World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 6.129

GRAVIDIC ACUTE HEPATIC STEATOSIS: ABOUT TWO CASES

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Article Received on 10/05/2021 **Article Revised on 31/05/2021** **Article Accepted on 20/06/2021**

ABSTRACT

Acute gravid hepatic steatosis is a serious pathology threatening the vital and maternal prognosis. Diagnosisshouldbe made in a woman with nausea and vomiting during the 3rd trimester. Early management improves fetal and maternal prognosis. We report the case of two patients presenting a SHAG, taken care of at the Souissi Maternity, Rabat, where the low pathway is admitted, with clinical and biological monitoring and with management of the complications, and recallthroughthiswork, Pathophysiology, diagnostic criteria and modalities of management of this condition.

KEYWORDS: Steatosis, Renal Insufficiency, Low Way.

INTRODUCTION

Acute hepatic steatosis of pregnancy (AHSP) is a condition of the third trimester of pregnancy, discovered by Sheehan in 1940, which causes acute liver failure. In the majority of cases, manifestations of gestational hypertension are present.[1] SHAG is a condition that should not be overlooked, as early management considerably improves the fetal and maternal prognosis.

We report the case of two patients presenting with SHAG, managed at the Souissi maternity hospital, Rabat, where vaginal delivery is admitted, with clinical and biological monitoring, and with management of complications.

The aim of our work is to clarify the possibility of vaginal delivery in case of hepatic steatosis gravidarum and the therapeutic means for the correction of the various disorders.

OBSERVATIONS

Observation 1

24 year old female patient, no significant pathological history, primigravida, primiparous, admitted for eclampsia on presumed unattended pregnancy at 8 months. Examination on admission found an obnoxious patient, blood pressure: 14/9, positive neurosensory signs such as headache, visual fog, negative fetal heart sounds, negative uterine contractions.

On vaginal touch; a soft median cervix 80% effaced admitting a finger, with mobile cephalic presentation and an intact water sac, Obstetrical ultrasound showed a non-progressive mono-fetal pregnancy, cephalic presentation; Grannum Grade II antero-lateral placenta; a biometry of 28-29 SA.

The initial work-up was Hb 10g/dl platelet 30.000 /mm3 TP 54% natremia 136mEq/l kalemia5.49mEq/l creatinine 23mg/l; urea 0.62g/l SGOT 2149 IU/l SGPT 1094 IU/l PAL 294 IU/l.

The patient was put under clinical and biological surveillance in the obstetric unit: during our surveillance there was a normalisation of the blood pressure figures under magnesium sulphate, a biological assessment was repeated after 48 hours haemoglobin 6.4 g/dl platelet 18.000 /mm3 TP 100% SGOT 1902 UI/L SGPT 991 UI/L; urea 0.99 g/l, creatinine 56,4 mg/l, TP at 100% before The patient was given a dialysis session when her renal failure worsened. dialysis session.

The patient went into labour spontaneously and The patient went into labour spontaneously and the delivery took place vaginally. The postpartum period was simple, with no postpartum haemorrhagic complications, stabilisation of blood pressure figures under nifedipine.

A biological check-up was repeated after 48 hours haemoglobin 6 g/dl, platelet count 30,000/mm 3, a TP of 100%, with a decrease in transaminases ASAT 591,

ALAT 571, urea 0.88g/l, creatinine 46.9mg/l and total bilirubin of 9mg/l.

Observation 2

26 year old female patient, no significant pathological history, G2P1. G1: abortion at 2 months G2: estimated current pregnancy at 8 months referred for gravidic steatosis.

Examination on admission: obnoxious patient, GCS 12, apyretic, mucocutaneous jaundice, blood pressure blood pressure 14/8, positive uterine contractions, fetal heart sounds negative. Touch vaginal: patient fully dilated.

The patient had a vaginal delivery of an IUF. The patient presented with a delivery haemorrhage, requiring delivery, requiring medical management with 40 IU of syntocinon + 5 tablets of misoprostol tablets and one gram of tranexamic acid tranexamic acid, A check-up was requested: haemoglobin 12.6 g/dl; platelet count 81,000/mm3; urea: 0.81g/l; creatinine: 42.19 mg/l ASAT 185UI/L; ALAT 73.5UI/L TP 13%; PAL 741 UI; Total bilirubin: 147mg/l; direct bilirubin 93mg/l; indirect bilirubin 54mg/l; PDF= 119 the patient was dialysed for patient was dialysed for the correction of her renal failure. The patient received 3 grams fibrinogen. Liver ultrasound was in in favour of a steatosis liver with the presence of ascites.

