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Case Report
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Late postpartum hypertension 8 weeks after delivery: a case report

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Abstract

We present the case of a 34-year-old female who was admitted to the department of cardiology for high blood pressure values accompanied by headache and dizziness. She gave birth to a boy by cesarean section 8 weeks a priori. At the time of admission, she was hemodynamically stable. Blood pressure was over 170/100 mmHg in both upper limbs, with no significant differences. The cardiac auscultation highlighted a systolic murmur (grade 2/6) at the level of the apex. The renal function was altered, but the electrocardiogram was normal. Transthoracic echocardiography revealed non-dilated cardiac chambers, good left and right ventricular global systolic function, and mild mitral regurgitation. When discussing the etiology of late postpartum hypertension, the following were taken into consideration: renal, endocrine, or neurological disorders.

Keywords: postpartum hypertension, pregnancy, non-steroidal anti-inflammatory drugs (NSAIDs).

Introduction

Hypertension affects 6–10% of pregnancies [1]. Postpartum hypertension complicates approximately 2% of pregnancies and is defined as systolic blood pressure (SBP) 140 mmHg or greater and/or diastolic blood pressure (DBP) 90 mmHg or greater on two or more occasions [2]. There are two types of postpartum hypertension: early postpartum hypertension in the first six weeks and late postpartum hypertension after six weeks. The most common cause of the early postpartum hypertension is persistence of hypertension that had been present during pregnancy or pre-existing chronic hypertension. Late post-partum hypertension includes primary hypertension or hypertension secondary to renal disease, endocrine or neurological disorders [1].

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Case report

We report the case of a 34-year-old patient with no significant family medical history who presented

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to the emergency care unit for headache, dizziness, and high blood pressure values. The patient gave birth to a boy by cesarean section 8 weeks prior to the presentation. During pregnancy, the blood pressure values were normal. Home medication included Ibuprofen 600 mg three times a day for abdominal pain. She denies smoking and alcohol consumption.

On presentation, the patient was afebrile, hemodynamically stable, with normal oxygen saturation (97%) and a normal respiratory rate. She had high blood pressure (170/100 mmHg in the left upper limb and 175/103 mmHg in the right upper limb) and a pulse rate of 85 beats/min with a grade 2/6 systolic mitral murmur.

The biological work up revealed normal blood count (WBC=9.730/mm^3, Hb=12.4 g/dL), hepatic cytolysis (alanine transaminase=45 U/L, aspartate transaminase=86 U/L), electrolyte imbalance (Na=133 mmol/L, Cl=97 mmol/L), abnormal renal function (creatinine=1.67 mg/dL, creatinine clearance=40 mL/min/1.73m^2), and dyslipidemia (total cholesterol=226 mg/dL; high-density lipoprotein cholesterol=34 mg/dL, low-density lipoprotein cholesterol=166 mg/dL, triglycerides=181 mg/dL).

The electrocardiogram (ECG) showed sinus rhythm with a heart rate of 58 bpm, intermediate axis, normal QRS complex, ST segment, or T wave (Figure 1). She was admitted to the department of cardiology for further evaluation.

Transthoracic echocardiography revealed non-dilated cardiac chambers, good left and right ventricular global systolic function (left ventricular end-diastolic diameter [LVEDD]=47 mm, left ventricular end-systolic diameter [LVESD]=29 mm, interventricular septum [IVS]=12 mm, left ventricular posterior wall [LVPW]=12 mm, left ventricular ejection fraction [LVEF]=60%, tricuspid annular plane systolic excursion [TAPSE]=21 mm, right ventricular fractional area change [RVFAC]=53%) (Figure 2). Mild mitral regurgitation was observed (Figure 2 A–C). Left ventricular diastolic function was normal (E/A=1.1, septal e' velocity=9 cm/s, E/e'=8.5, tricuspid regurgitation velocity=2.4 m/s) (Figure 3 A–D); an enlarged left atrium (LA volume index=58mL/m^2) can be seen in Figure 3B.

Given the patient's presentation and prior investigations, we considered that she had secondary hypertension, so we continued the investigation to identify the cause. We performed an extended biological workup in order to search for a possible cause for secondary hypertension. The patient had no clinical/paraclinical criteria for acromegaly or pheochromocytoma, and the thyroid workup was within normal limits (TSH= 2.908 µUI/ml, FT4=1.48 ng/dl, Anti-TPO=15.2 UI/ml).

