastectomy bed, the largest measuring 2.0 × 1.0 × 1.6 cm, categorised as BI-RADS 5 (Figure 1). Subsequent

ultrasound-guided core needle biopsy was reported to demonstrate adenocarcinoma morphologically consistent with the breast primary. Receptor reassessment showed ER positivity in 65% of tumour cells, absent PR expression, and persistent HER2 overexpression (IHC 3+), supporting recurrence at the sampled site. Serum carcinoembryonic antigen and CA 15-3 concentrations were reported as 7.8 ng/mL and 49.6 U/mL, respectively. The biopsy date and original pathology report were unavailable for verification, and whether positron emission tomography–computed tomography (PET-CT) was performed remained unspecified.

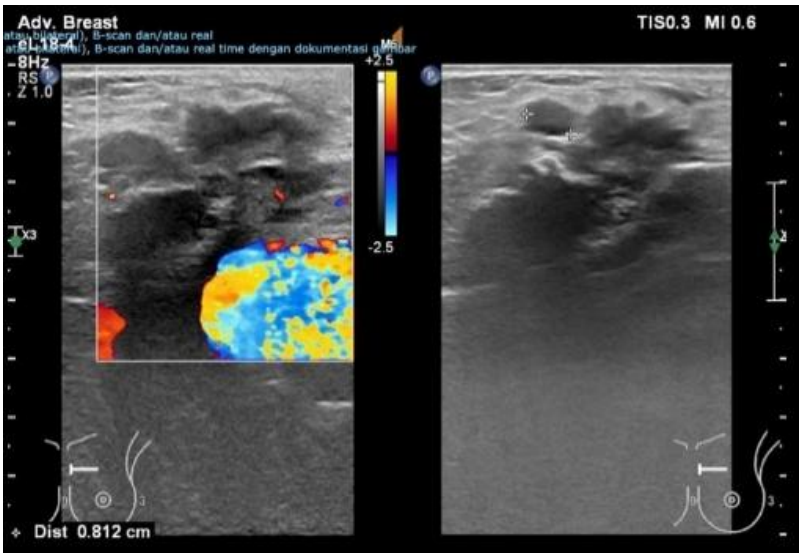


Figure 1. Targeted Ultrasonography of The Left Mastectomy Bed Demonstrating Multiple Irregular Hypoechoic Lesions Classified as BI-RADS 5, Highly Suggestive of Recurrent Malignancy

Persistent low-back pain began on 12 March 2025 and radiated into the right leg in an L5–S1 distribution, accompanied by paraesthesia and progressive gait instability. An accidental fall on 20 May was followed by worsening bilateral lower-limb weakness and loss of independent ambulation, although the causal relationship between the fall and neurological deterioration could not be established. By 5 August, the patient had developed marked sensory impairment below the reported T12 level, urinary hesitancy progressing to overflow incontinence, and loss of bowel control. She was admitted through the emergency department of RSUD Arifin Achmad on 26 October 2025 with severe paraparesis and was entirely confined to bed. Associated symptoms included reduced appetite and a weight decrease from 52 to 48 kg over the preceding month. At admission, she was alert, with a Glasgow Coma Scale score of 15 and reportedly stable vital signs. Physical examination identified a left mastectomy scar and a palpable sternal bony prominence. Reported functional assessments included a Walking Index for Spinal Cord Injury II score of 0, a Spinal Cord Independence Measure III score of 18/100, a Karnofsky Performance Status score of 30%, and an Eastern Cooperative Oncology Group performance status of 4. Earlier diagnostic evaluation was evident from the pre-admission ultrasonography and MRI, but the dates and circumstances of consultations specifically addressing the progressive neurological symptoms were unavailable.

Neurological examination demonstrated severe asymmetric paraparesis, predominantly flaccid lower-limb tone, and mild proximal spasticity. Upper-limb strength was preserved at 5/5 across the C5–T1 key muscle groups bilaterally, yielding an upper-extremity motor