A check-up was repeated after 48 revealed a haemoglobin 11.6 g/dl, platelet count 30,000/mm330,000/mm3; urea 0.78g/l; creatinine 30.5mg/l 30.5mg/l; PT 29%; AST 124 IU; ALT 51 IU; PAL 611i total bilirubin 169mg/l; bilirubin direct:106mg/l; bilirubin indirect 63mg/l; PDF 25.68, the patient received 3 grams fibrinogen. A liver biopsy was performed, The result was in favour of gravid hepatic steatosis. Hepatic steatosis.

DISCUSSION

Acute hepatic steatosis in pregnancy (AHSS) is a medical-obstetrical emergency. The rate of The maternal-fetal mortality rate reaches 20%. The pathophysiology of AHFS is still ambiguous, but there is an ambiguous, but there is a higher incidence in women in women who have a genetic mutation that affects affecting their mitochondrial fatty acid oxidation pathway fatty acid oxidation pathway and who carry a fetus with a deficiency of a long chain-3-hydroxyacyl-coenzymeA dehydrogenase.[2]

Acute hepatic steatosis in pregnancy is a pathology in the third trimester. The most common initial initial symptoms are nausea or vomiting, epigastralgia nausea or vomiting, epigastralgia sometimes simulating a subcapsular haematoma of the liver, or a polyuro-polydipsia.[3]

Jaundice is a late sign, appearing late. Our second patient was icteric with an elevated bilirubin level. Hypertension Arterial hypertension or proteinuria are common. Hepatic encephalopathy is a consequence of of neglect or poor treatment of the disease.[3] Both our cases had high blood pressure on admission admission 14/9 with an eclamptic state. We note the absence of jaundice in our patient with a normal normal blood bilirubin level.

Elevation of creatinine is constant in SHAG. It is due to renal mitochondrial dysfunction. mitochondrial dysfunction. It is indicative of functional renal failure. The mechanism of functional renal failure is mainly functional renal failure is mainly acute tubular necrosis with oliguria secondary to hypovolaemia, itself secondary to the haemorrhagic disorders caused by hepatocellular insufficiency.

The case of our 2 patients who had renal insufficiency from the time of theiradmissions. Both of our patients benefited from dialysis to improve their dialysis to improve their renal function.

Swansea diagnostic criteria for Hepatic Steatosis in Pregnancy.[13]

6 or 7 of these criteria are necessary in the absence of an alternative diagnosis.

- Vomiting
- Abdominal pain
- Polydipsia/polyuria
- Encephalopathy
- Bilirubin elevation (L 14 mmol/l)
- Hypoglycaemia (4 mmol/l)
- Uric acid elevation (L 340 mmol/l)
- Leukocytosis (L 11.106/l)
- Ascites or bright liver on ultrasound
- Elevated transaminases (L 42 IU/l)
- Hyperammonia (L 47 mmol/l)
- Renal failure (creatinine L 150 mmol/l)
- Coagulopathy (PT L 14 s or PTT L 34 s)
- Microvesicular steatosis on liver biopsy

Termination of pregnancy interrupts disease progression.[6] However, in the case of term less than 32 days' gestation, in the absence of FAS, in the There are no signs of severity. (Absence of liver failure, encephalopathy, severe ARF, DIC) and severe ARF, DIC) and under strict maternal-fetal maternal-foetal surveillance; an expectant attitude can be proposed. This allows the administration of corticosteroids to ensure fetal lung maturation. But this attitude is limited in case of worsening of the maternal condition or the appearance of fetal suffering. Our patients presented with fetal death in utero, the fetal prognosis is already engaged at their admission.

