Journal Homepage: -www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI:10.21474/IJAR01/14008
DOI URL: http://dx.doi.org/10.21474/IJAR01/14008

RESEARCH ARTICLE

ATYPICALPRESENTATION OF PREECLAMPSIA. ABOUT A CASE

F. Atfi, I. El Abbassi M. Jalal, A. Lamrissi, K. Fichtali and S. Bouhya Maternity El Harouchi Casablanca

Manuscript Info

Manuscript History
Received: 05 November 2021
Final Accepted: 09 December 2021
Published: January 2022

Abstract

3 to 5% of pregnancies are complicated by pre-eclampsia, which remains one of the main causes of fetal-maternal mortality and morbidity worldwide. The rate is higher in Morocco where the lack of prenatal consultation explains why pre-eclampsia is diagnosed at advanced stages. It is also responsible for 10 to 15% of maternal deaths in the Western world, and it remains the second leading cause of maternal death in France after the hemorrhage during delivery. Severe early pre-eclampsia (before 32 weeks of pregnancy) is associated with a risk of maternal mortality 20 times higher than after 37 weeks, and a higher risk of perinatal complications: prematurity, intrauterine growth retardation, premature detachment of the normoinsere placenta and perinatal mortality [3].Its pathophysiology is complex and multifactorial. The identification of biochemical and biophysical markers that point to placental and endothelial dysfunction allows us to improve our practices through new screening tests (4), which make it possible to target at-risk pregnancies, and to initiate treatment early. Currently, the coexistence of arterial hypertension, proteinuria and edema is arbitrary and inconstant. Preeclampsia can occurwithout the clinical data mentioned above or appear before the second half of pregnancy. Its symptoms are variable and reflect multisystem dysfunction [3]. Its development is unpredictable and can be overwhelming. The objective of this article is to report a case of atypical preeclampsia (before week 20 of gestation) associated with a HELLP Syndrome then to analyze the clinical features of atypical forms, differential diagnosis and progress in biochemical markers and biophysics that can aid in diagnosis.

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Introduction:-

3 to 5% of pregnancies are complicated by pre-eclampsia, whichremains one of the main causes of fetal-maternalmortality and morbidityworldwide. The rate ishigher in Moroccowhere the lack of prenatal consultation explainswhypre-eclampsiaisdiagnosedatadvanced stages.

Itisresponsible for 10 to 15% of maternaldeaths in the Western world, and itremains the second leading cause of maternaldeath in France after the hemorrhageduringdelivery. Severeearlypre-eclampsia (before 32 weeks of pregnancy) isassociatedwith a risk of maternalmortality 20 times higherthanafter 37 weeks, and a higherrisk of perinatal complications: prematurity, intrauterinegrowth retardation, prematuredetachment of the normoinsere placenta and perinatalmortality [3].Itspathophysiologyiscomplex and multifactorial. The identification of

Corresponding Author:- F. Atfi

biochemical and biophysical markers that point to placental and endothelialdysfunctionallows us to improveour practices through new screening tests (4), whichmakeit possible to targetat-riskpregnancies, and to initiatetreatmentearlypreventive.

Currently, the coexistence of arterial hypertension, proteinuria and edemaisarbitrary and inconstant. Preeclampsiacanstart in the absence of the clinical data mentionedabove or appearbefore the second half of pregnancy.Itssymptoms are variable and reflectmultisystemdysfunction [3]. Itsdevelopmentisunpredictable and canbeoverwhelming. The objective of this article is to report a case of atypicalpreeclampsia (beforeweek 20 of gestation) associatedwith a HELLP Syndrome then to analyze the clinicalfeatures of atypicalforms, differentialdiagnosis and progress in biochemical markers andbiophysicsthatcanaid in diagnosis.

