West Middlesex University Hospital

Date: 14 December 2017
Sources Searched: Embase, Medline, PubMed, Google Scholar.

Peripartum Cardiomyopathy

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1. Unrecognized peripartum cardiomyopathy in Haitian women

Author(s): Fett J.D.; Carraway R.D.; Christie L.G.; Ansari A.A.; Sundstrom J.B.; Murphy J.G.

Source: International Journal of Gynecology and Obstetrics; Aug 2005; vol. 90 (no. 2); p. 161-166

Publication Date: Aug 2005

Publication Type(s): Article

PubMedID: 15961090

Available at International journal of gynaecology and obstetrics: the official organ of the International Federation of Gynaecology and Obstetrics - from Wiley Online Library Science , Technology and Medicine Collection 2017

Abstract: Objective: Haitian women have a high relative incidence of clinical presentation with peripartum cardiomyopathy (PPCM): an incidence estimated at one case per three hundred live births, a ten-fold occurrence compared to American women. Our objective has been to test the hypothesis that some Haitian women may have a forme fruste of PPCM while still without clinical symptoms. Method: A preliminary case-control study was conducted at the Hospital Albert Schweitzer (HAS), Deschapelles, Haiti, in which 25 apparently healthy postpartum women, without cardiovascular symptoms and with a normal cardiovascular clinical examination, were selected from a consecutive list of obstetrical deliveries and screened by echocardiography for left ventricular dysfunction. Result: Four out of 25 patients (16%) had asymptomatic left ventricular dysfunction that subsequently evolved towards either improvement or deterioration. Supporting evidence for the existence of asymptomatic left ventricular dysfunction or forme fruste PPCM is presented. A hypothetical schema of the pathophysiology of PPCM explains how a latent phase of variable duration may exist prior to onset of detectable clinical heart failure. Conclusion: Screening Haitian women during the last month of pregnancy or in the early postpartum period may help to detect asymptomatic left ventricular dysfunction. Early detection and treatment of PPCM in a known high risk population could lead to improvements in maternal and fetal mortality and morbidity. © 2005 International Federation of Gynecology and Obstetrics. Published by Elsevier Ireland Ltd. All rights reserved.

Database: EMBASE

2. A case of peripartum cardiomyopathy: Successful early detection and treatment

Author(s): Koitabashi T.; Inomata T.; Ikeda Y.; Ako J.; Mochizuki J.; Unno N.

Source: Journal of Cardiac Failure; Sep 2016; vol. 22 (no. 9)

Publication Date: Sep 2016

Publication Type(s): Conference Abstract

Abstract: Background: Peripartum cardiomyopathy (PPCM) is a serious pregnancy-associated disorder of unknown etiology. It is difficult to diagnose in early timing because the symptoms of dyspnea and fatigue can result from normal physiologic changes during pregnancy. Moreover, low recognition of PPCM among obstetricians and cardiologists may delay the diagnosis. Case: A 41-year-old woman with pregnancy-induced hypertension as a risk factor for PPCM. At 29 weeks' gestation, the left ventricular ejection fraction (LVEF) was 67%, however, brain natriuretic peptide level was 130 pg/ml without symptoms of heart failure (HF). During careful follow-up, emergent caesarean section was done due to eclampsia and HELLP syndrome at 32 weeks' gestation. We performed a cardiac function evaluation following delivery. LVEF was decreased to 38%, which enabled early initiation of heart failure treatment in a timely fashion. Conclusion: Careful follow-up in cases with risk factors of PPCM is important for early diagnosis and treatment.

