Late manifestation of intracranial leptomeningeal carcinomatosis secondary to breast cancer: a case report

Nu Nu Win, Berrin Erok

Case Report

Late manifestation of intracranial leptomeningeal carcinomatosis secondary to breast cancer: a case report

Nu Nu Win[1], Berrin Erok[2]

[1]Department of Radiology, Medicana Bahcelievler Hospital, Istanbul, Turkey
[2]Department of Radiology, Prof. Dr. Cemil Tascioglu City Hospital, Istanbul, Turkey

Corresponding Author:
Berrin Erok, MD.
Department of Radiology,
Prof. Dr. Cemil Tascıoglu City Hospital,
Istanbul,
Turkey
ORCID: https://orcid.org/0000-0001-8036-547X
email Id - drberrinerok@hotmail.com
Contact number - 05302604150

ABSTRACT

Leptomeningeal carcinomatosis (LMC), also known as carcinomatous meningitis is defined as the spread of malignant cells to pia and arachnoid maters. Most of the cases occur as an uncommon, catastrophic and late complication of metastatic carcinomas. Breast cancer is the most common cause, followed by lung cancer, melanoma and haematologic malignancies. Occurance of LMC without brain parenchymal involvement is rare. We present a 31 year old female patient with with history of breast cancer treatment 2 years ago, who was admitted to the emergency department with neurological deterioration and diagnosed with LMC without intraparenchymal metastasis.

KEY WORDS: leptomeningeal carcinomatosis, carcinomatous meningitis, breast cancer, MRI

INTRODUCTION

Leptomeningeal carcinomatosis (LMC), also known as carcinomatous meningitis is defined as the spread of malignant cells to pia and arachnoid maters through the cerebrospinal fluid (CSF) spaces. LMC may be intracranial or spinal. These cells can be originated from primary CNS tumors as in drop metastases. However, most of the cases occur as an uncommon, catastrophic and late complication of metastatic carcinomas, likely via haematogenous spread with an approximate incidence of 5–8% in solid tumors and 5–15% in hematologic malignancies [1].

Over 50% of cases have concurrent brain parenchymal metastases [2]. Breast cancer is the most common cause, followed by lung cancer, melanoma and haematologic malignancies. Only about 5-8% of breast cancer patients develop leptomeningeal metastasis [3,4] and the prevalence of LMC without brain parenchymal involvement is around 3.5% [5]. We present a female patient with history of breast cancer treatment 2 years ago who was admitted to the emergency department with neurological deterioration and diagnosed with LMC without intraparenchymal metastasis.

CASE REPORT

A 31-year-old female patient presented to the emergency deparment with the complaints of headache, nausea, vomiting, diplopia, blurr speech, mental alteration and gait disturbance. She had a medical history of right breast lumpectomy and axillary dissection with the diagnosis of breast cancer 2 years ago. She had also received chemo-radiotherapy at that time. Head computed tomography (CT) performed at the day of admission and showed moderate triventricular hydrocephalus and periventricular hypodensities suggesting transependymal CSF leakage. Supra and infratentorial sulcal effacement, loss of cerebral fissures and all basal cisterns were noted. Mesencephalon, pons and medulla oblongata were markedly compressed as the result of increased intracranial pressure (fig. 1). Contrast enhanced brain magnetic resonance imaging (MRI) has been ordered and revealed diffuse hyperintensites of the tentorium and folia of cerebellum on pre-contrast T2W and FLAIR sequences. Faint, lineer cerebral leptomeningeal hyperintensities were also observed on FLAIR images. The

obliteration of basal cisterns, compression of brain stem and cerebellar tonsillar herniation were evidient on sagittal images (fig. 2). Diffuse linear leptomeningeal enhancement of both cerebral and cerebeller hemispheres, more prominently at posterior fossa, were seen on post-contrast T1W images (fig. 3). No intraparenchymal lesion was detected. An external ventricular drainage (EVD) catheter was placed urgently for acute management of the hydrocephalus. Infectious etiologies were excluded by cytological analysis of CSF obtained from EVD rather than lumbar puncture which is contraindicated in this case due to the patient acute hydrocephalus and raised ICP. Atypical pleomorphic malignant cells. were revealed. Based on both the cytological analysis of CSF (obtained from the EVD catheter) and on the MRI findings, the patient was diagnosed with LMC. MRI of entire spinal axis was recommended but it could not be done because of the unstable status of the patient. Despite intrathecal methotrexate therapy and all other supportive treatments, the patient gradually deteriorated and deceased 30 days post admission.

