Annals of Clinical and Medical Case Reports

Clinical Image

ISSN 2639-8109 | Volume 10

Coma Due to Wernicke Encephalopathy During Neoadjuvant Chemotherapy for Breast Cancer

Richard ME[1], Fayard J[2], Laadhari M[2], Kiavue N[3] and Langer A[2*]

[1]Department of Imaging, Sainte Anne Hospital, Paris, France

[2]Department of Imaging, Curie Institute, Saint-Cloud, France

[3]Oncology Department, Curie Institute, Saint Cloud, France

[*]Corresponding author:

Adriana Langer,
Department of Imaging, Curie Institute,
Saint-Cloud, France,
E-mail: adriana.langer@curie.fr

Received: 27 Oct 2022
Accepted: 05 Nov 2022
Published: 11 Nov 2022
J Short Name: ACMCR

Keywords:

Wernicke encephalopathy; Neurological; Hydroxychloroquine

Citation:

Langer A, Coma Due to Wernicke Encephalopathy During Neoadjuvant Chemotherapy forBreast Cancer. Ann Clin Med Case Rep. 2022; V10(5): 1-3

1. Abstract

We report the case of a 49 yr., woman who developed a coma during neoadjuvant chemotherapy for breast cancer. Brain MRI was crucial for the diagnosis of Wernicke encephalopathy.

2. Introduction

Wernicke encephalopathy is a neurological disorder induced by thiamine (vitamin B1) deficiency. Clinically, there is a classical triad consisting of ocular signs with ophthalmoplegia, ataxia and altered consciousness [1]. It is often associated with alcohol abuse [2]. Its diagnosis is essential as treatment requires rapid supplementation with thiamine, which determines prognosis [3]. Brain MRI can be a key to the diagnosis as it can show typical findings [4].

3. Case Report

We report the case of a 49 yo woman, with past history of lupus and cerebral vein thrombosis and long term treatment with Coumadine and Hydroxychloroquine. She presented with bloody nipple discharge, a palpable breast lump and axillary suspicious node (T3N1 by TNM classification) on the right side. Biopsy confirmed grade 2 invasive carcinoma of no special type, with an aggressive phenotype (ER and PR negative, Her2 positive) and axillary lymph node involvement. Breast MRI showed total lesion extension was 110mm, and there were no distant metastasis on Pet-scanner.

Neoadjuvant chemotherapy was decided and began with Cyclophosphamide and Epirubicin. The tolerance was mediocre, as the patient had nausea, vomiting, anorexia and diarrhea, which worsened with each cycle, in spite of the treatments and oral supplements. She lost a total of 13kg (16% of her weight in 6 months).

Two weeks after the fourth cycle, the patient was admitted to the emergency room for altered consciousness and seizure. Main hypothesis in this patient were: meningeal or brain metastasis due to the aggressive breast cancer context, cerebral vein thrombosis and brain or meningeal hemorragia due the patient's past history and treatment (Coumadine), status epilepticus, and unknown encephalopathy.

An initial brain CT scan showed no bleeding and no anomaly after contrast agent injection. Oral anticoagulation was suspended to perform lumbar puncture. Blood results showed hypernatremia (156 mEq/L), glycemia was normal. Anti-epileptic drug (Levetiracetam) was given. Corticoids were introduced in the eventuality of carcinomatous meningitis, but had no effect and were then discontinued. On electroencephalography there was no sign of infraclinic seizure. On the lumbar puncture there were no abnormal cells.

Brain MRI was performed, as there was no explanation to her coma, which aggravated rapidly. There was bilateral symmetric altered signal intensity in mamillary bodies, tectal plate, thalami, periaqueductal and periventricular (3rd and 4th ventricles) white matter and frontal cortex: high signal intensity on Flair and diffusion weighted imaging (without low apparent diffusion coefficient level) and contrast enhancement on T1-weighted imaging (Figure

1). There was no sign of recent cerebral thrombosis or bleeding.

MRI results were thus characteristic of Wernicke encephalopathy, probably aggravated by the hypernatremia. As clinical status worsened (Glasgow 6), the patient was transferred to intensive care unit where she was intubated and received high dose intravenous thiamine. Hypernatremia was also corrected.