Database: EMBASE

3. Isolated Left Ventricular Hypoplasia in a Postpartum Patient.

Author(s): Ding, Wern Yew; Meah, Mohammed; Rao, Archana; Fairbairn, Timothy; Hasleton, Jonathan

Source: The Canadian journal of cardiology; Jun 2016; vol. 32 (no. 6); p. 829

Publication Date: Jun 2016

Publication Type(s): Case Reports Journal Article

PubMedID: 26706664

Abstract:A 22-year-old woman presented with lethargy and shortness of breath at 13 weeks postpartum. She was clinically tachypnoeic with signs of fluid overload. Telemetry revealed 2 different morphologies of nonsustained ventricular tachycardia, associated with chest discomfort. Cardiac imaging demonstrated a truncated, spherical left ventricle (LV) with severe systolic dysfunction and fatty replacement of the LV apex but no evidence of myocardial fibrosis. The right ventricle was elongated wrapping around the LV apex and had moderate systolic impairment. A diagnosis of "isolated LV apical hypoplasia" was made with possible concomitant peripartum cardiomyopathy.

Database: Medline

4. Reverse Apical Ballooning Echocardiographic Pattern in Eclampsia-Related Cardiomyopathy.

Author(s): Gleich, Stephen J; Barbara, David W; Arendt, Katherine W; Rose, Carl H; Blauwet, Lori A

Source: A & A case reports; Jan 2016; vol. 6 (no. 1); p. 6-9

Publication Date: Jan 2016

Publication Type(s): Case Reports Journal Article

PubMedID: 26462164

Available at A & A case reports - from Ovid (LWW Total Access Collection 2015 - Q1 with Neurology)

Abstract: The diagnosis of heart failure during pregnancy has important management implications for the parturient and her fetus. A 19-year-old primigravida developed eclampsia at 29 weeks' gestation. Echocardiography demonstrated normal left ventricular size and ejection fraction of 35% with reverse apical ballooning (reverse takotsubo pattern). Under general anesthesia with invasive monitoring, she underwent urgent cesarean delivery of a preterm infant. Follow-up echocardiography 4 weeks after delivery showed complete normalization of her ejection fraction (56%). This case of eclampsia-related stress-induced cardiomyopathy is a distinct entity from peripartum cardiomyopathy. Using echocardiography, the diagnoses should be differentiated with appropriate management and counseling.

Database: Medline

5. Case study: Takotsubo cardiomyopathy in a postpartum woman

Author(s): Adzham K.N.S.; Zakariah S.Z.; Fuad M.N.S.

Source: Journal of Obstetrics and Gynaecology Research; Oct 2015; vol. 41 ; p. 42-43

Publication Date: Oct 2015

Publication Type(s): Conference Abstract

Available at Journal of Obstetrics and Gynaecology Research - from Wiley Online Library Science , Technology and Medicine Collection 2017

Abstract: Background: Takotsubo cardiomyopathy (TCM) or broken-heart syndrome, a type of non-ischemic cardiomyopathy causes temporary weakening of the cardiac muscle. Emotional stress such as death of loved ones, stressful events and anxiety triggers its onset. It primarily affects postmenopausal woman and is rare in postpartum woman. We report an interesting case of TCM following a sequel of stressful peripartum events in a multiparous woman. Case Description: A 27 year old Para 2 + 1 presented 5 hours post vacuum delivery from a private hospital. She was referred for massive postpartum haemorrhage (PPH) following manual removal of retained placenta with a prolapsed posterior fibroid. Assessment on arrival to our center revealed an incarcerated uterine inversion with grade 4 hypovolemic shock. An emergency laparotomy to manually reduce the uterus was done and B-Lynch sutures inserted for uterine atony. Intraoperatively, she developed massive PPH and disseminated intravascular coagulation (DIVC) requiring transfusion. Postoperatively, her condition was further complicated by cardiogenic shock secondary to a stunned myocardium due to prolonged hypotension. Electrocardiogram (ECG) showed incomplete right bundle branch block, left anterior hemiblock, depressed T wave and ST segment. Echocardiogram (ECHO) showed global left ventricular (LV) hypokinesia with ejection fraction (EF) 26%. Cardiac enzymes were also severely deranged. Coronary angiogram performed showed normal coronary arteries with basal to mid hypokinesia. Thus, she was diagnosed to have Takotsubo cardiomyopathy. She was treated conservatively with triple inotropes in the coronary care unit. 24 hours urine catecholamine was also collected showing normal results. She remained stable and was discharged on day 15 postpartum with resolved ECG changes and ECHO findings of improved LV function with EF 34%, global LV

