Case study

PULMONARY EMBOLISM REVEALING MEADOWS SYNDROME

Abstract

Meadows syndrome is a rare entity, defined as systolic heart failure occurring peripartum in the absence of underlying heart disease. Thromboembolic manifestations can complicate this syndrome but are rarely revealing. We report the case of a 30-year-old patient, without a history of cardiac, multiparous, who presented 3 months after delivery, acute dyspnea. Distal pulmonary embolism was objectified by pulmonary angiography. But the final diagnosis after an analysis of clinical, ultrasound and etiological data was peripartum cardiomyopathy complicated by embolism pulmonary. The evolution under medical treatment was hardly favorable with a worsening of systolic function complicated by a new episode of bilateral proximal pulmonary embolism after 1 month in a context of covid 19 infection.

Keywords: : Systolic heart failure; Pulmonary embolism; Dilated cardiomyopathy

1. INTRODUCTION

Meadows syndrome is a rare entity, defined by systolicheartfailureoccurringduring the last month of pregnancyor the first five months post partum in the absence of underlyingheartdisease.A pulmonaryembolism (embolus) is a serious, potentially life-threatening condition. It is due to a blockage in a bloodvessel in the lungs. A pulmonaryembolism (PE) can cause symptomssuch as chest pain or breathlessness. It may have no symptoms and be hard to detect [16].

2. OBSERVATION

Wepresent the observation of patient S. D, aged 30 years, Caucasian, multiparous, with no particularcardiachistory, admitted at 3eme months postpartum to the cardiologydepartment for management of acute dyspnoea.

On admission, the patient reported the notion of exertionaldyspnoea and paroxysmal palpitations.

Clinicalexaminationrevealed a thin, apyreticwoman, polypnic at 30 cycles per minute, tachycardic at 120 beats per minute.

Cardiac auscultation revealed a diffusesystolicmurmur at 4 foci, with a functionalappearance.

Peripheral pulses werepresent and symmetrical, and the calvesweresupplewith a negative Homans sign. There were no signs of right heartfailure. Pulmonary auscultation wasunremarkable.
On the frontal chest X-ray, the left pleural pouchwasblunted and the left middle archwasconvex, but therewas no obviouscardiomegaly (cardiothoracic index =0.5). The electrocardiogramshowed a regular sinus rhythm at 95 cycles per minute, incomplete right bundle-branch block and negativeanteroseptal T waves.
Blood gasmeasurementsshowedhypoxia (81 mm hg) and hypocapnia (31 mm hg)
Biological tests showedanaemia of 9.7g/dl (microcytichypochromia) and hypertriglyceridaemia of 3.3g/l. The platelet count, renalfunction tests and liverfunction tests were correct. Troponinwasnegative.
Given the moderateprobability of pulmonaryembolism (GENEVE score=6) with D-dimer >500ng/dl, pulmonaryangiographywasperformed, showingsub-segmental hypoperfusion (right apical, right anterobasal and leftposterobasal). The diagnosis of distal pulmonaryembolismwasthenaccepted.
Systematicechocardiographyrevealedmoderate dilatation of the leftventricle (telediastolicdiameter 56 mm, LVEDD index 2.8 cm/m²) and reducedsystolicfunction (leftventricularejection fraction = 40%, calculated by Simpson'smethod) with global hypokinesia, grade III diastolicdysfunction: (E/a=3). The right cavitiesweredilated and pulmonaryartery pressure wasestimated at 45mm hg.
The diagnosis of peripartumcardiomyopathy (PPMC) was made on the basis of clinical and echocardiographicfindings, after the aetiologicalwork-up had been rejected
Serology, immunology, thrombophilia.
In addition to treatment of the pulmonaryembolism, the patient wasplaced on fluid restriction and treatedsymptomaticallywithdiuretics, converting enzyme inhibitors and anti-aldosterones.
Progress wasrapidlyfavourable, withdisappearance of dyspnoea and partial improvement in general condition. After 1 month, the patient returned to us with a dramaticpicture of acute dyspnoea, painfulswelling of the left leg, fever and episodes of moderateepistaxis.
Biologicalfindingsincludedelevated D-dimer levels, CRP 160, lymphopenia 550 and positive CRP covid 19.
The angioscanrequestedshowed proximal pulmonaryembolism, and echocardiographyshowedimpairedleftventricularsystolicfunction (ejection fraction = 35%).
Recurrence of pulmonaryembolism on anticoagulant (VKA) in a context of covid 19.
The patient wasdischargedafter 10 days in hospital in a stable haemodynamic state with normalisation of the biologicalinflammatory syndrome and improvement in thrombocytopenia.
The patient wasseenagain 6 weekslater, clinically stable, with no abnormalities in herbiology. Echocardiographyshowedimprovement in LV systolicfunction, with LVEF at 48-50% SB. Her treatmentwasoptimised and a 3-month follow-up appointmentscheduled.

