wjpmr, 2021,7(6), 62-64

SJIF Impact Factor: 5.922

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Case Study

ISSN 2455-3301

WJPMR

IDIOPATHIC INTRACRANIAL HYPERTENSION IN PREGNANCY: A CASE REPORT

***Dr. F. Kamri, K. Tamim and S. Mchichou**

Department of Gynaecology-Obstetrics, Cancerology and High Risk Pregnancies Unit, Maternity Souissi, Hospital Ibn Sina, Rabat, Morocco.

***Corresponding Author: Dr. F. Kamri**

Department of Gynaecology-Obstetrics, Cancerology and High Risk Pregnancies Unit, Maternity Souissi, Hospital Ibn Sina, Rabat, Morocco.

Article Received on 02/04/2021 **Article Revised on 23/04/2021** **Article Accepted on 13/05/2021**

INTRODUCTION

Described for the first time by Quicke in 1897, benign HTIC, also called idiopathic HTIC or pseudotumour cerebri, is an increase in cerebrospinal fluid (CSF) pressure without clinical, biological or radiological arguments in favour of an intracranial pathology.[1][2] We report the case of a patient who was diagnosed with an idiopathic HTIC during the first trimester of her pregnancy. The objective of this work is to specify the particularities of the management of these patients by focusing on the obstetrical consequences of this pathology.

OBSERVATION

Mrs. S., 25 years old, 38 weeks of amenorrhea (SA) pregnant, with no notable pathological history, notably no history of thrombophilia or systemic disease, G2P1 (first pregnancy, vaginal delivery 6 years ago with good psychomotor development of the baby). Describing the sudden onset at 15 days of age of an HTIC syndrome consisting of headache, vomiting and visual disturbances, without meningeal syndrome and without signs of localization. The MRI (magnetic resonance imaging) of the brain did not find any signs of hydrocephalus, cerebral edema, or intracranial expansive process. The ophthalmological examination showed a bilateral marked alteration of the visual field. The lumbar puncture (LP) noted the figure of 33 cm H2O which allowed to confirm the intracranial hyperpressure, to relieve the patient and to carry out the biological analysis of the CSF which came back normal, thus allowing to assert the diagnosis. Also, a treatment based on Diamox250mg x 3/d. The course of the pregnancy was without anomalies; the morphology and fetal growth were correct.

The patient was admitted at 38SA+6D outside of labor for delivery, without signs of HTIC on clinical examination. Ultrasound examination showed a monofetal pregnancy, positive cardiac activity, cephalic presentation with a biparietal diameter of 94 and an east femoral length of 71. Given the context of idiopathic HTIC engaging the visual prognosis, a caesarean section under spinal anaesthesia was performed, giving birth to a newborn at term of 2950g, of male sex with an Apgar score of 9/10. The postpartum period was normal for the mother and the newborn. An ophthalmological check-up (fundus and visual field) and a thrombophilia assessment were planned. This pathological situation in no way contraindicates a possible subsequent pregnancy, given the low risk of recurrence.

DISCUSSION

Outside of pregnancy, people exposed to benign HTIC are young women, average age 24 years, of childbearing age, and often obese (BMI greater than 30).[3]

The diagnostic criteria advocated by Friedman and Jacobson and found by Peterson and Kelly are as follows.[4][5]

- Subjective symptoms of HTIC (sudden headache, decreased visual acuity, diplopia and vomiting).
- Neurologic signs of HTIC (papilledema, blind spot enlargement, more rarely retinal hemorrhages and exudates, bilateral sixth cranial pair paralysis, visual field amputation).
- CSF pressure increase greater than 25 cm of water.
- Normal CSF composition.
- Absence of hydrocephalus, tumor or parenchymal infection demonstrated on brain imaging.
- Absence of any other identified cause of HTIC.

Benign HTIC is, therefore, a diagnosis of elimination. The pathophysiology of idiopathic HTIC is still poorly understood, whether it is an increase in intracranial blood volume, hypersecretion of CSF by the choroid plexuses, cerebral edema following an increase in the permeability of the cerebral capillaries, which secondarily limits the absorption of CSF by the cerebral convexities, or a primary decrease in CSF absorption.[3] The hypothesis that currently predominates in the literature attributes the increase in CSF pressure to an increase in resistance to passive flow of CSF into the venous circulation via the arachnoid granulations, through an increase in venous pressure.[4] This venous hyperpressure of still poorly

determined etiology could, in fact, be due to an anatomical anomaly of the venous sinuses such as giant arachnoid granulations or congenital stenosis. In our second observation, there was agenesis of a lateral sinus. In fact, with the progress of nuclear magnetic resonance explorations, in particular thanks to three-dimensional venography, the "idiopathic" nature of benign HTIC is less and less frequent. Thus, if in their two successive series of 38 cases, then of 65 cases, Glueck et al. had found respectively only three and one aspect of cerebral venous sinus thrombosis, a recent series of 29 cases by Farb et al[9] shows substantial venous stenosis of the cerebral sinuses in 93% of the cases of HTIC.

