



SUBCAPSULAR RENAL HEMATOMA COMPLICATING A SEVERE PREECLAMPSIA: CLINICAL CASE AND LITERARY REVIEW

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ABSTRACT

Isolated subcapsular renal hematoma complicating a severe preeclampsia is a rare condition, to date, only six cases have been reported. Imaging (ultrasound or tomodesitometry examination) plays an important role in detecting this complication. We reported a 30 years old, at 28 weeks LMP presenting a blood pressure surge without any others symptoms and she was found to have an isolated subscapular hematoma in the right kidney on ultrasonography. Finding was confirmed on tomodesitometry examination.

INTRODUCTION

Subcapsular hepatic and renal hematomas are rare but potentially life-threatening complications of pregnancy associated with preeclampsia or the HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count)(1). Therefore, prompt laboratory and radiologic evaluations are essential and may reduce the associated morbidity and mortality. We report the case of a subcapsular renal Hematoma associated to HELLP syndrome, complication very rarely described.

MATERIALS AND METHODS

Observation

Patient female, 30 years, 3rd gravity, 3rd parity, 2 living children with a spontaneous miscarriage in week 20 LMP without any particular medical history (not known as carrier of nephropathy or systemic diseases).

At 28 weeks LMP, the occurrence of a blood pressure surge (TA =179/99 mmHg, proteinuria ++ with urinalysis strips) motivates the admission of the patient in the emergencies. An initial assessment shows HELLP syndrome (platelets at 80.000 elements /ml, hepatic cytolysis AST/ALT at 808/274 UI/L) with no signs of infection (negative urinalysis, normal radiographs, negative blood culture) with normal renal function, then she was admitted into maternity intensive care for her severe preeclampsia. Due to abundant metrorrhagia, a caesarian section was indicated at 29 weeks LMP for maternal rescue discovering a placenta completely covering and giving birth to a stillborn.

The evolution was marked by the installation of a decompensated anuric renal failure in acute pulmonary edema requiring hemodialysis sessions.

In addition, the biological test of 5th day post partum revealed renal impairment with serum creatinine at 113 mg/l (GFR = 4 ml/min), urea level at 1.76, Na⁺ at 136 mmol/l, Ca⁺ at 88 mg/l, Ph⁻ at 38 mg/l, serum protein at 62 g/l, albumin at 23 g/l, a normocytic anemia with normal chromium at 8.2 g/dl, LDH at 508 IU/l, normal

platelets, a correct hemostasis test, C reactive protein at 74 mg/l, erythrocyte sedimentation rate of 60 mm first hour, normal liver and lipid profile, negative hepatic B and C serology, with normal C3 and C4 complementemia, proteinuria of 24 at 0.7 g/24 h, with antinuclear antibodies: negative, Abs Anti DNA: negative, Abs Anti phospholipids positive controlled at 12 weeks interval: Abs β -2glycoprotein IgM (+ 259.6 IU/ml); IgG (+ at 40.96 IU/ml), Ac-anticardiolipin: IgM (-); IgG (-) Acs Anti sm: negative, ANCA: negative.

Radiologically; chest X-ray showed a right pleural effusion with cardiomegaly (CI = 0.7); Cardiac ultrasound: hypokinetic dilated cardiomyopathy with FE 33%, PAH, dry pericardium; Abdominal/Pelvic ultrasound objectifying cardiac liver, right kidney of normal size poorly differentiated with perinephretic collection of 2.5 cm, and left kidney is normal looking with Doppler ultrasound, no abnormalities mainly no intra renal aneurysms. A CT urography was performed which objectified a secretion and excretion bilateral retardation with a subcapsular hematoma of the right kidney measuring 3.2/6.5 cm.

During her hospitalization, the patient had partial seizures whose etiologic showed at the electroencephalogram: paroxysmal fronto-temporal left predominant abnormalities; Puncture of the cerebrospinal fluid: negative; brain Magnetic Resonance Imaging finds an extended thrombosis of the left internal jugular vein with no sign of cerebral thrombophlebitis.

In this clinical and para-clinical picture, we retained the diagnosis of antiphospholipid syndrome at fetal loss, venous thrombosis and positive antibodies anti B-2 glycoprotein.

