

# PULMONARY EMBOLISM REVEALING MEADOWS SYNDROME : Case study

## 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 systolic heart failure occurring during the last month of pregnancy or the first five months post partum in the absence of underlying heart disease. A pulmonary embolism (embolus) is a serious, potentially life-threatening condition. It is due to a blockage in a blood vessel in the lungs. A pulmonary embolism (PE) can cause symptoms such as chest pain or breathlessness. It may have no symptoms and be hard to detect [16].

## 2. CASE PRESENTATION

We present the observation of patient S. D, aged 30 years, Caucasian, multiparous, with no particular cardiac history, admitted at 3<sup>eme</sup> months postpartum to the cardiology department for management of acute dyspnoea.

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

Clinical examination revealed a thin, afebrile woman, polypnic at 30 cycles per minute, tachycardic at 120 beats per minute.

Cardiac auscultation revealed a diffuse systolic murmur at 4 foci, with a functional appearance.

Peripheral pulses were present and symmetrical, and the calves were supple with a negative Homans sign. There were no signs of right heart failure. Pulmonary auscultation was unremarkable.

On the frontal chest X-ray, the left pleural pouch was blunted and the left middle arch was convex, but there was no obvious cardiomegaly (cardiothoracic index =0.5).

The electrocardiogram showed a regular sinus rhythm at 95 cycles per minute, incomplete right bundle-branch block and negative anteroseptal T waves. Blood gas measurements showed hypoxia (81 mm hg) and hypocapnia (31 mm hg). Biological tests showed anaemia of 9.7g/dl (microcytic hypochromia) and hypertriglyceridaemia of 3.3g/l. The platelet count, renal function tests and liver function tests were correct. Troponin was negative.

Given the moderate probability of pulmonary embolism (GENEVE score=6) with D-dimer >500ng/dl, pulmonary angiography was performed, showing sub-segmental hypoperfusion (right apical, right anterobasal and left posterobasal). The diagnosis of distal pulmonary embolism was then accepted.

Systematic echocardiography revealed moderate dilatation of the left ventricle (telediastolic diameter 56 mm, LVEDD index 2.8 cm/m<sup>2</sup>) and reduced systolic function (left ventricular ejection fraction = 40%, calculated by Simpson's method) with global hypokinesia, grade III diastolic dysfunction: (E/a=3). The right cavities were dilated and pulmonary artery pressure was estimated at 45 mm hg.

The diagnosis of peripartum cardiomyopathy (PPMC) was made on the basis of clinical and echocardiographic findings, after the aetiological work-up had been rejected.

Serology, immunology, thrombophilia.

In addition to treatment of the pulmonary embolism, the patient was placed on fluid restriction and treated symptomatically with diuretics, converting enzyme inhibitors and anti-aldosterones.

Progress was rapidly favourable, with disappearance of dyspnoea and partial improvement in general condition. After 1 month, the patient returned to us with a dramatic picture of acute dyspnoea, painful swelling of the left leg, fever and episodes of moderate epistaxis.

Biological findings included elevated D-dimer levels, CRP 160, lymphopenia 550 and positive CRP covid 19.

The angioscan requested showed proximal pulmonary embolism, and echocardiography showed impaired left ventricular systolic function (ejection fraction = 35%).

Recurrence of pulmonary embolism on anticoagulant (VKA) in a context of covid 19. The patient was discharged after 10 days in hospital in a stable haemodynamic state with normalisation of the biological inflammatory syndrome and improvement in thrombocytopenia.

The patient was seen again 6 weeks later, clinically stable, with no abnormalities in her biology. Echocardiography showed improvement in LV systolic function, with LVEF at 48-50% SB. Her treatment was optimised and a 3-month follow-up appointment scheduled.

### 3. DISCUSSION

Peripartum cardiomyopathy is a rare entity first described in 1971 by Meadows [1] and Demakis [2] as systolic heart failure occurring during the last month of pregnancy or during the first 5 months postpartum without underlying heart disease and without obvious cause. In 1999, Judith et al [3] proposed ultrasound criteria for this diagnosis (table 1).