DISCUSSION

LMC is one of the late complications of metastastic cancers. Clinical presentations vary with the site of the nervous system infiltrated by tumor cells. Cerebral involvement manifests as headaches, nausea, seizures and communicating hydrocephalus. Cranial nerve involvement may cause diplopia, decreased acuity of vision, hearing loss, facial numbness. Spinal involvement may result in extremity weakness, paresthesias and/or pain [5]. Our case had all kinds of symptoms associated with cerebral, cerebeller and cranial nerve involvement. Rarely, LMC may be the first presentation of the patient with no history of primary malignancy. Shin *et al.* reported 3 cases of LMC who presented with meningitis symptoms to the emergency department without previous diagnosis of malignancy [6]. Although CSF cytology has a high specifty, it is not sensitive with high false-negative rates as reported in many studies [7,8]. Therefore, LMC is a diagnosis primarily based on MRI findings. The primary finding is diffuse or focal, lineer-nodular gyriform leptomeningeal enhancements often scattered over the brain in a sugar coated manner and possible nerve root enhancements [9]. However, leptomeningeal enhancement is not specific for LMC and can also be observed in other conditions resulting in leptomeningeal irritations such as infectious and inflamatory meningitis, chemical meningitis, vasculitis and neurosarcoidosis [10]. Additionally, false positive leptomeningeal enhancement may be seen in patients under immunotherapy [11], intrathecal therapy and patients with intracranial hypotension [12]. On precontrast images conventional T1w and T2w sequences may be normal but, abnormally elevated signals within the sulci on FLAIR images may often be seen [13].

CONCLUSION

In patients with previous history of malignancy and new onset or worsening headache and/or other neurologic manifestations contrast enhanced MRI of the brain and whole spinal axis should be performed. Early recognition of acute hydrocephalus secondary to LMC, together with timely placement of EVD catheter are pivotal steps in the initial management. In these patients, EVD is therapeutic and also diagnostic in that it allows

for CSF sample collection safely (in contrast to LP which should be avoided).In cases without malignant cells on CSF cytology, other possible causes of leptomeningeal enhancement on brain MRI should also be ruled out.

ACKNOWLEDGMENT

There is no conflict of interest.

Funding: none

Informed written consent: was obtained from the patient.

REFERENCES

1. Beauchesne P. Intrathecal chemotherapy for treatment of leptomeningeal dissemination of metastatic tumours (Review). Lancet Oncol. 2010; 11(9):871–879.

2. J.L. Clarke, H.R. Perez, L.M. Jacks, K.S. Panageas, L.M. DeAngelis. Leptomeningeal metastases in the MRI era (Retrospective study). Neurology 2010; 74(18):1449.

3. Le Rhun E, Taillibert S, Chamberlain MC. Carcinomatous meningitis: Leptomeningeal metastases in solid tumors (Review). Surg Neurol Int. 2013; (Suppl 4):S265-S288.

4. Gautam Nayar , Tiffany Ejikeme , Pakawat Chongsathidkiet , *et al*. Leptomeningeal disease: current diagnostic and therapeutic strategies (Review). Oncotarget. 2017; 9:73312-73328.

5. Posner JB. Neurologic complications of cancer. Philadelphia, PA: F A Davis Company, 1995; 143–68.

6**.** Shin Ahn , Kyung Soo Lim**.** Three Cases of Neoplastic Meningitis Initially Diagnosed with Infectious Meningitis in Emergency Department (Case Report). Case Rep Emerg Med. 2013; 2013:561475.

7. Wasserstrom WR, Glass JP, Posner JB. Diagnosis and treatment of leptomeningeal metastases from solid tumors: Experience with 90 patients (Comparative study). Cancer. 1982; 49:759–772.

8. Kak M, Nanda R, Ramsdale EE, Lukas RV. Treatment of leptomeningeal carcinomatosis: current challenges and future opportunities (Review). J Clin Neurosci. 2015; 22:632-637.