The therapeutic management was to establish 1 Methyl prednisolone bolus of 15 mg/kg during 3 consecutive days with relay with a corticosteroid dose of 1mg/kg/day, with standard heparin-based anticoagulation treatment with relay by VKA and a symptomatic treatment based on loop diuretics, antihypertensive, antiepileptic, but the evolution was unfavorable with death of the patient after a status epilepticus.



Fig1 Computed tomography urography shows subcapsular hematoma in right kidney measuring 3.2

/ 6.5cm.

RESULT AND DISCUSSION

The occurrence of HELLP syndrome during a severe preeclampsia is not uncommon. It is found in $10 \pm 20\%$ of severe preeclampsia and nearly half of eclampsia cases (2). The originality of this observation is related to the occurrence of spontaneous hemorrhage of the right kidney with no associated liver injury (gross lesions).

Isolated renal hematoma in the context of HELLP syndrome is extremely rare. In most reported cases, it is associated with liver, brain and/or lung damage, highlighting the multi-systemic nature of preeclampsia (3,4).

To date, only six cases of isolated renal hematomas in the context of preeclampsia have been reported. The usual clinical presentation is a picture of acute abdomen with a hemoperitoneum (or hemoretroperitoneum) causing hemodynamic instability occurring in the third trimester of pregnancy. In three cases, the in utero death could not have been avoided and caesarean sections were associated either to a simple evacuation of hematoma or to a radical nephrectomy (5,7). In the two cases reported by Kably and Chikhaoui (8), renal hematoma occurred in a context of eclampsia and maternal rescue caesarean sections allowed to extract viable fetuses. The acute renal failure was inconsistent and rapidly reversible in all cases.

Our case differs from the preceding tables described on several points; it was clinically latent, no back pain or acute abdomen signs, the initial aggravation (severe hypertension) was quickly controlled and renal failure which appears postpartum and remains irreversible after more than 3 weeks postpartum.

The etiologies of SRH (subcapsular renal hematoma) are multiple. Kidney tumors are responsible in 50% of cases (adenocarcinoma and angiomyolipoma) (15). In obstetrics, rare tumor cases reported concerns mainly the angiomyolipoma, a hypervascular benign tumor whose failure may be responsible for the peripartum hemorrhage of various severity (10); The high blood pressure was found outside the obstetrical context, as the only incriminated factor during genuine kidney hematoma (11,12). Other etiologies are represented by vascular malformations, hemostasis disorders, tumor-induced intracystic hemorrhages or spontaneous intracystic hemorrhages broken secondarily in subcapsular, and finally necrotizing angiitis represented mainly by Polyarteritis nodosa (PAN) in connection with the rupture of microaneurysms (1,14,15,9). Renal impairment in the PAN is very common. The PAN is responsible for a fibrinoid necrosis of vessels with microaneurysms formation that is likely to rupture, causing a perirenal or subcapsular hematoma (15), in 20% of cases, subcapsular hematoma remains without etiology (15,16).

It is noticeable that the patient has no coagulation disorder. In addition, no medication that interferes with hemostasis was administered during hospitalization.

In despite of the invasive examinations and even after careful histological study of total nephrectomy piece, the Subcapsular renal hematoma remains without obvious causes. Some authors think that a small tumor may be causing the hematoma and it would be infarcted by the pressure practiced by the subcapsular hematoma.

The diagnosis of subcapsular renal hematoma may be suspected by ultrasound when it shows: the hematoma had a fluid character, kidney contacting the collection is easily identified, the edge of the kidney in contacting the collection is flattened (13,14,15,6). However, it is difficult to differentiate a solid mass and a fresh blood collection especially outside of the clinical presentation; subcapsular renal hematoma is more fresh because the more echogenic (6). The performance of the scanner is

significantly greater than the ultrasound. The subcapsular hematoma appears on images without contrast in the same density as the renal parenchyma and confuses with the shadow of the parenchyma that is expanded and deformed, surrounded by fat perinephretic (15). After injection of contrast product, the opacification of the renal parenchyma and the capsule precise topography of the hematoma whose density is not enhanced (15).

CONCLUSION

Subcapsulaire renal hematoma arising in the context of preeclampsia remains exceptional. In addition, the positive diagnosis, imaging is essential to remove a tumor etiology and monitor this complication.

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