hypokinesia, akinesia in inferior, posterior and septal wall and resolved PH. CardiacMRI done on day 33 postpartum showed resolved postpartum cardiomyopathy with improved LVEF to 57% and only a small residual area of hypokinesia at the mid-apical anterolateral wall. Two months postpartum, repeat ECHO showed global improvement in cardiac function with LV EF 58%, hypokinesia in septal and anteroseptal wall and resolved PH. Conclusion: TCM is a transient, reversible cardiomyopathy with a good prognosis. It is precipitated by stressful life events. Diagnosis is made by the pathognomonic wall motion abnormalities similarly seen in acute coronary syndromes. TCM is highly preventable via early recognition and accurate diagnosis of peripartum complications which is essential in ensuring timely intervention.

Database: EMBASE

6. A case report of peripartum cardiomyopathy in an asymptomatic female in her third trimester of pregnancy

Author(s): Jayawardena G.R.M.U.G.P.; Guruparan K.; Gamage R.S.; Ratnasiri U.D.P.

Source: BJOG: An International Journal of Obstetrics and Gynaecology; Apr 2015; vol. 122 ; p. 278

Publication Date: Apr 2015

Publication Type(s): Conference Abstract

Abstract: Introduction Peripartum cardiomyopathy (PPCM) is a rare but serious complication of pregnancy with an incidence of 1: 1300 to 1:4000. Several pathogenic factors are suspected to play a role in causation including inflammation, infection, genetics, autoimmune and oxidative stress. Diagnosis is based on the presence of 1) development of heart failure during the last month of delivery or within 5 months postpartum 2) absence of identifiable cause of heart failure 3) absence of recognisable heart disease prior to the last month of pregnancy 4) left ventricular dysfunction determined during echocardiography with ejection fraction is 3/muL with neutrophils at 11.74 103/muL. However she had no clinical signs of infection. Liver and renal function assessment remained within normal limits. Echocardiography showed left ventricular ejection fraction of 30% with global hypokinesia and Grade 2 mitral regurgitation. She remained asymptomatic despite the low systolic function. Her treatment included a loop diuretic and selective b1 receptor blockers. At 37 weeks a planned caesarean section was performed under general anaesthesia and a male baby weighing 2.1 kg was delivered. Conclusion As the incidence of PPCM is low and symptoms are non-specific or absent as in this patient. Therefore diagnosis can often be delayed and may even be missed unless echocardiography is performed. Thus obstetricians should be aware of PPCM and consider it when diagnosing patients with incidental findings of cardiac murmurs to expedite management in a potentially lethal condition.

Database: EMBASE

7. Earlier detection can help avoid many serious complications of peripartum cardiomyopathy

Author(s): Fett J.D.

Source: Future Cardiology; Nov 2013; vol. 9 (no. 6); p. 809-816

Publication Date: Nov 2013

Publication Type(s): Review

PubMedID: 24180539

Abstract: Peripartum cardiomyopathy (PPCM) has a remarkable potential for recovery. It may be within our capability to help almost all women with PPCM not only to survive, but also to completely recover heart function. Time-of-diagnosis left ventricular ejection fraction (LVEF) >=0.35 is associated with better survival rates and higher full recovery rates. Increased mortality, chronic cardiomyopathy, thromboembolic complications and serious ventricular tachyarrhythmias are associated with diagnostic LVEF <0.30. Delays in diagnosis may result in lower LVEF at diagnosis and subsequent lower recovery rates. Greater awareness of the possibility of heart failure developing in previously healthy young women, with no history of heart disease, will contribute to earlier diagnosis, with potentially better preserved heart function. Women of African descent may be at higher risk for poorer outcomes. Recent investigations suggest newer biomarkers may help with earlier detection of PPCM. © 2013 Future Medicine Ltd.

