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Peripartum Cardiomyopathy : A Case Report

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Summary

Peripartum cardiomyopathy (PC), an unknown cause of peripartum heart failure is defined as a cardiomyopathy presented mainly in the first 6 months postpartum, without any evidence of preexisting cardiovascular diseases.

We experienced a 23-year-woman who was hospitalized because of cardiac symptoms three days after the third delivery followed two uneventful deliveries. The PC was diagnosed by the pericardial radiography, sonography and biopsy. Then, management of the disease was carried out through precise medication under Swan-Ganz catheterization. In Japan, the incidences of PC have numbered at least 60 cases so far and this number may increase, contrasting with that in US where approximately 1000 cases a year have been pointed out to be fatal. PC must be taken more seriously by Japanese gynecologists and family doctors.

KEY WORDS

peripartum, cardiomyopathy, case report

Peripartum cardiomyopathy (PC) was first reported by Richew (1849)[1] concerning a case of cardiac failure by partum who had never been ill. In Japan more than 50 cases of PC have been reported since Masugi (1938)[2] described the myocardial changes. The etiology of PC remains uncertain, although a relatively high incidence of myocarditis has been recently reported in patients with PC which is distinctly different from idiopathic dilatated cardiomyopathy. Possibly the mechanism and pathology were complicated by immunocomplex, malnutritiom,stresses, and/or endocrinologically compensated blood conditions during peripartum. There has not yet been uncertified discription or interpretation by now.

Case Report

A 23-year-old, non-smoking female was admitted to the Shinminato Municipal Hospital with orthopnea on Oct. 9, 1996. She had never been ill nor had symptoms of cardiac disease, until the third delivery through Caesarean section this year as the first and the second in 1991 and 1993. Although she had neither complaints nor intoxication before the third birth on Sept., she felt dyspnea when walking three days after delivery and this increased gradually to coughing and dyspnea on Oct. 5. She consulted with her family docter, who introduced her to our hospital because of the remarkable cardiomegaly.

On admission, a physical examination revealed a well-developed, well-nourished woman

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Table 1. Laboratory Findings on Admission

Urinalysis		Blood Chemistry			
Protein	(++)	Total Prot	6.4 g/dl	TSH	1.40 μU/ml
Sugar	(-)	GOT	23 IU/l	Free T_3	3.7 pg/dl
Acetone	(-)	GPT	30 IU/l	Free T_4	1.3 ng/dl
Urobilinogen(n)		LDH	446 IU/l	Cortisol	10.7 μg/dl
Sediment		CPK	89 IU/l	Aldo	< 25 pg/ml
RBC	10-15/HPE	ZTT	12.6 K-U	hANP	75.8 pg/ml
WBC	15-20/HPE	TTT	7.3 K-U	Arterial Blood Gas	
Epithel	4-5/HPE	Na	144 mEq/l	pH	7.482
Cast	1-4/HPE	K	3.8 mEq/l	$PaCO_2$	37.3 torr
Trycomonas	(+)	Cl	108 mEq/l	PaO_2	77.0 torr
Bacteria	(+)	BUN	16.0 mg/dl	HCO_3	26.2 mEq/l
Complete Blood Count		Cr	1.1 mg/dl	BE	5.1 mEq/l
WBC	10200	T-Chol	146 mg/dl	Autoimmunity and Virus	
(Stab 3, Seg 60,		TG	131 mg/dl	ANA	40 X
L 28, M 9 %)		HBs-Ag	(-)	C_3	69 mg/dl
Hb	13.1 g/dl	HCV-Ab	(-)	C_4	31.6 mg/dl
RBC	456 X10^4	TPHA	(-)	Comple	44 U/ml
Hct	38.2 %	CRP	0.4 mg/dl	Coxsackie 8	X
PLT	32.3 X10^4	ESR	38/95 mm, 1h/2h	Echo 7	< 8 X
Ret	5 ‰	FBS	102 mg/dl	Mumps	16 X

lying in the orthopneic position with moderate tachypnea. Her blood pressure was 108/86 mmHg, pulse 104 (regular) and temperature 36.3℃. The heart was enlarged to both the right and the left with percussion. A systolic murmer (Levin-II) in the cardiac apex and dry rales in both lungs were audible. Ascites and leg edema were not noted. The ocular fundi were normal,

Laboratory examinations showed albuminuria with a urinary tract infection shortly after admission. There were slightly high values of collagenous reaction, ANA and Complement (Table 1.), however, serological studies for autoimmune, collagen vascular disease and viral infection were regarded as negative. A chest radiograph revealed cardiomegaly and slight pulmonary edema. An electrocardiogram showed sinus tachycardia, left ventricular hypertrophy with depressed ST segment and inverted T waves. Echocardiography demonstrated the diffuse myocardial damage with mild pericardial effusion and the left ventricular dilatation. The myocardial perfusion study using ^{201}Tl-Cl cardiac pool scanning suggested a congestive cardiomyopathy. Additionally, transvenous endomyocardial biopsies via the right internal jugular vein were taken from the right ventricle. The biopsy specimens showed dilated cardiomyopathy with mild fibrosis and