score of 50/50. Lower-limb Medical Research Council strength scores, expressed as right/left, were 3/5 and 2/5 for hip flexion (L2), 2/5 and 1/5 for knee extension (L3), 1/5 and 0/5 for ankle dorsiflexion (L4), 1/5 and 0/5 for great-toe extension (L5), and 0/5 bilaterally for ankle plantarflexion (S1). The lower-extremity motor score was 10/50, comprising 7/25 on the right and 3/25 on the left. Biceps and triceps reflexes were preserved at 2+ bilaterally; the right patellar reflex was diminished at 1+, while the left patellar reflex and both Achilles reflexes were absent. Babinski, Chaddock, Oppenheim, and Gordon responses were reported as positive bilaterally, demonstrating upper motor neuron signs alongside predominantly lower motor neuron features. Light-touch and pinprick sensation were reported as intact from C2 through T12 bilaterally, with reduced sensation below T12, greater impairment on the left, and marked saddle sensory loss. Sacral examination demonstrated preserved deep anal pressure, absent voluntary anal contraction, reduced anal sphincter tone, and absent bulbocavernosus and anocutaneous reflexes. Urinary retention with overflow incontinence was associated with a post-void residual volume of 480 mL. A provisional American Spinal Injury Association Impairment Scale (AIS) grade C and neurological level of T12 were documented in the medical record. Formal verification was not possible because the complete classification worksheet was unavailable.

Contrast-enhanced MRI performed on 20 October 2025 and reported on 23 October 2025 demonstrated multifocal vertebral lesions extending from the cervical spine to the sacrococcygeal region, with additional involvement of both iliac bones. L1 vertebral body collapse, with approximately 40% height loss, was accompanied by paravertebral and epidural soft-tissue extension from T12 to L2, producing marked canal stenosis and compression of the distal spinal cord and conus medullaris (Figure 2). Additional findings included a diffuse posterior L5-S1 disc protrusion with bilateral nerve-root compression and C6-C7 disc bulging.

The final clinical diagnosis was mixed paraparesis with predominant lower motor neuron features secondary to presumed metastatic spinal cord compression at T12-L2 from recurrent HER2-positive breast cancer, accompanied by cauda equina syndrome, dyslipidaemia, mildly elevated hepatic enzymes, and hypokalaemia. Spinal metastatic involvement remained clinically and radiologically suspected because histopathological confirmation of the spinal lesions was unavailable. The interval from pain onset to MRI was 222 days.

Intravenous dexamethasone was initiated on 27 October with a 16-mg loading dose followed by 4 mg every six hours. Treatment commenced seven days after MRI and 160 days after loss of independent ambulation; however, the interval from complete paraplegia to treatment could not be determined. Multidisciplinary assessment took place on 28 October, and intravenous zoledronic acid (4 mg) was administered on 29 October. Supportive management included spinal precautions, analgesia, medication for neuropathic pain, intravenous fluids, electrolyte correction, bladder and bowel care, nutritional support, and physiotherapy.

At discharge, the patient reported improved lower-limb movement and better bladder and bowel control compared with admission. However, repeat motor scores, functional assessments, and objective sphincter outcomes were unavailable to quantify the magnitude of recovery. The patient was discharged on 12 November following clinical stabilisation and initiation of rehabilitation. The chronological sequence of major clinical events is summarised in Table 1.

