Intracranial Hypertension after mTCF Protocol in a Patient with Metastatic Gastric Cancer: A Case Report

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ABSTRACT

Idiopathic intracranial hypertension (IIH) is an entity characterised by the symptoms of increased intracranial pressure without intracranial lesions, which may lead to visual loss. In a patient with Stage IV signet ring cell gastric carcinoma treated with modified docetaxel--cisplatin fluorouracil (mTCF) (methotrexate, docetaxel, cisplatin) protocol. Intracranial hypertension was detected by clinical, laboratory, and imaging tests performed as a result of visual loss and headaches that developed during the treatment. No lesion or haemorrhage was detected on cranial imaging and no additional pathology was found on cerebrospinal fluid (CSF) examination. With all these findings, organic pathologies were excluded and the patient was diagnosed with IIH. Chemotherapy was discontinued and a pressure-reducing shunt operation was performed. The possible IIH-risk of the chemotherapy agents used in this case will contribute to physicians in predicting the additional possible risks of these agents.

Key Words: *Idiopathic intracranial hypertension, Methotrexate, Docetaxel, Cisplatin, Gastric cancer.*

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INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a condition characterised by symptoms of increased intracranial pressure without intracranial lesions. Although rare, it can cause macular complications, permanent nerve fibre defects, and visual impairment. Classically, four pre-requisites are needed for diagnosis: 1) Intracranial pressure increase is not accompanied by intracranial mass or hydrocephalus, 2) Increased CSF pressure, whereas its components are at normal levels, 3) No pathologic findings detected on imaging methods, and 4) Absence of localised findings on neurological examination except papilloedema and abducens paralysis due to increased intracranial pressure.[1]

As these criteria show, the diagnosis of IIH is possible with the exclusion of other possible pathologies. Our case was diagnosed with IIH after the exclusion of organic pathologies in investigations performed due to the complaints of headache and visual loss and will contribute to the literature because of the development of IIH due to chemotherapeutic agents.

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CASE REPORT

The imaging studies of a 52-year woman with metastatic gastric signet ring cell adenocarcinoma revealed multiple bone metastases with left adnexal metastases. Treatment with mTCF (docetaxel 40 mg/m^2, cisplatin 40 mg/m^2, folic acid 400 mg/m^2, 5-fluorouracil 400 mg/m^2 bolus, and 5-fluorouracil 2400 mg/m^2-infusion) protocol was started every 14 days in the first line due to HER-2-negative disease. The patient was evaluated in consultation with the ophthalmology and neurology departments due to the intermittent visual loss and concurrent back and headaches that started after the fifth cycle of the treatment. Neurological examination revealed no lateralisation findings and no abnormality in blood parameters. Brain and diffusion MRI and MR venography showed no findings in favour of metastasis or haemorrhage. Cranial MRI showed papilloedema supporting IIH (Figure 1 A, B). Grade-3 papilloedema was found on fundus examination and cerebrospinal fluid (CSF) pressure was 310 mm H_2O on lumbar puncture examination. The meningitis and encephalitis panel were negative on CSF examination. After the exclusion of all the possible organic pathologies, it was contemplated that the patient developed IIH due to mTCF chemotherapy regimen. A shunt operation was performed by the neurosurgery team to reduce CSF pressure and the chemotherapy was revised as cisplatin-free.

DISCUSSION

IIH occurs secondary to increased intracranial pressure and normal CSF composition without a secondary cause. Approximately 3/100,000 cases are observed annually.[2] The most common complaints are headaches (92%) and transient visual loss (72%).[3] Papilloedema is seen in all the patients with IIH except anecdotal cases and may rarely be detected without a headache. If left

untreated, progressive visual loss and blindness due to optic atrophy may be observed.[4] In order to make a diagnosis of IIH, all causes leading to increased intracranial pressure should be excluded. A detailed anamnesis, laboratory, and imaging tests are important for the correct diagnosis.[5]

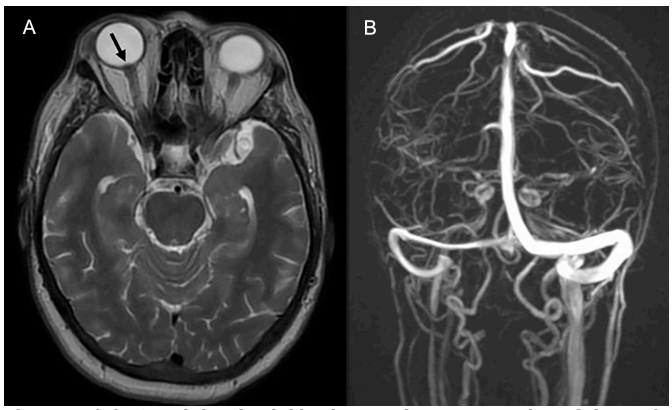

Figure 1: (A) T2-weighted axial brain MRI shows protrusion of the optic nerve-head towards the globe on the right, consistent with intracranial hypertension. (B) Normal MR venography; no evidence of venous thrombosis was observed.