3. DISCUSSION

Peripartumcardiomyopathyis a rare entity first describedin 1971 by Meadows [1] and Demakis [2] as systolicheartfailureoccurringduring the last month of pregnancy or during the first 5 months postpartum withoutunderlyingheartdisease and

withoutobvious cause. In 1999, Judith et al [3] proposedultrasoundcriteria for thisdiagnosis (table 1).

Table 1 : criteriadefiningperipartumcardiomyopathy

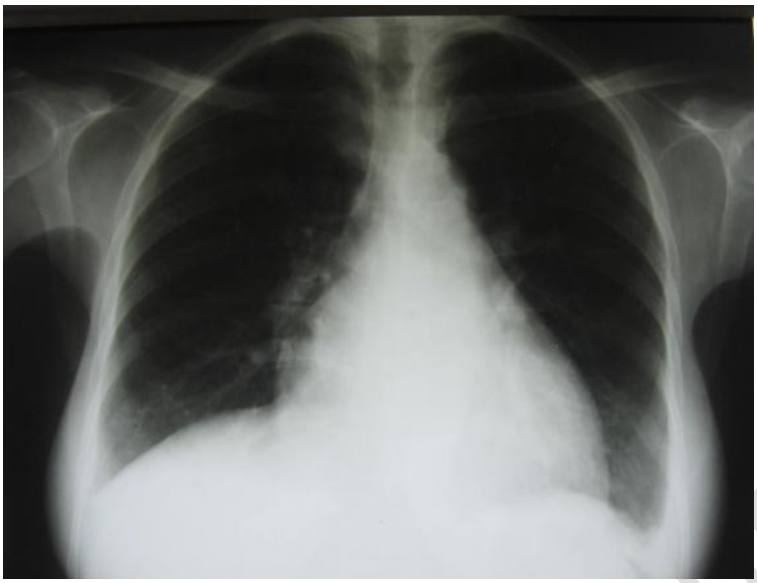
1. Heartfailureappearing - last month of pregnancy - the first five months postpartum 2. No underlyingheartdisease 3. No known cause 4. Echocardiographiccriteria : - EF<45% and/or RF<30 - DTDVG>2.7cm/m2

The incidence of Meadows syndrome varies from 1 in 3,000 to 1 in 15,000 [4] depending on the geographicalregion. It ispredominant in Africa.
In the literature, the preponderance of patients are black, obese, multiparous and elderly (>30 years) [5]. Twinpregnancies, tocolysiswithbetamimetics and gestationaltoxaemia are favourablefactors[6]. The aetiopathogenesis of this syndrome isstillpoorlyunderstood, and severalhypotheses have been established but not confirmed. Viral myocarditis (adenovirus, parvovirus B19, herpes, etc.) isoftenassociatedwith CMPP.
Physiological and haemodynamic changes explain the susceptibility of pregnantwomen and new mothers to viral infection. An immunologicalorigin has also been suggested by the production of autoantibodies as a result of the passage of foetalcells.
in the motherduringpregnancy or the release into the blood of myosin, actin and othermetabolitessecondary to the degradation of tropocollagenduringdelivery. Otherhypotheses have also been put forward, such as nutritionaldisorders and mitochondrial mutation [7]. The usualclinicalpictureis one of acute onsetleft-sided or congestive heartfailure in the form of acute pulmonaryoedema or progressive onsetlimited to dyspnoeaworsening over time, coupledwith an oftenlatediagnosissincedyspnoea and oedema of the lowerlimbs can beseenduring a normal pregnancy.
Thromboembolic complications are not uncommon in CMPP (7 to 50% depending on the series) but rarelyreveal the syndrome, as in the case of our patient, and mayeven lead to misdiagnosis if the signs of heartfailure are not obvious. In fact, on the one hand, thereis a state of hypercoagulabilityduringpregnancywhichpersists for severalmonthsafterdelivery, and on the other hand, the dilation of the heartchamberswillberesponsible for bloodstasis and consequentlythrombi formation. The management of these accidents in cases of CMPP does not differfromthat for otheraetiologies.
The electrocardiogramusually shows sinus tachycardia, but atrial arrhythmiaiscommon [4].
The chest X-ray shows cardiomegaly.