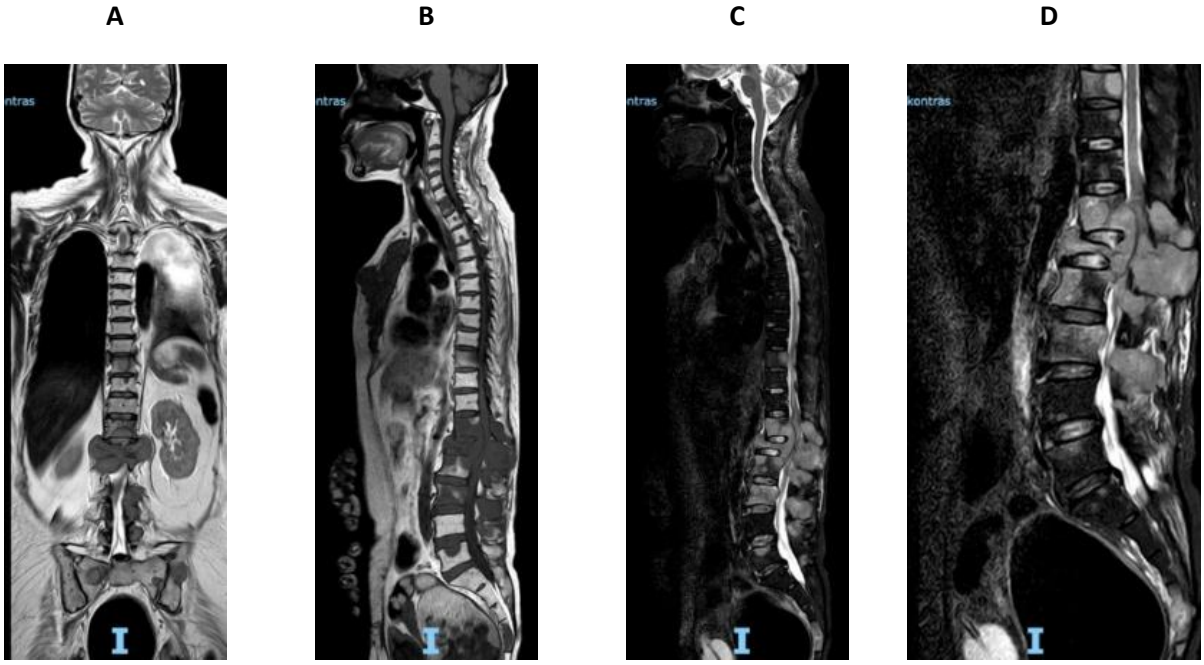


Figure 2. Contrast-enhanced Whole-spine MRI Demonstrating L1 Vertebral Collapse with Paravertebral and Epidural Soft-tissue Extension from T12 to L2 Causing Compression of the Distal Spinal Cord and Conus Medullaris. (A) Coronal Image. (B–D) Sagittal Images, Including a Focused Thoracolumbar View (D)

Table 1. Clinical Timeline

Date	Clinical Event
2021	Left breast cancer diagnosed (pT2N1aM0, Stage IIB)
2021–2022	Mastectomy, chemotherapy, radiotherapy
Early 2023	Completion of trastuzumab
30 Dec 2024	BI-RADS 5 lesion detected in mastectomy bed
Early 2025	Core biopsy consistent with recurrent breast cancer
12 Mar 2025	Progressive low-back pain began
20 May 2025	Fall followed by worsening weakness
5 Aug 2025	Sensory loss and bladder-bowel dysfunction developed
20 Oct 2025	Whole-spine MRI performed
26 Oct 2025	Hospital admission
27 Oct 2025	Dexamethasone initiated
28 Oct 2025	Multidisciplinary evaluation
29 Oct 2025	Zoledronic acid administered
12 Nov 2025	Discharged and referred for rehabilitation

Discussion

This case illustrates the clinical–radiological correlation of predominantly flaccid paraparesis with bilateral extensor plantar responses in a breast cancer survivor with L1 vertebral collapse, suspected metastatic epidural compression at T12–L2, and coexisting L5–S1 disc protrusion. Previously published breast cancer cases have described flaccid weakness with extensor plantar responses in intramedullary metastasis and cauda equina dysfunction caused by an epidural mass resembling a sequestered lumbar disc.[7,8] Distinctive features in this patient therefore arise from the combination of vertebral destruction, epidural extension, distal cord–conus compression, and additional degenerative nerve-root compression, rather than from mixed motor signs alone. MESCC and pathological vertebral collapse are clinically important complications of spinal metastases and may lead to substantial neurological morbidity.[9]

Reassessment of receptor status is recommended in recurrent breast cancer because receptor discordance may occur and influence

subsequent treatment decisions.[10] In this patient, the recurrent lesion reportedly demonstrated persistent HER2 overexpression with loss of PR expression. However, spinal involvement remained a clinical-radiological diagnosis because the local biopsy did not establish the histology or receptor profile of the vertebral lesions. Breast cancer commonly causes osteolytic vertebral destruction, which may contribute to pathological fracture, spinal instability, and epidural extension resulting in neural compression.

Thoracolumbar anatomy explains how a single regional lesion can produce overlapping upper and lower motor neuron manifestations. Anatomical variation places the conus medullaris near the affected vertebral levels, allowing compression to involve distal spinal cord structures and adjacent descending nerve roots simultaneously.[11] Corticospinal tract dysfunction offers an anatomical explanation for bilateral extensor plantar responses, while involvement of segmental motor structures and lumbosacral roots can account for flaccidity, diminished reflexes, and disproportionate distal weakness. Epidural compression may also contribute to oedema and progressive neurological deterioration.[12] These processes provide a theoretical explanation for progressive motor and sensory deterioration, although the available MRI description does not independently establish ischaemia or tissue necrosis. Coexisting L5–S1 disc protrusion may contribute to radicular pain and distal root dysfunction, but an isolated lumbar root lesion would not explain bilateral extensor plantar responses. Clinical localisation therefore requires integration of the neurological examination with the T12–L2 epidural abnormality demonstrated in (Figure 2), rather than attribution of the entire syndrome to the degenerative disc finding.