Database: EMBASE

8. Is Tako-tsubo syndrome in the postpartum period a clinical entity different from peripartum cardiomyopathy?

Author(s): Citro, Rodolfo; Giudice, Roberta; Mirra, Marco; Petta, Raffaele; Baldi, Cesare; Bossone, Eduardo; Piscione, Federico

Source: Journal of cardiovascular medicine (Hagerstown, Md.); Aug 2013; vol. 14 (no. 8); p. 568-575

Publication Date: Aug 2013

Publication Type(s): Journal Article Review

PubMedID: 23519095

Abstract:AIMSTo conduct a systematic review of case reports about Tako-tsubo syndrome (TTS) after delivery in order to assess whether TTS in the postpartum period is a peculiar entity or only a variant form of peripartum cardiomyopathy.METHODSWe performed a systematic literature search on the occurrence of TTS after Cesarean section or spontaneous delivery using the scientific literature databases Medline, EMBASE and the Cochrane library. We selected 14 case reports in English. Primary/elective cesarean section or spontaneous delivery; absence of preexisting cardiovascular disease or fetal malformations; identification of diagnostic criteria for TTS; onset of TTS symptoms after delivery were the inclusion criteria.RESULTSFifteen cases were selected. Cesarean section 24 h before the onset of TTS was reported in 13. All patients presented dyspnea or chest pain. The majority had mild troponin elevation, non-ST-segment elevation. Apical ballooning was observed in 60% of cases, midventricular ballooning in 33%, basal ballooning in 7%. Although 13 patients experienced acute cardiac complications (pulmonary edema, cardiogenic shock, cardiac arrest), in all left ventricular systolic function normalized within 13.43 ± 10.96 days.CONCLUSIONWomen in the postpartum period, notably after Cesarean delivery, may represent another new vulnerable group at increased risk for TTS. TTS in the postpartum period should be considered a clinical entity different from peripartum cardiomyopathy with specific clinical, therapeutic and prognostic implications.

Database: Medline

9. Asymptomatic left ventricular dysfunction in puerperal women: An echocardiographic-based study

Author(s): Vettori D.V.; Rohde L.E.; Clausell N.

Source: International Journal of Cardiology; Jun 2011; vol. 149 (no. 3); p. 353-357

Publication Date: Jun 2011

Publication Type(s): Article

PubMedID: 20199817

Abstract: Background: Peripartum cardiomyopathy is a rare but significant cause of maternal morbidity and mortality. Identification of silent forms of ventricular dysfunction associated with the peripartum period is challenging, yet necessary to establish specific counseling and therapeutic measures to prevent progression to overt heart failure. Our aims were to determine the prevalence of asymptomatic left ventricular systolic dysfunction in puerperium and compare its progression with that of cases of peripartum cardiomyopathy occurring in the same study period. Methods: Cross-sectional study conducted from September 2002 to April 2005 to determine by echocardiography the prevalence of asymptomatic ventricular dysfunction in early puerperium and a nested cohort study from November 2007 to January 2008 to obtain clinical and echocardiography follow-up data of positively screened patients. All clinically diagnosed cases of peripartum cardiomyopathy occurring in the same study period were also examined. Results: We screened 1182 puerperal women; ten cases (0.85%) of asymptomatic ventricular dysfunction were detected characterized by either decreased left ventricular systolic function and/or increased end-diastolic diameter. Incidence of peripartum cardiomyopathy was 6 cases/10,866 deliveries (1/1811 live births) in the same period. An echocardiogram-based follow-up study performed after a mean of 4.0 years (2.9-5.2 years), showed significant and similar improvement in parameters of left ventricular function in both groups ($p > 0.05$). Conclusions: Asymptomatic left ventricular dysfunction in puerperal women shows a high prevalence and a pattern of long term echocardiographic changes similar to those found in overt peripartum cardiomyopathy.