Observation:-

35-year-old patient, fifthprocedure, havinghad 3 fetaldeaths in utero at 5 months of pregnancy and 1 child living vaginally (11 years), with no particularpathologicalhistory, admitted for pre-eclampsiaat 19 WA + 4 days, calculatedfromher last menstrualperiod (09/05/2020), withblood pressure of 170-110 mmHg, headache and sensation of epigastric bar. Moreover, on examination, a closedcervix, with no detectablebleeding. The fetalheart rate was 145 bpm and the biometricswere 19 weeks. An emergency assessmentwasperformed, resulting in hemoglobinat 14 g / dL, 142,000 platelets per mm3, ASAT at 1550IU / L and ALAT at 938IU / L, LDH at 2251, 5.2 mg / L of creatinine, and a total bilirubinat 5.21, withlabstix ++. The patient was put on antihypertensive and magnesium sulfate treatment.Transferred for monitoring to the intensive care unit. The fetuswas male, weighing 290 grams. The evolutionwasmarked by the appearance of thrombocytopeniaat 61000 / mm and improvement in the rest of the balancesheetat 48 hours: LDH at 490, ALAT at 256, ASAT at 55 and PT at 100%. Afterstabilization of herblood pressure under alpha aldopa and nicardipine in SAP and improvement of the thrombocytopenia to 119,000 after a course of corticosteroidtherapy, the patient wastransferred to the postpartum department and declareddischarged on day 5 with a balancedblood pressure under alpha metildopa. Antinuclear, circulating anticoagulant and anticardiolipinantibodieswerenegative. The anti and pro-angiogenicfactorscould not becarried out for lack of means, the urine protein-creatinine ratio wasincreased to 600 mg / g.

Discussion:-

According to the latestcriteriaestablished by the American colleague in gynecology and obstetrics, proteinuriais no longer essential for the diagnosis of preeclampsia. It iscurrentlydefinedby the appearance of arterial hypertension after 20 weeks of amenorrhea, associated with new proteinuria and / or single or multiple organinvolvement [2]. It shouldbe remembered hatarterial hypertension isdefined by blood pressure values ≥ 140/90 mmHg.

Althoughstilldebated, some classifications includeuteroplacentalinsufficiencywithintrauterinegrowth retardation (IUGR) in the definition of preeclampsia [2].

Indeed, the symptoms of preeclampsiacanbeheterogeneous. For example, hypertension and, or proteinuria, are absent in 10 to 15% of Hellp Syndrome and in 20 to 38% of eclampsia.

Facedwith the non-specificity of the symptoms of preeclampsia, biochemical markers have been described to identify patients withtypical manifestations.

Since the clinicalcriteria for definingpreeclampsia (hypertension, proteinuria and edema) are arbitrary, Sibai and Stella proposed the termatypicalpreeclampsiathatencompassesdifferentclinicalentities: [4]

1. Pregnancy hypertension associatedwith one or more of the followingcriteria: severearterial hypertension, microangiopatichemolysis, thrombocytopenialessthan 100,000, or hepaticcytolysiswith ASAT greaterthan or equal to 70 IU / L.
2. Preeclampsiaoccurringbefore 20 weeks of amenorrhea. Described in connectionwithantiphospholipidantibody syndrome, hydatidiform moles and fetalhydrops.
3. Late postpartum preeclampsia, from 48 hours postpartum and up to 4 weeksthereof.
4. Gestationalproteinuriawithoutarterial hypertension associatedwithonly one or more of the followingcriteria: symptoms of preeclampsia, hemolysis, thrombocytopenia, elevatedliver tests.

The management isidentical to that of classicpreeclampsiawhere the choice of therapy (antihypertensive, magnesium sulfate) and the decision to give birth is guided by the presence or absence of signs of severity [2].

Whenthis syndrome appearsbefore the 20th week, it isoftenrelated tohydatidiform moles, complete or partial, and triploidy.

The literaturefindsvery rare cases of preeclampsiabefore 20 weeks of gestation affecting progressive pregnancieswithoutmolardegeneration of the placenta [2].

It has also been described forpatientswithcantiphospholipid syndrome and other cases associatedwithHellp Syndrome [3].

A risk factor alsofoundwasegg donation.

In the case of our patient, preeclampsiawasdiagnosedat 19SA + 4 days. No molardegenerationhas been documented.