A CSF pressure above 250 mm H_2O in patients with headaches is one of the diagnostic criteria for IIH. CSF pressure measurement is important in the patients with papilloedema when no intracranial lesion is found.[1]

All patients should undergo a detailed physical examination and tests including blood pressure, fever, fasting blood glucose, haemogram, and biochemical parameters in terms of possible pathologies included in the differential diagnosis.[6]

Reversible posterior leukoencephalopathy related to cisplatin has been reported in the literature.[7] Similarly, posterior reversible encephalopathy syndrome was reported in a patient receiving a fluorouracil, leucovorin, oxaliplatin, and docetaxel (FLOT) regimen including 5-fluorouracil, oxaliplatin, and docetaxel.[8] There are no cases of cisplatin and 5-fluorouracil-related IH-H diagnosed with characteristic MRI findings, but a few cases of methotrexate-related IIH have been reported.[9]

Although the aetiology of IIH has not been fully clarified, there are many treatment modalities. Medical treatment aims to decrease CSF production or increase drainage. Weight-loss in combination with medical treatment has favourable effects on headaches and papilloedema.[10] Some surgical methods are used in resistant cases. Treatment is personalised and determined according to the clinical presentation of the patient. The aim is to prevent headaches and vision loss by reducing intracranial pressure.

In conclusion, we observed the development of IIH due to chemotherapeutic drugs in a patient receiving mTCF chemotherapy for metastatic gastric cancer. This case report of IHH secondary to mTCF will contribute to the literature.

PATIENT'S CONSENT:

Informed consent was obtained from the patient

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

ND: Conception or design of the work, acquisition, analysis, and interpretation of the data.
BY: Conception or design of the work.
SS: Drafting the work and critical review for the important intellectual content.
All authors approved the final version of the manuscript to be published.

REFERENCES

1. Gjerris F, Soelberg Sorensen P, Vorstrup S, Paulson OB. Intracranial pressure, conductance to cerebrospinal fluid outflow, and cerebral blood flow in patients with benign intracranial hypertension (pseudotumor cerebri). *Ann Neurol* 1985; **17(2)**:158-62. doi: 10.1002/ana.410170209.
2. Sergott RC, Savino PJ, Bosley TM. Modified optic nerve sheath decompression provides long-term visual improvement for pseudotumor cerebri. *Arch Ophthalmol* 1988; **106(10)**:1384-90. doi: 10.1001/archopht.1988.01060140548021.
3. McCluskey G, Mulholland DA, McCarron P, McCarron MO. Idiopathic intracranial hypertension in the northwest of Northern Ireland: epidemiology and clinical management. *Neuroepidemiology* 2015; **45(1)**:34-9. doi: 10.1159/000435919.
4. Kesler A, Stolovic N, Bluednikov Y, Shohat T. The incidence of idiopathic intracranial hypertension in Israel from 2005 to 2007: Results of a nationwide survey. *Eur J Neurol* 2014; **21(8)**:1055-9. doi: 10.1111/ene.12442.
5. Wall M. The headache profile of idiopathic intracranial hypertension. *Cephalalgia* 1990; **10(6)**:331-5. doi: 10.1046/j.1468-2982.1990.1006331.x.
6. Wall M, George D. Idiopathic intracranial hypertension. A prospective study of 50 patients. *Brain* 1991; **114(Pt 1A)**:155-80. doi: 10.1093/oxfordjournals.brain.a101855.
7. Bono F, Quattrone A. Idiopathic intracranial hypertension without papilloedema in headache sufferers. *Cephalalgia* 2009; **29(5)**:593-4. doi: 10.1111/j.1468-2982. 2008.01765_1.x.
8. Marcelis J, Silberstein SD. Idiopathic intracranial hypertension without papilledema. *Arch Neurol* 1991; **48(4)**:392-9. doi: 10.1001/archneur.1991.00530160060014.
9. Wall M, Corbett JJ. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. *Neurology* 2014; **83(2)**:198-9. doi: 10.1212/01.wnl.0000452039.32455.3e.
10. Skau M, Brennum J, Gjerris F, Jensen R. What is new about idiopathic intracranial hypertension? An updated review of mechanism and treatment. *Cephalalgia* 2006; **26(4)**:384-99. doi: 10.1111/j.1468-2982.2005.01055.x.