The diagnosis of idiopathic HTIC related to pregnancy should be evoked in any pregnant woman presenting with a syndrome of HTIC with a normal exhaustive etiological work-up. Indeed, it is a diagnosis of exclusion that will be retained if all the modified Dandy criteria are met.[5]

1) Signs of HTIC (headache, nausea, vomiting, papilledema, visual eclipse).
2) Normal neurological examination except for IV palsy.
3) Elevation of CSF pressure to more than 20cmH2O (more than 25cmHg in obese subject).
4) Normal CSF composition.
5) Neuroimaging showing small symmetrical ventricles and excluding a mass syndrome or any other cause of elevated intracranial pressure (ICP).

The diagnosis in our patient was retained with reference to these diagnostic criteria. Indeed, she presented a syndrome of HTIC, without meningeal syndrome and without signs of focalization on neurological examination. The lumbar puncture allowed to objectify the CSF hyperpressure with a figure of 33 cm H2O and to carry out a cytobacteriological and biochemical analysis which came back normal. The MRI (magnetic resonance imaging) of the brain did not find any signs of hydrocephalus, cerebral edema, or intracranial expansive process.

ICP can be measured either during LP or by invasive methods with intra-parenchymal sensors. In all cases, continuous recording is desirable (histogram over 15-30 min).[1] Imaging of the venous sinuses plays an increasingly important role in the evaluation of benign HTIC because venous return is frequently found to be impaired.[6]

The course of pregnancy does not seem to be influenced by the disease, except in the experience of Huna-Baron and Kupersmith who found two MFIUs out of 16 cases. Acetazolamide should be avoided before 20 days' gestation since a case of sacrococcygeal teratoma has been reported after treatment. Unlike "classic" HTIC, idiopathic HTIC is not associated with the risk of life-threatening involvement. However, the visual prognosis is engaged with risk of permanent blindness making all the gravity of this neurological pathology.[2] In our patient, no obstetrical repercussions were noted. Nevertheless, the repetitive attacks of HTIC were complicated by a marked amputation of the visual field.

This is a therapeutic emergency whose management differs little from that of other types of HTIC and is based on: 1) Hygienic and dietary measures. Indeed, a weight reduction of 35kg is associated with a mean decrease of the ICP of about 19cmH2O. 2) Iterative LP. 3) Initiation of corticosteroid therapy that does not cross the placental barrier, based on prednisone, at a dose of 40-60mg/d. 4) In order to reduce the production of CSF by the choroid plexuses, acetazolamide, unsuccessful in our patient, is prescribed. This drug is compatible with breastfeeding according to the American Academy of Pediatrics. In case of intolerance, it will be replaced by a loop diuretic (furosemide) at a maximum dose of 40 mg three times a day for a limited period of time, given the risk of dehydration.[2][7][8]

In case of failure of these therapies, surgical management will be indicated particularly in malignant forms compromising the visual prognosis, which was the case of our patient, who despite taking acetozolamide continued to present peaks of HTIC compromising considerably her visual prognosis. The surgical treatment consists of a fenestration of the optic nerve sheath with CSF bypass through a lumbo-peritoneal or ventriculo-peritoneal shunt which allows decompression of the optic nerve and improvement of vision in 75% of cases within 3 to 4 weeks. Recently, the placement of a dural sinus stent by endovascular approach has been proposed in some cases of sinus venous stenosis.[9][10]

Surgical treatment is only indicated in patients with intractable headaches, progressive visual deterioration, visual disturbances not responding to medical treatment, or "malignant" HTIC with rapid decline in visual acuity. It consists of fenestration or decompression of the optic nerve, a lumboperitoneal shunt or a ventriculoperitoneal shunt. In fact, the lumboperitoneal shunt improves vision in 75% of patients resistant to medical treatment and the papilledema resolves in two to four weeks. The placement of this shunt must be accompanied by treatment for uterine contractions and fetal heart monitoring. During delivery, instrumental extractions are classically recommended in order to avoid expulsive efforts, but the benefit of a systematic assistance to the release is not formally demonstrated. Epidural or intrathecal anaesthesia is recommended, as general anaesthesia is often risky in these frequently obese patients, because of ventilatory problems.[24,25] Despite the theoretical risk of injury to the lumboperitoneal shunt, locoregional analgesia seems possible in the case of caesarean section.[14] [15] [16]

