CASE REPORT

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Dilated Cardiomyopathy in Pregnancy: A Review of ACEI Exposure and Fetal Risks

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Abstract

Dilated cardiomyopathy (DCM) is a rare disease that can lead to serious cardiac complications, especially during pregnancy. In this study, a case of DCM in an advanced pregnancy with no known history of the disease resulting in spontaneous delivery is presented. Pregnancy in patients with DCM may have adverse outcomes because of increased cardiac output and plasma volume. This case demonstrates that pregnancy carries serious risks, particularly in patients with an ejection fraction <40%. The fetotoxic effects of angiotensin-converting enzyme inhibitors use during pregnancy are emphasized, and the importance of a multidisciplinary approach is highlighted.

Keywords: Dilated cardiomyopathy, pregnancy, angiotensin receptor blocker, premature birth, Down syndrome

INTRODUCTION

Dilated cardiomyopathy (DCM) is characterized by enlarged left ventricle and systolic dysfunction. The approximately 50% increase in plasma volume and cardiac output during pregnancy and the ability to adapt to these dynamic changes are of great clinical importance, particularly in pregnant women with reduced cardiac function. The occurrence of DCM during pregnancy or worsening of preexisting DCM during pregnancy pose a high risk to the mother and fetus. Management of DCM during pregnancy requires great care to protect maternal and fetal health. However, some drugs used during pregnancy may have teratogenic and fetotoxic effects on the fetus. Angiotensin-converting enzyme inhibitors (ACEIs) are commonly used as first-line therapy for DCM, but the fetal toxicity of ACEIs is well known. In this case report, we present a case of DCM treated with ACEIs throughout pregnancy and postpartum follow-up.

CASE REPORT

A 38-year-old female patient with no known medical history and two previous pregnancies was admitted with shortness of breath and orthopnea. Echocardiography revealed an ejection fraction (EF) of 30% and severe functional mitral regurgitation. There was no history of peripartum cardiomyopathy after previous deliveries. Electrocardiography revealed sinus rhythm and tachycardia (Figure 1). Cardiomegaly and pleural effusion, more prominent in the right hemithorax, were observed in the thorax computed tomography scan to exclude additional lung parenchymal pathologies that could explain the current clinical presentation (Figure 2). After patient decompensation was achieved, coronary angiography was performed to exclude ischemic cardiomyopathy, and normal coronary anatomy was observed. She was hospitalized twice with decompensation 5 and 3 months after her first admission. The patient, who did not come for routine checkups in the following period, was

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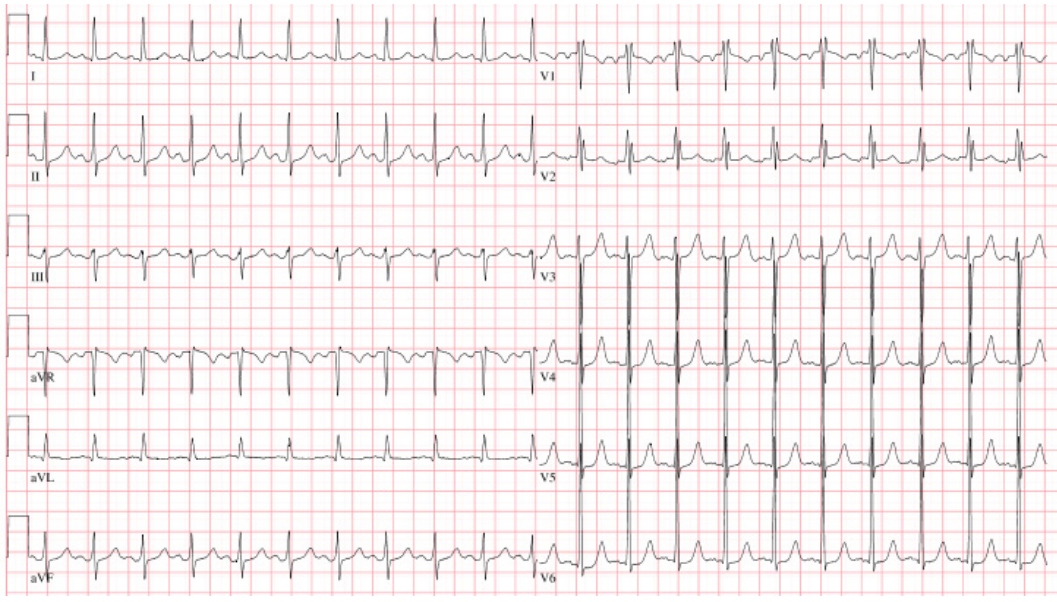