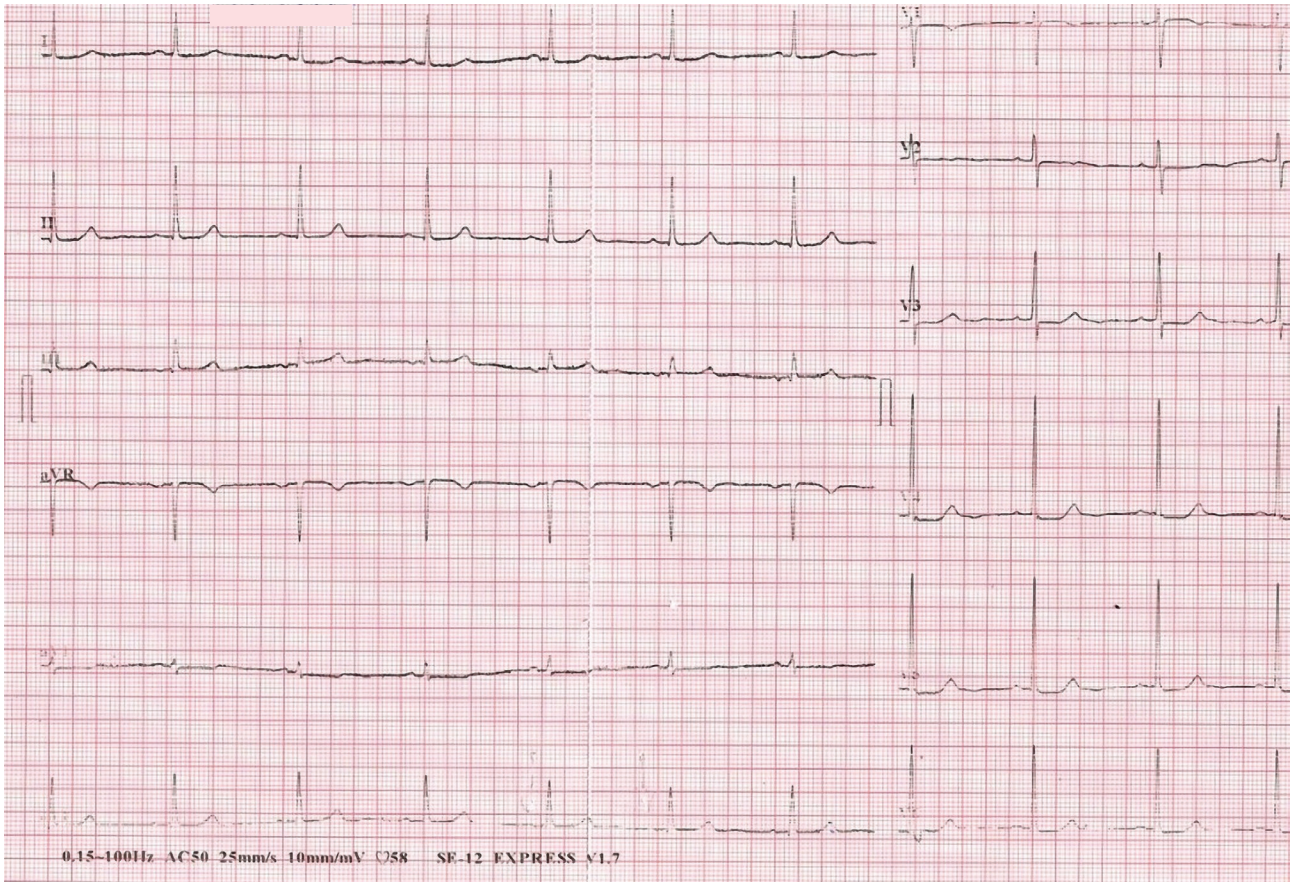

Figure 1. Electrocardiogram showing sinus rhythm with a heart rate of 58 beats per minute.