The patient developed septic shock, which was due to Klebsiella inhalation pneumonia. Cambylobacter colitis was also diagnosed and both infections were treated.

The patient recovered subnormal consciousness and was discharged from intensive care unit at day 8.

Control MRI at 3 weeks shows partial regression of the signal anomalies with persistent high FLAIR signal intensity of periaqueductal region (Figure 2).

Because of these complications, it was decided to stop chemotherapy. The patient has been successfully operated (mastectomy and axillary dissection: ypT1b N2a). Trastuzumab was initiated and radiotherapy will soon begin. The patient has for sequelae severe anterograde amnesia.

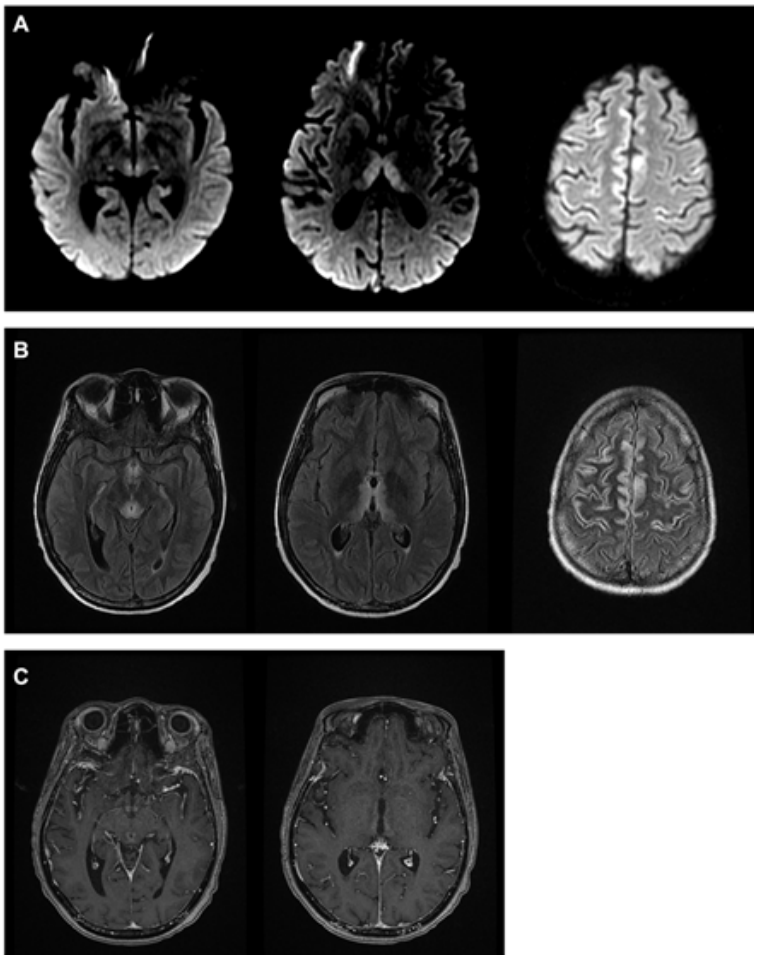

Figure 1: Initial MRI scan showing anomalies in DWI of mamillary bodies, periaqueductal, thalami and frontal cortex: high DWI (A), high flair intensity (B) and contrast-enhancement T1 weighted (C).

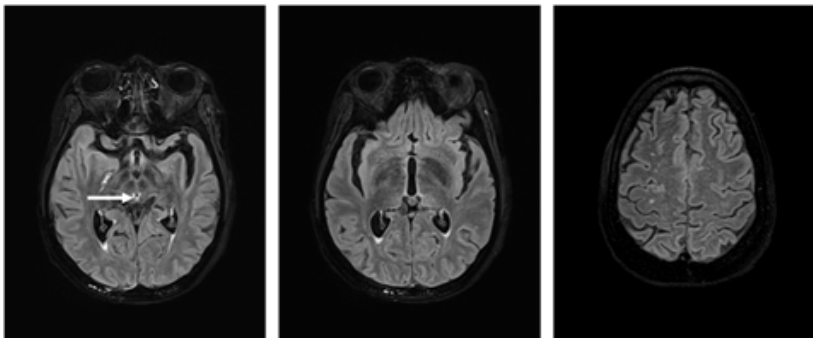

Figure 2: Control MRI at 3 weeks showing persistent Flair hyperintensity of periaqueductal region (arrow) and regression of other signal anomalies.