Figure 1. ECG at admission
ECG: Electrocardiogram

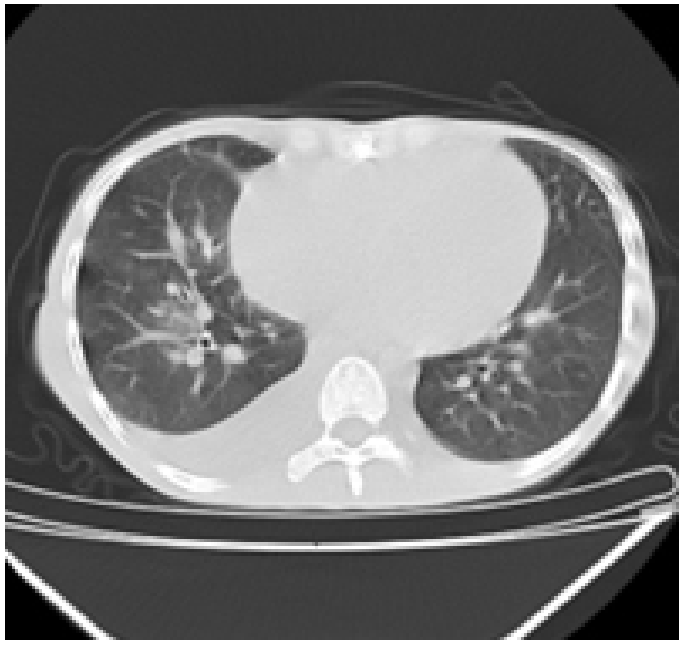

Figure 2. Thoracic CT scan at the time of initial presentation
CT: Computed tomography

admitted to the hospital after a spontaneous birth at home 1 year after his last hospitalization. At the patient's last hospital admission before pregnancy, the pro-BNP level was 6106 ng/L, NYHA 2-3. In the anamnesis, it was learned that the patient was unaware of her pregnancy, did not go to the gynecology and cardiology clinic for a check-up during the pregnancy, and continued to take the medications she had used before pregnancy, ramipril 5 mg, metaprolol 200 mg and furosemide 40 mg, throughout the pregnancy. No pathological findings were observed after birth. In the evaluation performed by the pediatric clinic, it was found that the premature baby was born with Down syndrome, but there was no additional pathology. The patient, for whom cardiac treatment was rearranged, was discharged with recommendations. Two months after discharge, he was hospitalized again due to decompensation. The patient, who did not adequately respond to diuretics and developed increased of, acute renal failure, and sepsis during follow-up, died. Informed consent was obtained from the patient's family for the publication of this case report and any accompanying images.

DISCUSSION

Increased cardiac output and plasma volume due to pregnancy may lead to adverse outcomes in patients with DCM. The clinical consequences of DCM in pregnancy depend on pre-pregnancy cardiac health. It has been shown to be associated with poor outcomes, particularly in patients with EF <40%.[1] Patients with EF >30% tolerated pregnancy well, but the rate of preterm birth was higher. Peripartum cardiovascular events occurred more frequently in patients with high pre-pregnancy BNP levels, advanced diastolic dysfunction, and NYHA 2.[2] ACEIs used as first-line therapy in patients with DCM are contraindicated due to the risks of teratogenicity, fetal renal failure, and neonatal death. Diuretics and selective beta-blockers, such as metoprolol, are relatively safe. Captopril, enalapril, and lisinopril have

been shown to cross the human placenta in pharmacologically significant amounts.[3] Similar results are likely to be observed in other groups of ACEIs. Once in the fetus, most ACEIs are excreted in active forms by the kidneys in urine and may reenter the circulation via the swallowed amniotic fluid.[4] In an animal study conducted with rats and rabbits, the incidence of major malformations was not increased in offspring administered ACEI during organogenesis, whereas decreased uteroplacental flow, low birth weight, hypotension, premature birth, and fetal death were observed.[5] It is known that, unlike exposure to ACEIs in the first trimester, exposure in the second and third trimesters is fetotoxic and may lead to anuria associated with oligohydramnios, limb contractures, craniofacial deformities, and pulmonary hypoplasia.[4] Intrauterine growth retardation, prematurity, patent ductus arteriosus, severe neonatal hypotension, neonatal anuria, and neonatal or fetal death are expected outcomes in this patient group.[6] There is no scientific evidence showing that the use of ACEIs increases the risk of Down syndrome. It is believed that Down syndrome detected in the fetus in this case most likely occurred coincidentally. The commonly known fetotoxic effects of ACEI were not observed in our patient. It has been shown that the risk of malformations in live births is not significantly increased after exposure to ACE inhibitors/ARBs in early pregnancy.[7] This may be due to the patient not using her medications regularly or to the complete discontinuation of ACE inhibitors in advanced pregnancy due to hypotension that occurs during pregnancy. Another issue affecting the prognosis of patients with DCM is social and environmental factors. DCM is associated with high post-discharge mortality, especially in patients from low-income areas with high income inequality.[8] Low education level and socioeconomic status are associated with an increased incidence of sudden cardiac death.[9] The sociocultural level of the patient is of great importance, especially in DCM patients with low education levels, to maintain regular follow-ups, ensure patient compliance with treatment, and provide pre-pregnancy counseling and close management of prenatal care in cases such as possible pregnancies requiring medication revision. Disciplined management with obstetricians, cardiologists, and anesthesia during the birth and postpartum periods is important for achieving optimum outcomes.

CONCLUSION

DCM increases the risk of mortality and morbidity, along with physiological changes during pregnancy. It is important to provide counseling with a multidisciplinary team before pregnancy, during pregnancy, and during the postpartum period to share pregnancy-related risks and make necessary medical treatment revisions. However, to manage this process optimally, patient cooperation and compliance are very important.

Ethics

Informed Consent: Informed consent was obtained from the patient's family for the publication of this case report and any accompanying images.

Authorship Contributions

Surgical and Medical Practices: H.A., Y.A., Concept: N.A., S.T., Design: N.A., H.A., Y.A., S.T., Data Collection or Processing: N.A., Analysis or Interpretation: H.A., Y.A., S.T., Literature Search: H.A., Y.A., S.T., Writing: N.A.

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