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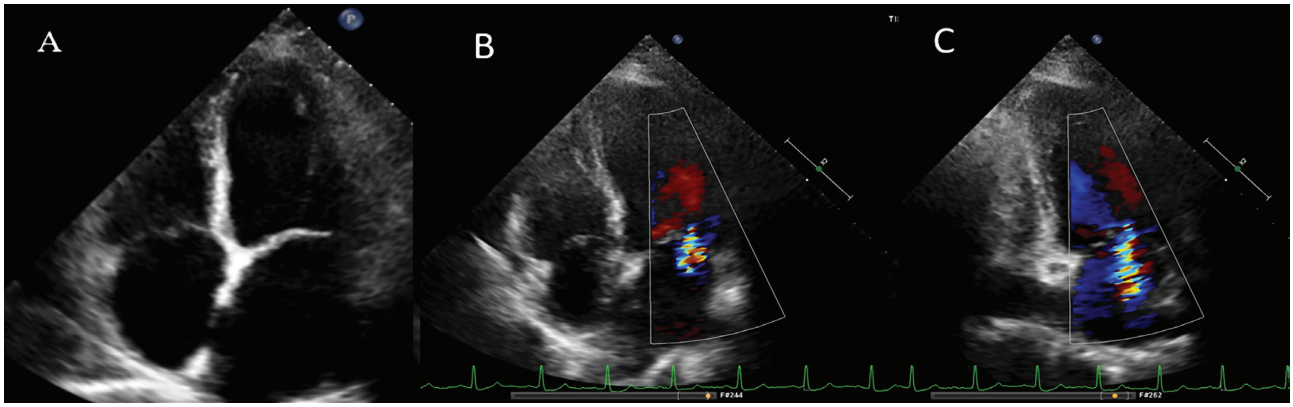

Figure 2. Transthoracic echocardiography. A – Apical 4-chamber view: LV and RV with normal size; B – Apical 4-chamber view, color Doppler; C – Apical 2-chamber view, color Doppler: Mild mitral regurgitation. LV – left ventricle; RV – right ventricle.

Sleep apnea was ruled out by polysomnography. A complete computed tomography scan including cranial, thorax, abdomen, and pelvis was performed to exclude a malignant mass, coarctation of the aorta, or renal artery stenosis.

Therefore, we considered that our patient had late postpartum hypertension secondary to renal disease due to the ingestion of non-steroidal anti-inflammatory drugs (Ibuprofen).

The Ibuprofen administration was stopped. We started treatment with iv Labetalol for a rapid decrease in blood pressure values. Long-term treatment included a calcium channel blocker, Nifedipine 20 mg three times a day. During the first follow-up