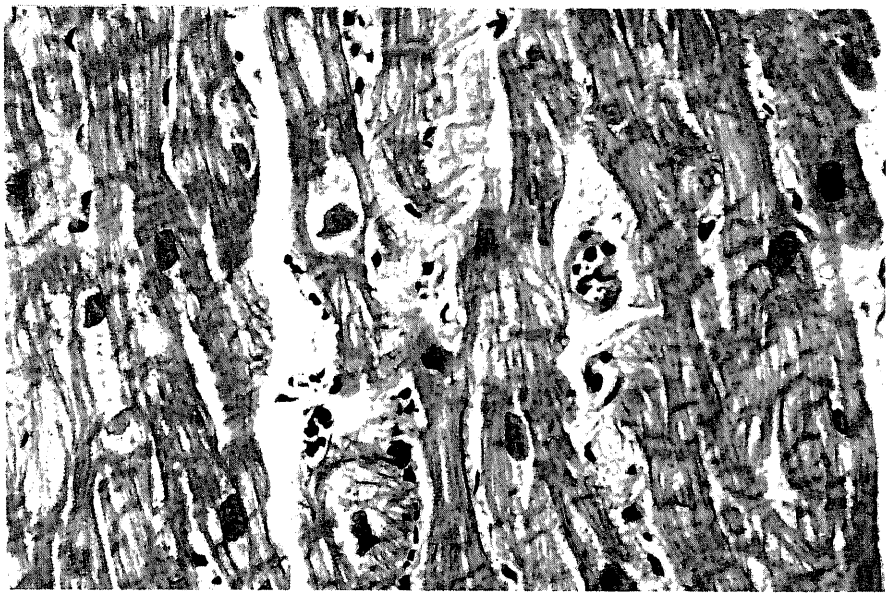

Figure 1. The myocardial biopsy shows a mild fibrosis and nuclear enlargiment in the dilated cardiomyopathy

nuclear enlargement. Myocarditis or hypertrophic cardiomyopathy was negative (Figure 1).

Those laboratory data were all consistent with PC. Intravenous furosemide 40 mg, isosorbide mononitrate and aminobendyl penicillin were commenced. This conservative treatment showed an improvement clinically ; a cardiothoracic ratio from 65.2 to 50.0% in X-P and echocardiography of the left ventricular ejection fraction increasing in two weeks. The patient felt perfect on QOL and was discharged within three weeks and has now completed 24 months of follow-up with stable cardiac and is asymptomatic.

Comment

Demakis (1966)[3] suggested three conditions for a PC as follows ;

1) within the last 3 months of pregnancy to 6 months postpartum, the patient suffered from cardiopathy.
2) no basic heart disease was recognized and an intoxication or hypertension is ruled out.
3) there has been no history of cardiac disease before delivery, no signs and symptoms were recognized during previous peripartum.

Our case can be called "postpartum cardiomyopathy" which is now included in PC and considered to be a dilated cardiomyopathy just as was observed pathologically in our case. There are no specific features of PC as to clinical signs and symptoms, though, symptoms of dyspnea ; 91%, coughing ; 80%, orthopnea ; 65%, and physical signs of cardiomegaly ; 95%, tachycardia ; 84%, and pulmonary rales ; 72% were reported[4] statistically, like mostly seen in our case.

Incidence of PC was 1 : 3000 - 4000 pregnancies in Europe (Veille, et al)[5] or 1 : 6147 in Taiwan (Hsieh)[6] depending on life style, contrarily to 1 : 100 in Nigeria where excessive NaCl is given[7]. Approximately 1000 US women will have PC this year, and for many it would be fatal[8], with a higher incidence in Africa. The etiology is still unknown. Risk factors are considered to be an older age, multiparous and twin birthing and the background conditions are malnutrition, infection, autoimmune complex too more or less degree. Numerous studies have reported a histological evidence of myocarditis of PC[9] in various incidences according to the timing of the biopsy even also in vitro[10]. Recent compelling data suggest PC may be

caused by some form of myocarditis and is an unusual form of dilated cardiomyopathy as shown by our data of echocardiography as well as histology.

The main goal of the treatment is a recovery of cardiac function and to prevention of myocardosis. Generally, diuretics are effective to reduce the amount of volume returning to the heart (preload reduction), nitroglycerine or amlodipine is used to reduce the resistance against which the heart must pump (afterload reduction), and digoxin is picked up as oral inotropic to increase the contractile force of the heart (inotropy)[8]. In case of a hypercoagulable state or immunoreactive, the use of anticoagulant or immunosuppression including steroid might be considered. Depending on complications[12], even cardiac transplantation is applicable. With the initial patient contact, obstetricians and family practitioners must recognize this disease early and rapidly institute the proper medical therapy.

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