Transthoracicechocardiographyis the gold standard for diagnosing CMPP. It shows dilatation of the leftventriclewithimpairedcontractility and rules out anyunrecognisedunderlyingheartdisease.
The treatment of CMPP issimilar to that of cardiomyopathy of otheraetiologies, but certain drugs, which are of major interest, are contraindicated in the event of pregnancy or breast-feeding.
The aim of treatmentis to reducepreload and afterload and improvemyocardial contraction. Converting enzyme inhibitors are the most effective in reducingafterload, but are contraindicated in pregnantwomen [8] (foetalrenalinsufficiency, oligohydramnios, bone malformation, etc.). In this case, amlodipine or a combination of hydralazine and nitrate derivativesmaybeused [4]. Nitrates and diuretics can beused to reducepreload, takingintoaccount the risk of dehydration.
In severeforms, intravenoustreatmentisinstituted (nitrate derivatives and diuretics). Dopamine and dobutamine have a positive inotropiceffect.
Termination of pregnancymaysometimesbejustified if heartfailureworsensdespite optimal medicaltreatment [9].
The total duration of this long-termmedicaltreatmentis not welldefined.
It isguided by ultrasound monitoring data.
Someauthors have reported the efficacy of immunosuppressive therapy in patients whoremainsymptomaticdespite optimal medicaltreatment and in whommyocarditis has been proven by endomyocardialbiopsy [7].
Effective anticoagulation has been indicated by some teams when the ejection fraction islessthan 35%. However, antivitamin K shouldbeavoidedduring the last month of pregnancy (risk of haemorrhage) and during the postpartum period (breast-feeding)[10].
Cardiac transplantation is the last alternative when the patient remainssymptomatic (dyspnoea stage III-IV NYHA) under maximum medicaltreatment.
In the past, the outcomewasconsidered to befavourable in 50% of cases, with a mortality rate of up to 85% [11]. In fact, new publications report a mortality rate of mortality rate of 30% and total recovery of systolicfunction in 75% of patients [12]. This isprobably due to earlydiagnosisfollowing the introduction of ultrasoundcriteria and to therapeuticprogress.
The elements of poorprognosisseem to be :
Black race, age> 30 years, multiparity, twins, cardiothoracic index > 0.60, shortening fraction < 15%, persistent cardiomegalybeyondtwoweeks [4].
Womenwhoremainsymptomaticbeyondtwoweeks postpartum and thosewhosesymptomsappearafterdeliveryappear to have a more unfavourableoutcome [4].
Within et al [13] have shownthat LVEF<21% and LV end-diastolicdiameter>60mm are predictive of failure to recoversystolicfunction.
The question stillremains:isthere a risk of recurrenceduringsubsequentpregnanciesafter total recovery of cardiacfunction?
In fact, severalstudies have shownthat the risk of recurrenceis 21% if systolicfunction has returned to normal and 44% if LV dysfunctionpersists [14].
It iscurrentlyrecommendedthat a new pregnancyshouldbecontraindicated if ventricularfunctionisreduced. In such cases, close ultrasound monitoring isrequired.
In fact, Lambert et al [15] have shownthatsome patients alter theirsystolicfunctionduringexercisedespitehaving correct function at rest. Similarly, Judith et al [3] have shownthat contractile myocardialreserveislower in womenwith a history of CMPP than in controls. Hence the idea of predicting the risk of recurrence

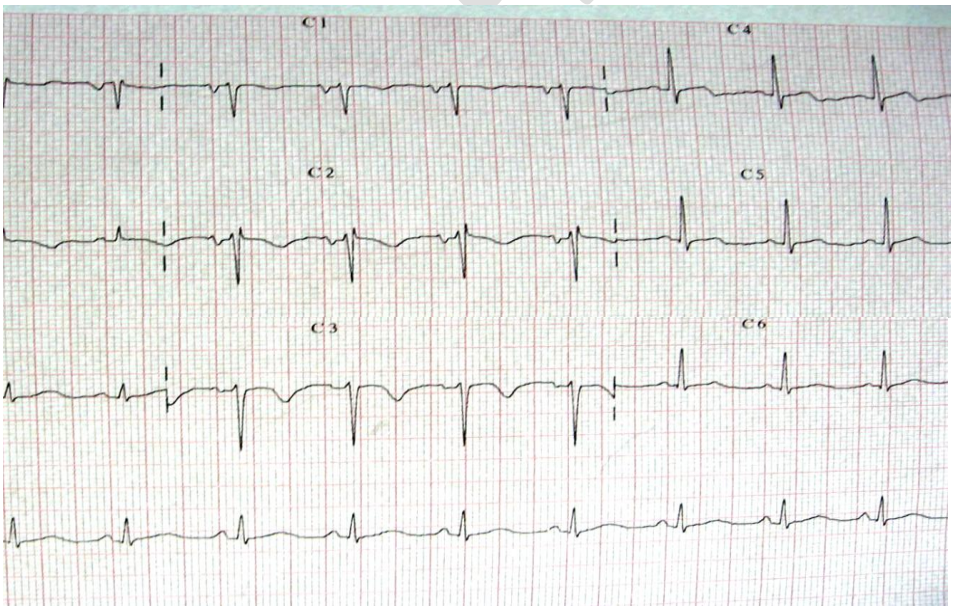
by performing a stress echocardiogram, but furtherstudies are needed to discuss the contribution of thisexamination in monitoring Meadows syndrome.