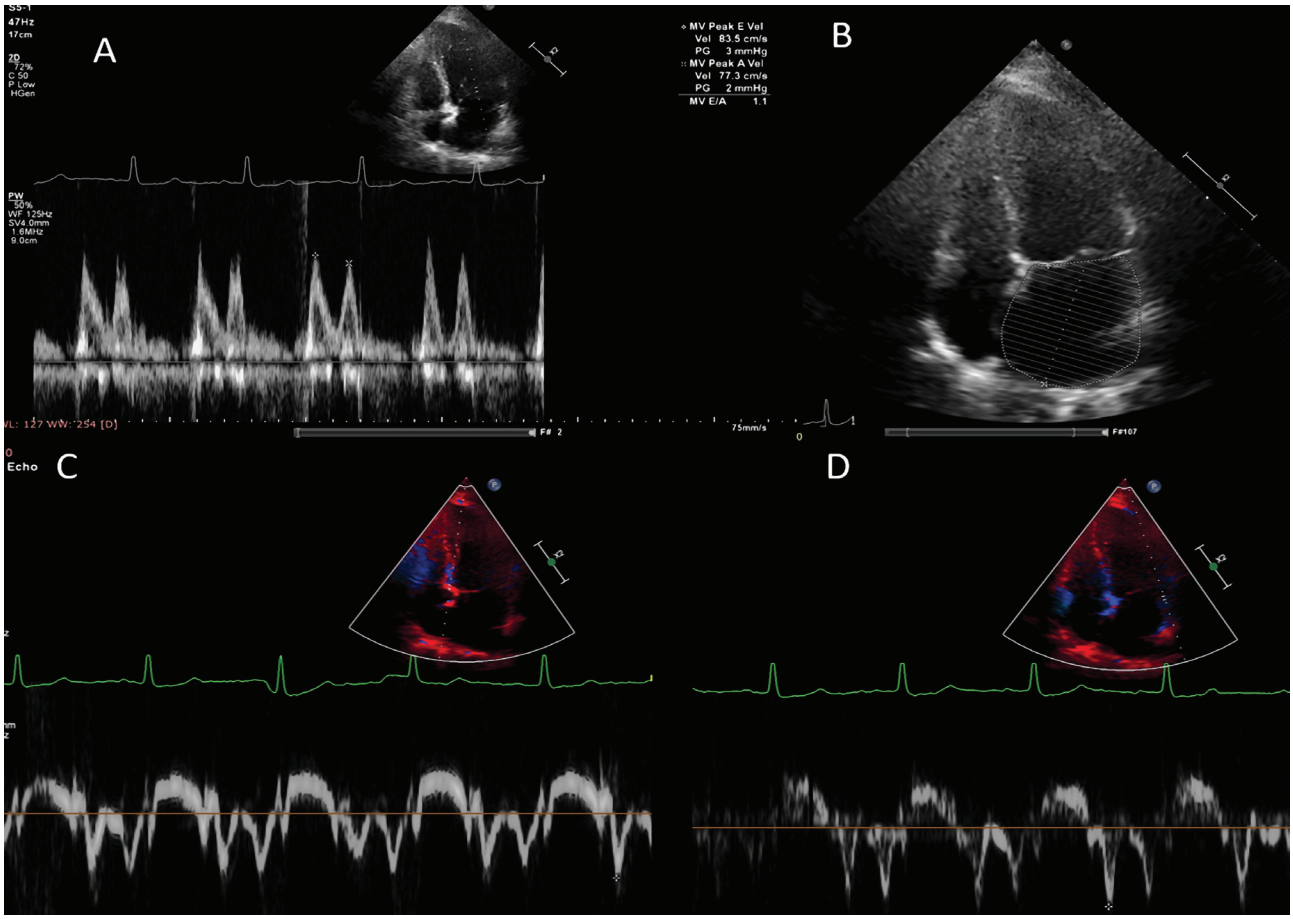

Figure 3. Transthoracic echocardiography. A – Apical 4-chamber view, pulse wave (PW) Doppler: E/A=1.1; B – Apical 4-chamber view: Enlarged left atrium; C, D – Tissue Doppler Imaging: septal e' velocity=9 cm/s, E/e'=8.5.

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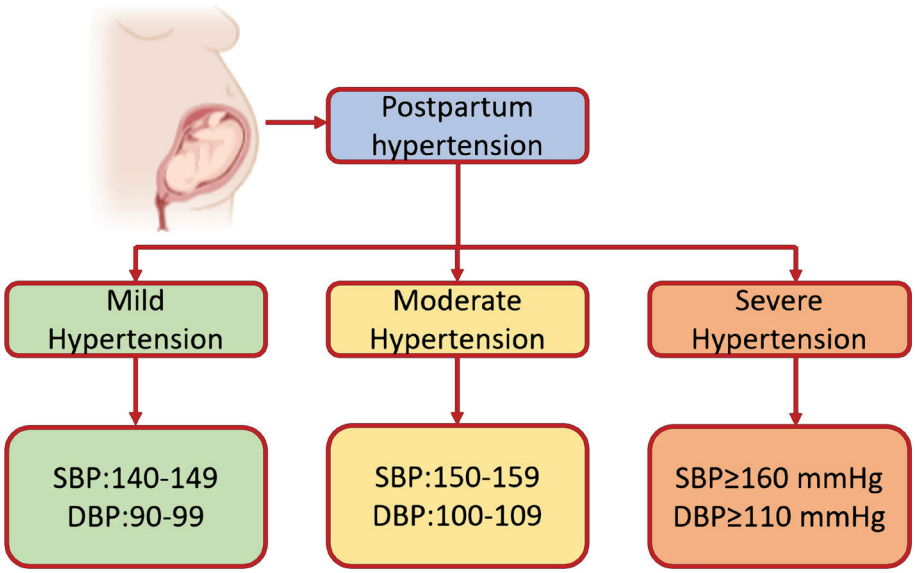

Figure 4. Classification of postpartum hypertension. SBP, systolic blood pressure, DBP, diastolic blood pressure.

visit at one month, the patient achieved good blood pressure control and normal kidney function.

Discussion

Hypertensive disorders of pregnancy (HDP) include preeclampsia, eclampsia, gestational hypertension, chronic hypertension, and postpartum hypertension and affect 10% of pregnancies [3–5]. Gestational hypertension and preeclampsia are thought to account for approximately 86% of the cases of postpartum hypertension [1]. Women with a history of hypertensive disorders during pregnancy have an increased risk of cardiovascular events, including coronary artery disease (CAD), peripheral arterial disease (PAD), and cerebrovascular disease [4]. HDP are responsible for 12% of maternal death [6]. Providers must be aware of the risks associated with postpartum hypertension and educate patients about the symptoms and signs of preeclampsia in the postpartum period [1].