Database: EMBASE

10. Peripartum cardiomyopathy may also present as "fulminant right ventricular myocarditis".

Author(s): Fett, James D

Source: The American journal of emergency medicine; Nov 2010; vol. 28 (no. 9); p. 1056

Publication Date: Nov 2010

Publication Type(s): Letter Case Reports Comment

PubMedID: 21036307

Database: Medline

11. Association between HELLP syndrome and peripartum cardiomyopathy presenting as myocardial infarction with normal coronary arteries.

Author(s): Ballo, Piercarlo; Betti, Irene; Mangialavori, Giuseppe; Campatelli, Carlo; Rapisardi, Gherardo; Zuppiroli, Alfredo

Source: European journal of obstetrics, gynecology, and reproductive biology; Jul 2010; vol. 151 (no. 1); p. 110-111

Publication Date: Jul 2010

Publication Type(s): Letter Case Reports

PubMedID: 20444535

Database: Medline

12. Morbidly obese complex obstetrical patient with undiagnosed peripartum cardiomyopathy and development of flash pulmonary edema in PACU

Author(s): Donald R.R.; Crews L.K.

Source: Anesthesia and Analgesia; Mar 2010; vol. 110 (no. 3)

Publication Date: Mar 2010

Publication Type(s): Conference Abstract

Available at Anesthesia and analgesia - from Ovid (LWW Total Access Collection 2015 - Q1 with Neurology)

Abstract: Introduction : Peripartum cardiomyopathy (PPCM) is a rare disorder of unkown cause that occurs during peripartum period. The relationship between heart failure and pregnancy was first recognized in 1870's by Virchow and Porack who noted myocardial degeneration in patients who died in the postpartum period. However, it was first described by Gouley et al as a distinctive form of cardiomyopathy in 1937. Incidence in United States is 1 per 3000 to 4000 live births. Reported mortality rates are between 18 to 56%. A latent form of PPCM has also been described in the literature. We report here a case of latent PPCM in a morbidly obese patient who developed dramatic flash pulmonary edema in postanesthesia care unit (PACU). Case Report : 31-year-old morbidly obese African American female (BMI 53) vaginally delivered twins uneventfully under epidural analgesia. Same epidural was used next day to provide epidural anesthesia for tubal ligation. Intraoperative course was uneventful. Initially patient was stable in PACU, but soon developed dyspnea and suddenly progressed into flash pulmonary edema requiring emergency intubation. Cardiac consult was obtained. Echocardiogram demonstrated severely decreased left ventricular systolic function, LVEF 25%, without other significant findings. After excluding other possible causes, diagnosis of peripartum cardiomyopathy was made. Patient was aggressively treated in ICU and was extubated on second postoperative day. Patient was discharged in stable condition after four days. Discussion : PPCM is a form of dilated cardiomyopathy in which other causes of heart dysfunction are excluded. Identified risk factors include advance maternal age (>30 years), multiparity, multiple gestation, obesity, nutritional disorder, preeclampsia, gestational hypertension and African American race. PPCM is diagnosed by presence of four criteria: (1) development of cardiac failure in the last month of pregnancy or within five months of delivery; (2) absence of an identifiable cause for cardiac failure; (3) absence of recognizable heart disease prior to the last month of pregnancy; and (4) left ventricular systolic dysfunction demonstrated by echocardiogram as depressed ejection fraction. The etiology of PPCM remains unknown. Proposed causes include myocarditis, abnormal immune response to pregnancy, viral infections, maladaptive response to the hemodynamic stresses of pregnancy and autoantibodies against myocardial

proteins. Management goals include preload reduction, afterload reduction and increased inotropy. Anticoagulation may be considered. In postpartum patient ACE-Inhibitors are utilized. Prognosis depends on recovery of LV systolic function, which usually recovers within 6-12 months after delivery. Patients who fail medical management may be considered for heart transplant. Counseling is required concerning the risk of subsequent pregnancy.