9.Chamberlain MC. Neuraxis imaging in leptomeningeal metastasis: A retrospective case series (Retrospective study). J Clin Oncol 2012;30.

10. Aiken AH. Central nervous system infection (Review). Neuroimag. Clin N. Am. 2010; 20(4), 557–580.

11.Bier G, Klumpp B, Roder C, Garbe C,*et al*. Meningeal enhancement depicted by magnetic resonance imaging in tumor patients: neoplastic meningitis or therapy-related enhancement? (Retrospective study). Neuroradiology. 2019; Jul;61(7):775-782.

12. Wang N, Bertalan MS, Brastianos PK. Leptomeningeal metastasis from systemic cancer: Review and update on management (Review). Cancer. 2018; Jan 1;124(1):21-35.

13. Singh SK, Agris JM, Leeds NE *et al*. Intracranial leptomeningeal metastases: comparison of depiction at FLAIR and contrast-enhanced MR imaging (Comparative study). Radiology. 2000; 217 (1): 50-3.

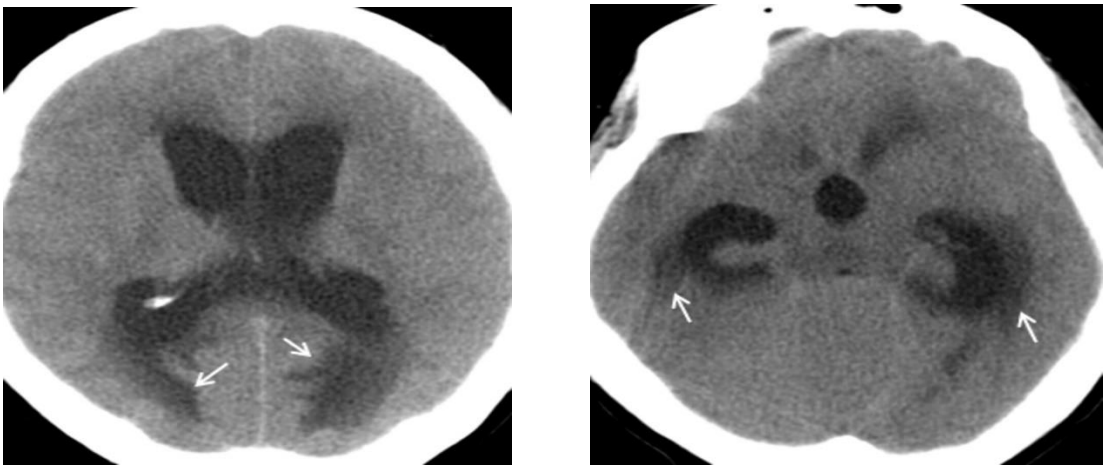

Figure 1: a, b) Head CT scan performed at the day of admission and showed howing moderate triventricular hydrocephalus and periventricular hypodensities suggesting transependymal CSF leakage **(a,b, arrows).** Note the effacemat of sulci, cerebral fissures and basal cisterns. The compressed brain stem is recognizable.