4. Discussion and Review of the Literature

Wernicke encephalopathy (WE) is caused by thiamine deficiency, and alcohol abuse is its most known etiology, responsible for almost half of the cases. WE were also described in patients treated for cancer, its physiopathology being multifactorial. The most common malignancies reported in these cases were hematological (depletion of thiamine is accelerated in cancers with high cell turnover), the second were gastrointestinal (through malnutrition and malabsorption) [6].

In our case, the patient was under chemotherapy for breast cancer, which induced major malnutrition because of repeated vomiting, anorexia and diarrhea. Clinical symptoms in the patient were wide with altered consciousness and seizure, but no ataxia or ophtalmoplegia (the classical clinical trial was incomplete, which is often the case [1,6,7]) and WE diagnosis was not considered before the MRI scan. The main causes considered (after biological exams dismissed hypoglycemia and other conditions susceptible to explain the progressive coma), were thus, because of the severity of her cancer (which was Her2 positive) meningeal or brain dissemination, and because of her own medical history cerebral vein thrombosis or hemorrhage (she was under Coumadine).

Typical imaging findings on MRI associate symmetric signal anomalies (high signal intensity on Flair and diffusion, which can enhance after Gadolinium injection) of the thalami, mamillary bodies, tectal plate, around the 3rd and 4th ventricles as well as around the aqueduct [4]. MRI findings are key to the diagnosis as clinical features can be wide: classical clinical triad (acute mental confusion, ataxia, and ophthalmoplegia) occurs in only 12 to 38% [1, 6, 7].

Our patient also had signal anomalies of frontal cortex, which could be due to status epilepticus. Cortical anomaly is not typical, but can be seen.

Oncologists must be aware of WE as symptoms can be wide. It is often under-recognized among patients with no history of alcoholism and patients with cancer are at risk for thiamine deficiency, especially in case of repeated vomiting and anorexia as was the case for our patient. Considering the possibility of this disorder and performing quickly a brain MRI is essential to diminish the mortality and morbidity of this condition, which are still high, by rapid thiamine intra-venous infusion. Indeed, Isenberg-Grzeda et al [6] recommend the administration of thiamine at the earlier suspicion of WE, even when other causes are still being investigated, as thiamine is safe, and treatment delay is often linked to worse outcomes.

References

1. Harper CG, Giles M, Finlay-Jones R. Clinical signs in the Wernicke-Korsakoff complex: a retrospective analysis of 131 cases diagnosed at necropsy. J Neurol Neurosurg Psychiatry. 1986; 49: 341-5.
2. Ogershok PR, Rahman A, Brick J, Nestor S. Wernicke Encephalopathy in Nonalcoholic Patients. The American Journal of the Medical Sciences. 2002; 323: 107-11.
3. Galvin R, Bråthen G, Ivashynka A, Hillbom M, Tanasescu R, Leone MA. EFNS guidelines for diagnosis, therapy and prevention of Wernicke encephalopathy. Eur J Neurol. 2010; 17: 1408-18.
4. Zuccoli G, Pipitone N. Neuroimaging Findings in Acute Wernicke's Encephalopathy: Review of the Literature. American Journal of Roentgenology. 2009; 192: 501-8.
5. Lindboe CF, Løberg EM. Wernicke's encephalopathy in non-alcoholics. An autopsy study. J Neurol Sci. 1989; 90: 125-9.
6. Isenberg-Grzeda E, Rahane S, DeRosa AP, et al. Wernicke-Korsakoff syndrome in patients with cancer: a systematic review. The Lancet Oncology. 2016; 17: e142–8.
7. Zuccoli G, Gallucci M, Capellades J, et al. Wernicke encephalopathy: MR findings at clinical presentation in twenty-six alcoholic and nonalcoholic patients. AJNR Am J Neuroradiol. 2007; 28: 1328–31.