Database: EMBASE

13. Delayed-enhanced cardiac magnetic resonance imaging features in peripartum cardiomyopathy.

Author(s): Marmursztejn, J; Vignaux, O; Goffinet, F; Cabanes, L; Duboc, D

Source: International journal of cardiology; Nov 2009; vol. 137 (no. 3); p. e63

Publication Date: Nov 2009

Publication Type(s): Letter Case Reports

PubMedID: 19439378

Abstract: Peripartum cardiomyopathy (PPCM) is a rare disorder in which left ventricular systolic dysfunction and symptoms of heart failure occur in the peripartum period. Although cardiac magnetic resonance (CMR) is largely used for diagnosis and prognosis assessment in cardiomyopathies, its interest in PPCM is unknown. We reported two cases of patients with PPCM who underwent CMR. One patient had no CMR abnormality, while the second patient had several areas of myocardial delayed enhancement (MDE) on CMR images. During follow up, the patient with normal CMR was asymptomatic and had full recovery of cardiac function, whereas the patient with MDE was still symptomatic with persistence of a left ventricular dysfunction. CMR could have prognosis value in PPCM as demonstrated in other cardiomyopathies.

Database: Medline

14. Emergency management of decompensated peripartum cardiomyopathy.

Author(s): Lata, Indu; Gupta, Renu; Sahu, Sandeep; Singh, Harpreet

Source: Journal of emergencies, trauma, and shock; May 2009; vol. 2 (no. 2); p. 124-128

Publication Date: May 2009

Publication Type(s): Journal Article

PubMedID: 19561973

Available at Journal of emergencies, trauma, and shock - from Europe PubMed Central - Open Access

Available at Journal of emergencies, trauma, and shock - from ProQuest (Hospital Premium Collection) - NHS Version

Abstract: Peripartum cardiomyopathy (PPCM) is a rare life-threatening cardiomyopathy of unknown cause that occurs in the peripartum period in previously healthy women.[1] the symptomatic patients should receive standard therapy for heart failure, managed by a multidisciplinary team. The diagnosis of PPCM rests on the echocardiographic identification of new left ventricular systolic dysfunction during a limited period surrounding parturition. Diagnostic criteria include an ejection fraction of less than 45%, fractional shortening of less than 30%, or both, and end-diastolic dimension of greater than 2.7 cm/m(2) body surface-area. This entity presents a diagnostic challenge because many women in the last month of a normal pregnancy experience dyspnea, fatigue, and pedal edema, symptoms identical to early congestive heart failure. There are no specific criteria for

differentiating subtle symptoms of heart failure from normal late pregnancy. Therefore, it is important that a high index of suspicion be maintained to identify the rare case of PPCM as general examination showing symptoms of heart failure with pulmonary edema. PPCM remains a diagnosis of exclusion. No additional specific criteria have been identified to allow distinction between a peripartum patient with new onset heart failure and left ventricular systolic dysfunction as PPCM and another form of dilated cardiomyopathy. Therefore, all other causes of dilated cardiomyopathy with heart failure must be systematically excluded before accepting the designation of PPCM. Recent observations from Haiti[2] suggest that a latent form of PPCM without clinical symptoms might exist. The investigators identified four clinically normal postpartum women with asymptomatic systolic dysfunction on echocardiography, who subsequently either developed clinically detectable dilated cardiomyopathy or improved and completely recovered heart function.

Database: Medline

15. Peripartum cardiomyopathy presenting as a cardiac arrest at induction of anaesthesia for emergency Caesarean section

Author(s): McIndoe A.K.; Hammond E.J.; Babington P.C.B.

Source: British Journal of Anaesthesia; 1995; vol. 75 (no. 1); p. 97-101

Publication Date: 1995

Publication Type(s): Article

PubMedID: 7669479

Available at British Journal of Anaesthesia - from HighWire - Free Full Text

[location] : Patricia Bowen Library and Knowledge Service West Middlesex university Hospital.