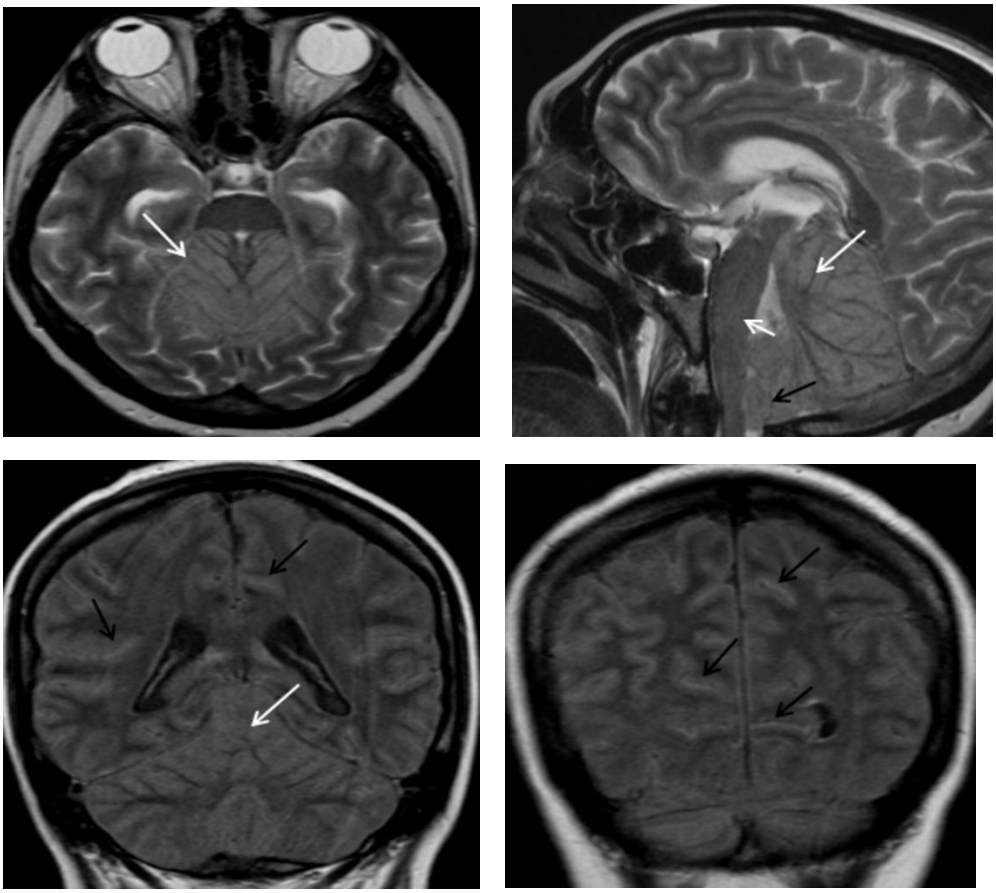

Figure 2: a,b,c,d) Precontrast brain MRI images showing diffuse hyperintensites of the tentorium and folia of cerebellum on T2W **(a,b, White arrows)** and FLAIR **(c, white arrow)** sequences. The lineer cerebral leptomeningeal hyperintensities are also seen on the FLAIR images **(c,d, black arrows).** Note the compression of the brain stem **(b, short white arrow)** and the cerebellar tonsillar herniation **(b, black arrow).**

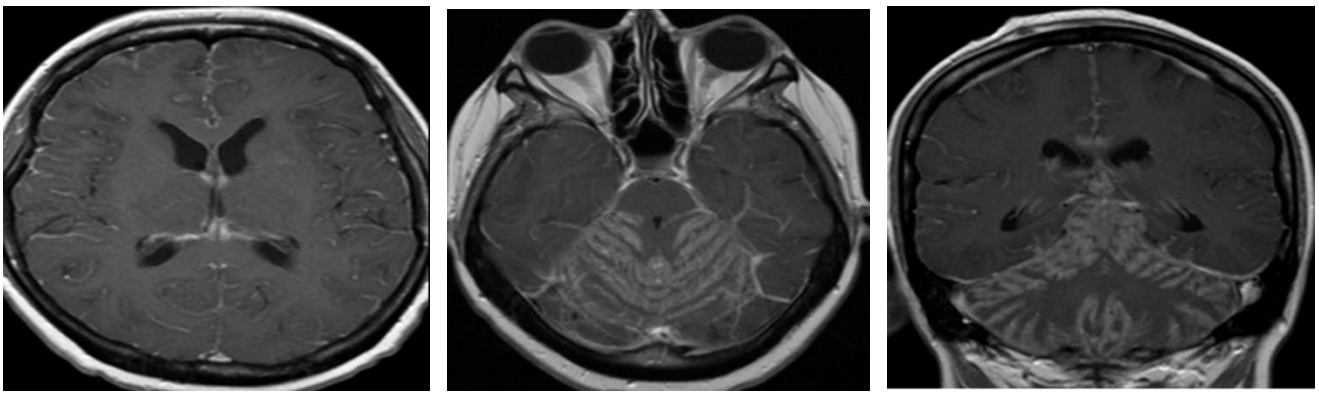

Figure 3: a,b,c) Postcontrast brain MRI images showing diffuse linear leptomeningeal enhancement of both cerebral **(a)** and cerebeller hemispheres **(c,d)**, more prominently at posterior fossa on post-contrast T1W images.