**Table 1** : criteria defining peripartum cardiomyopathy

1. Heart failure appearing
  - last month of pregnancy
  - the first five months postpartum
2. No underlying heart disease
3. No known cause
4. Echocardiographic criteria :
  - EF < 45% and/or RF < 30
  - DTDVG > 2.7 cm/m<sup>2</sup>

The incidence of Meadows syndrome varies from 1 in 3,000 to 1 in 15,000 [4] depending on the geographical region. It is predominant in Africa. In the literature, the preponderance of patients are black, obese, multiparous and elderly (>30 years) [5]. Twin pregnancies, tocolysis with betamimetics and gestational toxemia are favourable factors [6]. The aetiopathogenesis of this syndrome is still poorly understood, and several hypotheses have been established but not confirmed. Viral myocarditis (adenovirus, parvovirus B19, herpes, etc.) is often associated with CMPP.

Physiological and haemodynamic changes explain the susceptibility of pregnant women and new mothers to viral infection. An immunological origin has also been suggested by the production of autoantibodies as a result of the passage of foetal cells.

in the mother during pregnancy or the release into the blood of myosin, actin and other metabolites secondary to the degradation of tropocollagen during delivery. Other hypotheses have also been put forward, such as nutritional disorders and mitochondrial mutation [7]. The usual clinical picture is one of acute onset left-sided or congestive heart failure in the form of acute pulmonary oedema or progressive onset limited to dyspnoea worsening over time, coupled with an often late diagnosis since dyspnoea and oedema of the lower limbs can be seen during a normal pregnancy.

Thromboembolic complications are not uncommon in CMPP (7 to 50% depending on the series) but rarely reveal the syndrome, as in the case of our patient, and may even lead to misdiagnosis if the signs of heart failure are not obvious. In fact, on the one hand, there is a state of hypercoagulability during pregnancy which persists for several months after delivery, and on the other hand, the dilation of the heart chambers will be responsible for blood stasis and consequently thrombi formation. The management of these accidents in cases of CMPP does not differ from that for other aetiologies.

The electrocardiogram usually shows sinus tachycardia, but atrial arrhythmias are common [4].

The chest X-ray shows cardiomegaly.

Transthoracic echocardiography is the gold standard for diagnosing CMPP. It shows dilatation of the left ventricle with impaired contractility and rules out any unrecognised underlying heart disease.

The treatment of CMPP is similar to that of cardiomyopathy of other aetiologies, but certain drugs, which are of major interest, are contraindicated in the event of pregnancy or breast-feeding.

The aim of treatment is to reduce preload and afterload and improve myocardial contraction. Converting enzyme inhibitors are the most effective in reducing afterload, but are contraindicated in pregnant women [8] (foetal renal insufficiency, oligohydramnios, bone malformation, etc.). In this case, amlodipine or a combination of hydralazine and nitrate derivatives may be used [4]. Nitrates and diuretics can be used to reduce preload, taking into account the risk of dehydration.

In severe forms, intravenous treatment is instituted (nitrate derivatives and diuretics). Dopamine and dobutamine have a positive inotropic effect.

Termination of pregnancy may sometimes be justified if heart failure worsens despite optimal medical treatment [9].

The total duration of this long-term medical treatment is not well defined.

It is guided by ultrasound monitoring data.

Some authors have reported the efficacy of immunosuppressive therapy in patients who remain symptomatic despite optimal medical treatment and in whom myocarditis has been proven by endomyocardial biopsy [7].

Effective anticoagulation has been indicated by some teams when the ejection fraction is less than 35%. However, anti-vitamin K should be avoided during 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 remains symptomatic (dyspnoea stage III-IV NYHA) under maximum medical treatment.