Abstract: Peripartum cardiomyopathy is defined as the onset of acute heart failure without demonstrable cause in the last trimester of pregnancy or within the first 6 months after delivery. It occurs in about 1 in 4000 deliveries and is often unrecognized as symptoms of normal pregnancy commonly mimic those of mild heart failure. We describe a previously asymptomatic patient who presented with a cardiac arrest at induction of general anaesthesia for emergency Caesarean section and subsequently developed acute heart failure. This case is unique both in its mode of presentation and the total absence of antecedent symptoms or signs of cardiac disease.

Database: EMBASE

16. Peripartum cardiomyopathy: the value of endomyocardial biopsy in diagnosis, prognostication, and therapy.

Author(s): Fuentes, F; Sybers, H D

Source: Texas Heart Institute journal; 1988; vol. 15 (no. 1); p. 55-58

Publication Date: 1988

Publication Type(s): Journal Article

PubMedID: 15227281

Abstract: We describe the case of a 21-year-old black female who was readmitted postpartum because of increasing exertional dyspnea, orthopnea, pleuritic chest pain, and pedal edema. The patient underwent a successful course of clinical treatment for peripartum cardiomyopathy consisting of a regimen of digoxin, diuretics, captopril, and heparin. The results of an endomyocardial biopsy done at readmission were normal: there was no evidence of inflammation, necrosis, or fibrosis; the endocardium, intramural arterioles, mitochondria, and ultrastructure were normal, as was the amount of glycogen; nuclear chromatin were evenly dispersed; and no antibodies were found. Previous studies have shown that approximately half of patients who suffer from peripartum cardiomyopathy recover, while half develop a more severe form of dilated cardiomyopathy. We venture to speculate that normal endomyocardial biopsy findings during the acute stage of the disease may be predictive of recovery. With more certainty, we propose that histologic findings from material taken during an acute episode can and should guide the course of therapy.

Database: Medline

Strategy 334557

#	Database	Search term	Results
1	Medline	("Peripartum cardiomyopathy").ti,ab	971
2	Medline	("postpartum cardiomyopathy").ti,ab	67
3	Medline	("post partum cardiomyopathy").ti,ab	29
4	Medline	exp CARDIOMYOPATHIES/	84199
5	Medline	exp "POSTPARTUM PERIOD"/	57214
6	Medline	(4 AND 5)	129
7	Medline	(1 OR 2 OR 3 OR 6)	1104
8	Medline	(asymptomatic).ti,ab	132699
9	Medline	(7 AND 8)	23
10	Medline	(without ADJ2 "heart failure").ti,ab	2130
11	Medline	(7 AND 10)	5
12	Medline	(mimic*).ti,ab	160341
13	Medline	(7 AND 12)	9
14	Medline	(rapid* ADJ2 recover*).ti,ab	9694
15	Medline	(7 AND 14)	10
16	Medline	(absence ADJ2 "heart failure").ti,ab	299
17	Medline	(7 AND 16)	4
18	Medline	exp "ASYMPTOMATIC DISEASES"/	4687

19	Medline	(7 AND 18)	1
20	Medline	(normal).ti	175218
21	Medline	(7 AND 20)	3
24	Medline	(normal).ti,ab	1501127
25	Medline	(7 AND 24)	114
26	Medline	(transient).ti,ab	250110
27	Medline	(7 AND 26)	14
28	Medline	exp "DIAGNOSIS, DIFFERENTIAL"/	421239
29	Medline	(7 AND 28)	55
30	EMBASE	("Peripartum cardiomyopathy").ti,ab	1508
31	EMBASE	("postpartum cardiomyopathy").ti,ab	114
32	EMBASE	("post partum cardiomyopathy").ti,ab	47
33	EMBASE	exp "PERIPARTUM CARDIOMYOPATHY"/	1302
34	EMBASE	(30 OR 31 OR 32 OR 33)	1962
35	EMBASE	(absence ADJ2 "heart failure").ti,ab	213
36	EMBASE	(34 AND 35)	5
37	EMBASE	(asymptomatic).ti,ab	188409
38	EMBASE	(34 AND 37)	52
39	EMBASE	(normal).ti	189680
40	EMBASE	(34 AND 39)	7