CONCLUSION

Although rare, idiopathic HTIC should be considered in any pregnant woman presenting with a HTIC syndrome

without obvious etiology. The diagnosis is based on the modified Dandy criteria. It is a medical-surgical emergency. This neuropathy does not affect the evolution of the pregnancy. Delivery by vaginal route is not contraindicated in controlled forms outside of any context of HTIC attack. In the postpartum period, a thrombophilia test should be performed. The risk of recurrence if a new pregnancy is envisaged is low.

SUMMARY

We report the case of a 25-year-old woman whose pregnancy was complicated, from the first trimester, by idiopathic intracranial hypertension (ICHT) with visual repercussions, having benefited from a lumbo-peritoneal shunt without obstetrical consequences. The objective of this work is to specify the characteristics of this rare pathological entity whose physiopathological mechanism is poorly elucidated. It is thought to be related to a defect in cerebrospinal fluid (CSF) reabsorption in the arachnoid granulations. The main risk factors are obesity, polycystic ovary syndrome, thrombophilia and hyper fibrinolysis. The diagnosis is based on the modified Dandy criteria after a negative clinico-biological and radiological investigation. As with "classic" HTIC, the visual prognosis is compromised. However, there is no risk of life-threatening involvement. Furthermore, this disease does not influence the course of the pregnancy. That said, prompt and effective treatment should be instituted to preserve the visual function of these patients.

REFERENCES

1. Chazal J, Klein O. Hypertension intracrânienne bénigne: historique, définition et physiopathologie. Neurochirurgie, 2008.
2. Jacopin-Bruneau L, Gommier B, Pierre F, Boog G. Hypertension intracrânienne bénigne et grossesse - À propos de deux cas. Journal de Gynécologie Obstétrique et Biologie de la Reproduction, 2010.
3. Biousse V, Bousser MG. L'hypertension intracrânienne bénigne, 2001.
4. Friedman DI, Jacobson DM. Diagnostic criteria for idiopathic intracranial hypertension, 2002.
5. Peterson CM, Kelly JV. Pseudotumor cerebri and pregnancy, 1985.
6. Glueck CJ, Iyengar S, Goldenberg N, Smith LS, Wang P. P. Idiopathic intracranial hypertension: associations with coagulation, 2003.
7. Biousse V, Ameri A, Bousser MG. Isolated intracranial hypertension as the only sign of cerebral venous thrombosis, 1999.
8. I, Riss. OEdème papillaire dans l'hypertension intracrânienne idiopathique, 2005.
9. Bracard S, et al. Hypertension intracrânienne bénigne: imagerie et thérapeutiques endovasculaires, 2008.
10. Dhellemmes P, Defoort S, Vinchon M. Hypertension intracrânienne bénigne: place du traitement médical, 2008.
11. Erguig L, Benomar A, et al. Hypertension intracrânienne bénigne - Aspects cliniques et thérapeutiques, 2004.
12. Metellus P, et al. Traitement endovasculaire de l'hypertension intracrânienne bénigne étiqueté « idiopathique » - Analyse de 8 cas consécutifs, 2007.
13. Metellus PH, et al. Traitement endovasculaire d'un syndrome d'hypertension intracrânienne bénigne par mise en place d'un stent dans le sinus latéral: considérations thérapeutiques et physiopathologiques à propos d'un cas, 2005.
14. Tang RA, Dorotheo EU, Schiffman JS, Bahrani HM. Medical and surgical management of idiopathic intracranial hypertension in pregnancy, 2004.
15. Bédard JM, Richardson MG, Wissler RN. Epidural anesthesia in a parturient with a lumboperitoneal shunt, 1999.
16. Shapiro S, Yee R, Brown H. Surgical management of pseudotumor cerebry in pregnancy, 1995.
17. Glueck CJ, Iyengar S, Goldenberg N, Smith LS, Wang P. Idiopathic intracranial hypertension: associations with coagulation disorders and polykystic-ovary syndrome, 2003.
18. Biousse V, Bousser MG. L'hypertension intracrânienne bénigne. Rev Neurol, 2001.
19. Klein O, Joud A, Marchal J-C. Prise en charge de l'hypertension intra-crânienne bénigne : analyse de la série nancéenne, 2008.