The diagnosis of postpartum hypertension is based on the same criteria for hypertension in gestation. An SBP of 140 to 149 mmHg and a DBP of 90 to 99 mmHg are considered mild hypertension. An SBP of 150 to 159 mmHg and a DBP of 100 to 109 mmHg are considered moderate hypertension. Severe hypertension is SPB over 160 mmHg and DBP over 110 mmHg (Figure 4) [7]. The treatment is

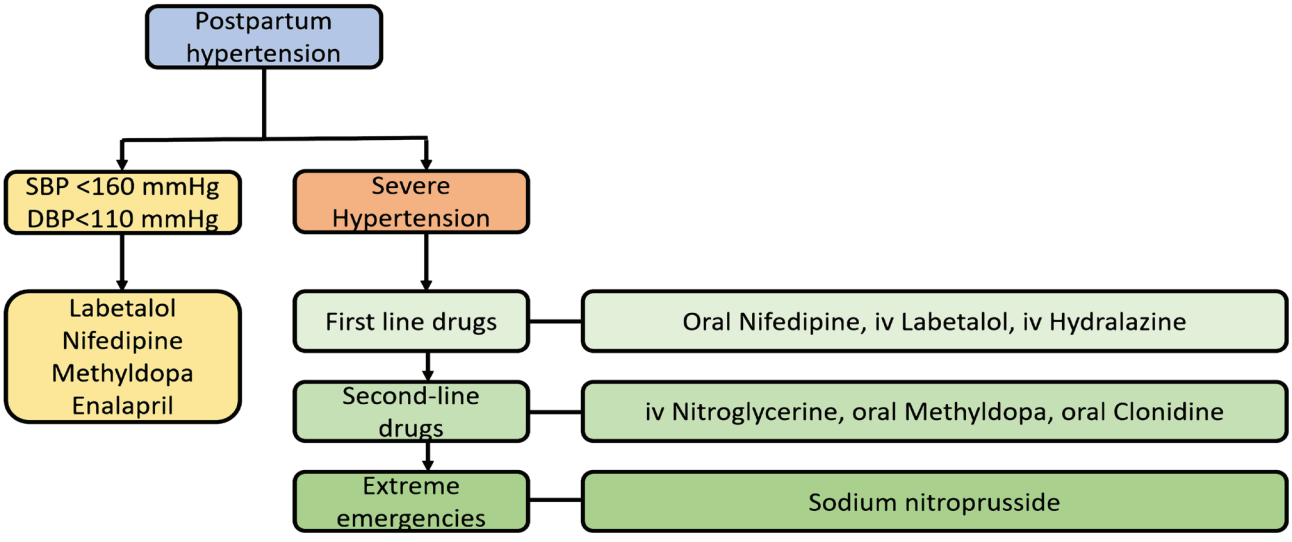

Figure 5. Management of postpartum hypertension. SBP – systolic blood pressure; DBP – diastolic blood pressure, *iv*, intravenous.

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different depending on the severity of hypertension. For severe hypertension, antihypertensive drugs are mandatory. According to the clinical examination, our patient had severe hypertension, and she needed emergency treatment.

Prior to establishing an etiology for postpartum hypertension, management may be required for acute severe elevations in blood pressure (SBP ≥160 mmHg and/or DBP ≥110 mmHg). First-line drugs include oral nifedipine, iv labetalol, or iv hydralazine. Second-line agents are iv nitroglycerine, oral methyldopa, and oral clonidine (Figure 5) [7]. For our patient, we opted for iv labetalol, with adequate control of blood pressure.

Treatment options for postpartum blood pressure values less than 160/110 mmHg are oral nifedipine, oral labetalol, oral methyldopa, or oral enalapril [8]. In a study of 50 women, Sharma *et al.* demonstrated that both labetalol and nifedipine were effective in controlling persistent postpartum hypertension (Figure 5) [9].

After initiating treatment, it is essential to identify a possible cause of secondary hypertension. The practitioners need to perform an extended biological and imaging workup in order to search for a possible cause of hypertension. The patient received an almost complete workup to find the underlying etiologies, and the negative results led to the diagnosis of late postpartum hypertension secondary to renal disease due to the ingestion of non-steroidal anti-inflammatory drugs.

The association between the use of NSAIDs and postpartum hypertension has been controversial in the literature. Future large-scale randomized controlled trials are needed to verify if the administration of NSAIDs to women with HDP during the postpartum period is not significantly associated with elevated blood pressure or the occurrence of severe hypertension.

Conclusion

The presented case highlights late postpartum hypertension secondary to renal disease in a patient who received NSAIDs for 8 weeks after giving birth. Severe postpartum hypertension requires immediate treatment to reduce the rate of complications. It is also necessary to perform an extended biological and imaging workup to search for a possible cause of hypertension.

Conflict of Interest

The authors confirm that there are no conflicts of interest.

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