As soon as SHAG is diagnosed, uterine evacuation is required. The choice of delivery method depends on the maternal and fetal status. If the patient is in labour and there are no signs of acute fetal distress, vaginal delivery will be accepted. If the woman is not in labour or if there are signs of severity (single or multiple visceral failure,

presence of a retroplacental haematoma, sub capsular haematoma of the liver, respiratory distress or acute fetal distress), an emergency caesarean section is indicated.[3] In our first case, it was an MFIU, with the onset of labour, our attitude was to wait and see, admitting the vaginal delivery with correction of the renal insufficiency and biological monitoring. The patient gave birth by vaginal delivery after 4 days of her admission. The postpartum period was straightforward, with no delivery haemorrhage. Our second patient was fully dilated, the patient delivered vaginally, she presented with a delivery haemorrhage that was controlled by medical treatment.

In case of a term higher than 34 weeks of amenorrhea, the pregnancy will be interrupted, labour will be induced by vaginal delivery unless contraindicated.

During labour, monitoring of maternal and foetal parameters is mandatory.

Rigorous maternal monitoring is maintained for at least 48 hours postpartum, given the possible worsening of visceral damage linked to pre-eclampsia during this period.

For pregnancies between 26-32 weeks of amenorrhoea, this can be delayed under strict maternal-foetal control, with the aim of achieving lung maturity. This requires continuous clinical and biological surveillance with maternal and fetal monitoring and mother-infant resuscitation.[5] The aim of this expectant wait is to limit the complications of prematurity.

The therapeutic means available and the objectives to be achieved in the patient with acute renal failure and pregnancy and pre-eclampsia and renal failure are as follows.

Optimise plasma volume

These patients all have plasma hypovolaemia with the risk of capillary leak syndrome[6] Cautious volume expansion (crystalloid or human albumin) is undertaken under strict tolerance control Some authors propose to perform this expansion under the control of the measurements obtained by right catheterisation:[6] the filling being modulated according to the pulmonary capillary pressure (objectives Pcap 10-15 cmH2O). Some authors propose to carry out this expansion under control of the measurements obtained by right catheterisation:[6] the filling being modulated according to the pulmonary capillary pressure (objectives Pcap 10-15 cmH2O). The main risk is lesional pulmonary oedema and/or massive pleural effusion leading to potentially dangerous hypoxia for the foetus. If hypoxia is present, stopping volume expansion, oxygen therapy and non-invasive ventilation will usually overcome the risk of complications.[6]

Control blood pressure.

Lowering blood pressure is a mandatory goal, but an abrupt drop in blood pressure may be responsible for fetal death in utero as a result of placental ischaemia. Labetalol is the oldest product used and its long-term safety in the fetus has been established. However, it is increasingly being replaced by nicardipine, which is also thought to have a cholinergic effect, in both our cases nifedipine, which is also a calcium channel blocker like nicardipine, was used.[7]

Corticosteroid therapy.

Given the risk of preterm delivery, a short course of corticosteroids[5] is given for 24-48 hours to accelerate fetal lung maturation, reduce neonatal mortality, and reduce the risk of neonatal respiratory distress and intraventricular haemorrhage in the newborn. Betamethasone or dexamethasone is usually used in two 12 mg intramuscular injections 12 hours apart.

Magnesium sulphate.

In the Anglo-Saxon countries and on the basis of recent prospective studies confirming the appropriateness of these practices[8,9], intravenous magnesium sulphate (loading dose 2-4 g, maintenance 1-3 mg h-1) makes it possible to reduce the risk of convulsions and perhaps delay the date of delivery by its tocolytic action. Its use in association with calcium channel blockers is not recommended because of an increased risk of complications linked to magnesium sulphate (alveolar hypoventilation).

Its value in women with renal insufficiency has not been demonstrated. It is contraindicated in cases of renal insufficiency as there is a risk of overload.+

Correction of haemostasis disorders.

Platelet transfusions are only considered in cases of bleeding or caesarean section.

If the platelet count is below 30,000 ml-l. In cases of DIC associated with delivery haemorrhage, in addition to packed red blood cells, fibrinogen and antithrombin III may be required.[10]

Indications for dialysis[11]

Dialysis is considered in cases of persistent oligo-anuric renal failure or life-threatening fluid and electrolyte disorders after fetal extraction.

CONCLUSION

SHAG is a rare disease that can lead to hepatocellular failure and death of both mother and child if not diagnosed in time. The main treatment is uterine evacuation; vaginal delivery may be acceptable depending on maternal and fetal conditions. Maternal complications are fatal, their management involves the collaboration of gynaecologists and obstetricians and the Reanimators.

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