Fig1: Front chest X-ray:



Blunting of the pleural cul-de-sac on the left medium leftconvexarch

Fig2.ECG: Electricalsigns suggestive of pulmonaryembolism



4. CONCLUSION

CMPP is a rare etiology of dilatedcardiomyopathy, whichiswhythere are not manystudiesrelating to thisentity. The etiopathogenesisremainspoorlyelucidated. Positive diagnosisisfacilitated by echographiccriteria, which are of great help in certain clinicalforms, as in the case of our patient. Untilnow, treatment has been symptomatic, pending a betterunderstanding of the pathophysiology of this syndrome.

Patient consent

The patient gave her consent.

RÉFÉRENCES

[1] Meadows WR. Idiopathic myocardial failure in the last trimester of pregnancy and the puerperium. Circulation 1957;
15: 903–14.
[2] Demakis JG, Rahimtoola SLI, Sutton GC, Meadows NR ,Szanto PB, Tobin JR et al. Natural course of peripartum
cardiomyopathy. Circulation 1971;44: 1053–61.
[3] Hibbard JU, Lindheimer M, Lang RM. A modified definition for peripartum cardiomyopathy and prognosis based
onechocardiography . ObstetGynecol1999; 94(2):311–6.
[4] Fennira S, Demiraj A, Khouaja A, Boujnah MR. La cardiomyopathie du péripartum. Ann CardiolAngeiol2006;
55:271–5.
[5] Brown CS, Bertolet BD. Peripartum cardiomyopathy: a comprehensive review. Am J Obstet Gynecol 1998; 178:409– 14.
[6] Lampert MB, Hibbard J, Weinert L, Briller J, Lindheimer M, Lang RM. Peripartum Heart failure associated with
prolonged tocolytic therapy. Am J obstet Gynecol 1993:493–5.
[7] Lamparter S, Pankuweit S, Maisch B. Clinical and immunologic characteristics in peripartum cardiomyopathy .
Int J cardiol 2007; 118 : 14 -20.
[8] Pryde PG, Thorpe SS, Lamont CA. Angiotensinconverting enzyme inhibitors foetopathy. J Am Soc Nephrol
1993; 3:1575–82.

[9] Merson L, Stecken L, Morel N, Lélias A, Dabadie P Défaillance cardiaque en postpartum Annales Francaises
d'Anesthésie et de Réanimation 2008 ;27 : 731 – 4 .
[10] Kaufman I, Bondy R, Benjamin A. Peripartumcardiomyopathy and thromboembolism;anesthetic
management and clinical course of an obese, diabetic patient. Can J Anaesth 2003;50:161–5.
[11] Homans DC. Peripartum myocarditis. N Engl J Med 1985;312:1432–7.
[12] Felker GM, Jaeger CJ, Klodas E, Thiemann DR, Hare JM,Hruban RH et al. . Myocarditis and longtermsurvival in
peripartum cardiomyopathy. Am Heart J 2000;140:785–91.
[13] Witlin AG, Mabie WC, Sibai BM. Peripartum cardiomyopathy: a longitudinal echocardiographic study. Am J

Obstet Gynecol 1997; 177:1129–32.
[14]Elkayam U, Tummala PP, Rao K, Akhter MW, Karaalp IS, Wani OR.Maternal and fetal outcomes of subsequent
pregnancies in women with peripartum cardiomyopathy. N
Engl J Med 2001 ; 344: 1567 - 71
[15]Lampert MB, Weinert L, Hibbard J, Korcarz C, Zindheimer M, Lang RM.
Contractile reserve in patient with
peripartum cardiomyopathy and recovered left ventricular function. Am J
ObstetGynecol1997;176: 189–95.

16. Carson JL, Kelley MA, Duff A, Weg JG, Fulkerson WJ, Palevsky HI, Schwartz JS, Thompson BT, Popovich Jr J, Hobbins TE, Spera MA. The clinical course of pulmonaryembolism. New England Journal of Medicine. 1992 May 7;326(19):1240-5.