Sacral findings strengthen the interpretation of combined conus–cauda involvement and clarify the functional consequences of compression. Marked saddle sensory loss, reduced anal tone, absent sacral reflexes, and urinary retention with overflow incontinence are compatible with disruption of sacral sensory, motor, and autonomic pathways.[11] A post-void residual volume of 480 mL provides objective evidence of impaired bladder emptying, although it does not establish a specific urodynamic pattern. Management of suspected MESCC requires multidisciplinary assessment that considers neurological status, oncological factors, spinal stability, and overall functional condition.[13] In this patient, L1 collapse and severe functional impairment highlighted the need for coordinated evaluation and individualized treatment planning.

Progressive weakness and sphincter dysfunction in a patient with cancer require an emergency pathway for suspected metastatic spinal cord compression. Current guidance recommends urgent specialist coordination and MRI as soon as possible, within 24 hours of suspected compression, rather than waiting for complete paralysis.[14] Available dates indicate 222 days between initial pain onset and MRI, seven days between MRI and dexamethasone initiation, and 160 days between loss of independent ambulation and corticosteroid treatment. These intervals demonstrate prolonged symptom evolution but do not establish when a clinician first suspected compression or whether treatment had been initiated elsewhere. Pre-admission ultrasonography and MRI confirm earlier diagnostic contact, while the timing and content of consultations addressing neurological deterioration remain unavailable. Corticosteroid administration addresses a potentially reversible component of compression-related oedema while definitive treatment is considered. Dexamethasone maintenance at 4 mg every six hours provides 16 mg daily, matching the daily dose recommended for neurological manifestations of metastatic spinal cord compression while awaiting surgery or radiotherapy.[14] Unspecified details of decompression, stabilisation, or spinal radiotherapy prevent assessment of definitive treatment delivery and preclude attributing neurological improvement to corticosteroids alone.

Supportive treatment addressed pain, skeletal morbidity, immobility, and autonomic dysfunction during hospitalisation. Zoledronic acid was administered as a bone-modifying agent to

reduce skeletal-related complications associated with metastatic bone disease.[15] Supportive care, including rehabilitation, bladder and bowel management, nutritional support, and prevention of secondary complications, remained important components of treatment.[16] Concurrent hypokalaemia may have contributed to weakness but did not explain the sensory level, extensor plantar responses, and sacral deficits observed in this patient.

Reported partial motor recovery and improved bladder and bowel control suggest clinical improvement but remain insufficient to establish its magnitude or durability. Repeat muscle-group scores, standardised functional assessments, and objective bladder measurements would provide stronger evidence of neurological and functional change.[16,17] Baseline WISCI II, SCIM III, and performance-status scores document severe disability, but isolated baseline values cannot demonstrate treatment effectiveness. Missing spinal histopathology, unverified original biopsy reports, and unspecified definitive treatment further constrain causal interpretation.

Despite these limitations, this case demonstrates that predominantly flaccid paraparesis does not exclude thoracolumbar spinal cord compression. Recognition of mixed upper and lower motor neuron signs, particularly when accompanied by sensory level impairment and sphincter dysfunction, remains essential for timely diagnosis and management in patients with a history of breast cancer.

Conclusion

This case highlights the importance of clinical-radiological correlation in patients presenting with mixed upper and lower motor neuron signs. Predominantly flaccid weakness does not exclude thoracolumbar spinal cord compression, particularly when accompanied by sensory level impairment and sphincter dysfunction. Early recognition and multidisciplinary management are essential to avoid delayed diagnosis and preserve neurological function in breast cancer survivors.

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Author Contribution

Bima Diokta Alparisi made the primary contribution to this study, including case conceptualization, clinical data collection, literature review, analysis and interpretation of findings, and drafting of the manuscript. Sucipto, Yossi Maryanti, Daril-Alrasyid, Novia Aiko, Riki Sukiandra, Desby Juananda, and Agus Tri Joko contributed to clinical discussion, critical appraisal of the manuscript, interpretation of neurological and radiological findings, and provided important revisions to enhance scientific accuracy and clarity. M. Ihsan contributed substantially to study design, manuscript structuring, scientific formatting, and critical revision for important intellectual content.

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Data and Materials Availability

All relevant data supporting the findings of this case report are included within the article. Additional information may be requested from the corresponding author.

Consent for Publication

Written informed consent for publication of this case report and any accompanying identifiable data was obtained from the patient.

Competing Interests

The authors declare that there are no conflicts of interest regarding the publication of this article.

AI Usage Declaration

Artificial intelligence tools were used for language refinement and improving clarity of the manuscript. All clinical information, interpretations, and conclusions were reviewed, verified, and approved by the authors. The authors take full responsibility for the content of this manuscript.

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