In the past, the outcome was considered to be favourable in 50% of cases, with a mortality rate of up to 85% [11]. In fact, new publications report a mortality rate of 30% and total recovery of systolic function in 75% of patients [12].

This is probably due to early diagnosis following the introduction of ultrasound criteria and to therapeutic progress.

The elements of poor prognosis seem to be :

Black race, age > 30 years, multiparity, twins, cardiothoracic index > 0.60, shortening fraction < 15%, persistent cardiomegaly beyond two weeks [4].

Women who remain symptomatic beyond two weeks postpartum and those whose symptoms appear after delivery appear to have a more unfavourable outcome [4].

With et al [13] have shown that LVEF < 21% and LV end-diastolic diameter > 60mm are predictive of failure to recover systolic function.

The question still remains: is there a risk of

recurrence during subsequent pregnancies after total recovery of cardiac function?

In fact, several studies have shown that the risk of recurrence is 21% if systolic function has returned to normal and 44% if LV dysfunction persists [14].

It is currently recommended that a new pregnancy should be contraindicated if ventricular function is reduced. In such cases, close ultrasound monitoring is required.

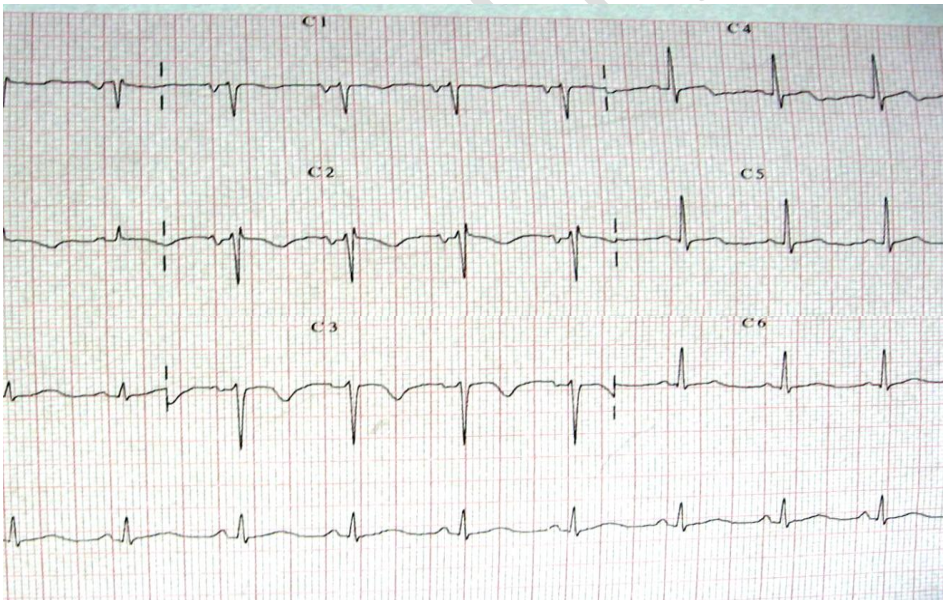
In fact, Lambert et al [15] have shown that some patients alter their systolic function during exercise despite having correct function at rest. Similarly, Judith et al [3] have shown that contractile myocardial reserve is lower in women with a history of CMPP than in controls. Hence the idea of predicting the risk of recurrence by performing a stress echocardiogram, but further studies are needed to discuss the contribution of this examination in monitoring Meadows syndrome.

Fig1: Front chest X-ray:



Blunting of the pleural cul-de-sac on the left medium left convex arch

Fig2.ECG:Electrical signs suggestive of pulmonary embolism



#### 4. CONCLUSION

CMPP is a rare etiology of dilated cardiomyopathy, which is why there are not many studies relating to this entity. The etiopathogenesis remains poorly elucidated. Positive diagnosis is facilitated by echographic criteria, which are of great help in certain clinical forms, as in the case of our patient. Until now, treatment has been

symptomatic, pending a better understanding of the pathophysiology of this syndrome.

#### Ethical Approval:

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

#### Consent

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

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