41	EMBASE	(without ADJ2 "heart failure").ti,ab	2739
42	EMBASE	(34 AND 41)	12
43	EMBASE	(spontaneous* ADJ2 recover*).ti,ab	7967
44	EMBASE	(34 AND 43)	3
45	EMBASE	(rapid* ADJ2 recover*).ti,ab	10580
46	EMBASE	(34 AND 45)	13
47	EMBASE	(normal*).ti,ab	2263521
48	EMBASE	(34 AND 47)	316
49	EMBASE	exp "DIFFERENTIAL DIAGNOSIS"/	339894
50	EMBASE	(34 AND 49)	67
51	EMBASE	(without).ti,ab	2164687
52	EMBASE	(34 AND 51)	235
53	EMBASE	(latent*).ti,ab	79046
54	EMBASE	(34 AND 53)	9
55	EMBASE	exp "LATENT PERIOD"/	46217
56	EMBASE	(34 AND 55)	0
57	EMBASE	(indolent).ti,ab	16246
58	EMBASE	(34 AND 57)	0
59	Medline	(latent*).ti,ab	65842
60	Medline	(7 AND 59)	7
61	EMBASE	(without ADJ2 symptoms).ti,ab	20654

62	EMBASE	(34 AND 61)	7
63	EMBASE	(unrecognised OR unrecognized).ti,ab	34157
64	EMBASE	(34 AND 63)	16
65	EMBASE	(without ADJ2 symptom*).ti,ab	23901
66	EMBASE	(34 AND 65)	8
67	Medline	("Peripartum cardiomyopathy").ti,ab	971
68	Medline	("postpartum cardiomyopathy").ti,ab	67
69	Medline	("post partum cardiomyopathy").ti,ab	29
72	Medline	(without ADJ2 symptom*).ti,ab	20624
73	Medline	(7 AND 72)	3
74	EMBASE	(transient).ti,ab	306026
75	EMBASE	(34 AND 74)	27
76	EMBASE	(silent).ti,ab	44971
77	EMBASE	(34 AND 76)	2
78	Medline	(silent).ti,ab	35031
79	Medline	(7 AND 78)	2
80	EMBASE	(subclinical).ti,ab	51503
81	EMBASE	(34 AND 80)	7
82	Medline	(subclinical).ti,ab	35427
83	Medline	(7 AND 82)	4
84	EMBASE	(without ADJ2 heart).ti,ab	9445

85	EMBASE	(34 AND 84)	19
86	PubMed	("Peripartum cardiomyopathy").ti,ab	982
87	PubMed	("postpartum cardiomyopathy").ti,ab	69
88	PubMed	("post partum cardiomyopathy").ti,ab	30
89	PubMed	(86 OR 87 OR 88)	1059
90	PubMed	(latent OR silent OR subclinical OR "sub clinical").ti,ab	136717
91	PubMed	(89 AND 90)	13
92	PubMed	(asymptomatic).ti,ab	134991
93	PubMed	(89 AND 92)	25
94	PubMed	(lack*).ti,ab	666731
95	PubMed	(89 AND 94)	21
96	EMBASE	(cardiac ADJ2 symptoms).ti,ab	4318
97	EMBASE	(34 AND 96)	14
98	EMBASE	(lack*).ti,ab	859503
99	EMBASE	(34 AND 98)	59
100	EMBASE	("forme fruste").ti,ab	675
101	EMBASE	(34 AND 100)	3
102	Medline	("forme fruste").ti,ab	581
103	EMBASE	exp "EARLY DIAGNOSIS"/	92405
104	EMBASE	(34 AND 103)	57