In order to rule out the differential diagnoses that couldmimicseverepreeclampsia, an antiphospholipidassessmentwasrequested in front of a history of unexplainedfetaldeath in utero, which came back negative. The autoimmuneworkupalsoruled out systemic lupus erythematosus. The clinical and laboratorysignswerealso not in favor of a thromboticthrombocytopenic purpura or ahemolyticuraemic syndrome (unalteredkidneyfunction).

Once the diagnosis of atypicalpreeclampsia has been made, the pregnancyshouldbeterminatedquickly, after the patient has stabilized. Magnesium sulfate isused to prevent or control aseizure. As was the case withour patient, the clinical and biologicalparameters improve darkedly as soon as the placenta wasremoved, on the 3rd day of Postpartum.

We are currentlytrying to implement new earlydetectionstrategies to bettertarget patients atrisk. UterineArtery Doppler (DAU) is a non-invasive test thatis the recording of blood flow in the uterinearteries. In patients whowilldeveloppreeclampsia, severalstudies have shown an increase in the resistance index and the pulsatility index as well as the presence of a proto-diastolicnotch in the 2nd trimester of pregnancy.

Someauthors have more recently been interested in DAU in the 1st trimester, because the aimis to institute a preventivetreatmentwithaspirin in high-risk patients, whichis more likely to be effective if itisstarted as soon as possible, the end of the 1st trimester.

Thus, according to the study of a cohort of 6015 patients atundeterminedrisk of preeclampsia (61), the mean PI adjusted for gestationalage, ethnicity and BMI, has a detection rate of 41.1 % for preeclampsia and 81.8% for earlypreeclampsia, for a false positive rate of 10%. The predictivevalidity of clinical markers aloneislessthanthat of the combination of clinical markers and velocimetric indices of SAD.

As for the use of maternalbiochemical markers as a screening tool for pre-eclampsia, we cite PAPP-A (Pregnancy-associated plasma protein-A) whichplays an important role in the local proliferativeresponse, in particulartrophoblast invasion. [8].A decreasedlevel of PAPP-A in the 1st trimesterisassociatedwith an increasedrisk of preeclampsia. However, the proportion of subjectswithpreeclampsiawho have a PAPP-A concentration below the 5th percentile isonly 8 to 23% [9], thereforeinsufficient for PAPP-A to beused in isolation for screening.

Recently, severalmodels of combined screening, combiningclinical, biochemical and biophysical (DAU) data, have been reported. Thus, for the screening of earlypreeclampsiafrom the 1st trimester of pregnancy, the following have been combined:

1. Mean PI of uterinearteries, serumPlGFlevel, meanarterial pressure, BMI, ethnicity, familyhistory of preeclampsia, parity and personalhistory of preeclampsia [5].
2. Serumlevel of PAPP-A, PP-13 and mean IP of uterinearteries. [7]
3. Lowest PI of uterinearteries, ethnicity, chronic hypertension, parity and mode of conception and meanarterial pressure. [11]
4. Mean PI of uterinearteries, serum PAPP-A level, parity and history of preeclampsia [9]

However, thesedifferentmodels have not been comparedwitheachother, and the procedure for choosing the best predictive model in the differentstudiesis not always explicit.

Wealso cite in the context of the evaluation of angiogenic markers, Calculation of the fms-liketirosina cinasa-1 / placentalgrowth factor index, has also been describeduseful in the diagnosis of precociouspreeclampsia and Hellp Syndrome, with a sensitivity of 92%. [4]

Conclusion:-

Preeclampsiaisa syndrome groupingtogether a set of non-specificsigns and symptoms and multiple possible etiologies.

Despiterefinement of diagnostic toolsoffered to clinicians, there are stillclinicalpresentationsthatfalloutside the scope of definitions.

Any good clinician must know the atypicalforms in order to initiate correct management, withoutdelay, and thusavoidincreasingmaternal and perinatalmorbidity and mortality. There are currentlybiophysical and biochemical markers, underevaluation, with the aim of providing the clinicianwith an effective tool to identify patients withatypicalpresentationsat an early stage.

Declaration of interest

The authorsdeclarethatthey have no conflict of interest.

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