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Case report

Hypertension in pregnancy: A case discussion

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ABSTRACT

Gestational hypertension and preeclampsia are common disorders during pregnancy, with the majority of cases developing at or near term. The development of mild hypertension or preeclampsia at or near term is associated with minimal maternal and neonatal morbidities. In contrast, the onset of severe gestational hypertension and/or severe preeclampsia before 35 weeks' gestation is associated with significant maternal and perinatal complications. Women with diagnosed gestational hypertension– preeclampsia require close evaluation of maternal and fetal conditions for the duration of pregnancy, and those with severe disease should be managed in-hospital. The decision between delivery and expectant management depends on fetal gestational age, fetal status, and severity of maternal condition at time of evaluation. Expectant management is possible in a select group of women with severe preeclampsia before 32 weeks' gestation. Steroids are effective in reducing neonatal mortality and morbidity when administered to those with severe disease between 24 and 34 weeks' gestation. Magnesium sulfate should be used during labor and for at least 24 hours postpartum to prevent seizures in all women with severe disease. There is an urgent need to conduct randomized trials to determine the efficacy and safety of antihypertensive drugs in women with mild hypertension–preeclampsia. There is also a need to conduct a randomized trial to determine the benefits and risks of magnesium sulfate during labor and postpartum in women with mild preeclampsia.

KEY WORDS: pregnancy induced hypertension, primi gravida mothers, pregnancy

INTRODUCTION

Blood pressure is the force of the blood pushing against the walls of the arteries (blood vessels that carry oxygen-rich blood to all parts of the body). When the pressure in the arteries becomes too high, it is called hypertension. Up to 5 percent of women have hypertension before they become pregnant. This is called chronic hypertension. Another 5 to 8 percent develop hypertension during pregnancy. This is referred to as gestational hypertension. Gestational hypertension generally goes away soon after delivery: however, women who develop it may be at increased risk of developing hypertension later in life. High blood pressure usually causes no noticeable symptoms, whether or not a woman is pregnant. However, hypertension during pregnancy can cause serious complications for mother and baby. Fortunately, serious problems usually can be prevented with proper prenatal care.

ANATOMY AND PHYSIOLOGY

When most people hear the term cardiovascular system, they immediately think of the heart. We have all felt our own heart "pound" from time to time, and we tend to get a bit nervous when this happens. The crucial importance of the heart has been recognized for a long time. However, the cardiovascular system is much more than just the heart, and from a scientific and medical standpoint, it is important to understand why this system is so

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vital to life. Most simply stated, the major function of the cardiovascular system is transportation. Using blood as the transport vehicle, the system carries oxygen, nutrients, cell wastes, hormones, and many other substances vital for body homeostasis to and from the cells. The force to move the blood around the body is provided by the beating heart. The cardiovascular system can be compared to a muscular pump equipped with one-way valves and a system of large and small plumbing tubes within which the blood travels.

CASE REPORT

A 27-year-old white woman was transferred to the vijayalakshmi Hospital, suryapet, at 25 weeks in her first pregnancy because of a blood pressure of 150/100 mm Hg and intermittent proteinuria. A right-sided carotid arteriogram was normal. Her blood pressure was 120—160/70—96 mm Hg. The neurologic abnormalities persisted.

Shortly afterwards she conceived while taking propranolol and bendrofluazide. At 10weeks into gestation, her blood pressure was 160/100 mm Hg, settling to 140/90 at 16weeks. At that time she had no proteinuria. By the time of her transfer (at 21 weeks), methyldopa, debrisoquine, and chlorpromazine had been added to the regimen to control the increasing blood pressure. Physical examination was normal apart from wasting and reduced function in the left arm. The kidneys were not palpable and there were no renal artery bruits. Peripheral pulses were present and synchronous. The retinal arterioles were not narrowed and no hemorrhages or exudates were present. Fetal size and heart rate pattern also were normal. The plasma creatinine was 0.6mg/dl; uric acid, 5.1 mg/dl; and 24-hour urinary protein excretion, 0.88 g. Platelet count was 283,000/mm3; the ratio of Factor Vill-related antigen to clotting activity was 1.28. Liver function tests were normal. Her treatment was changed to methyldopa, labetalol, and hydralazine, for which oral diazoxide was later substituted. Nevertheless her blood pressure continued to be unstable, repeatedly reaching 160/100 mm Hg.The peaks in arterial pressure were associated with prostrating paroxysms of severe headaches, vomiting, and tachycardia. She continued to have proteinuria (up to 2.3 g/24 hours), but renal function remained normal; plasma creatinine was always less than 0.7 mg/dl and uric acid less than 5.0 mg/dl. Repeated midstream samples of urine were unremarkable except for asymptomatic bacilluna that was treated appropriately. The platelet count was always greater than 250,000/mm3, but the Factor VIII ratio rose slowly to 2.27 by the 35th week of pregnancy. At 35 weeks, labor was induced. The patient delivered vaginally a healthy son weighing 2340 g (lOth—25th percentile) who did not need special care. After delivery the patient's symptoms and the instability of her blood pressure remitted. On discharge, propranolol, 130 mg four times a day, was prescribed. Four weeks later the blood pressure was 128/90 mm Hg, renal function was normal, and no proteinuria was present. She was feeling extremely well.

DISCUSSION

She was taking atenolol and the blood pressure was 160/100 mm Hg.the first, by paroxysms of prostrating headaches, vomiting, extreme hypertension, and tachycardia. A CT scan of the skull was normal. By the end of her pregnancy she was taking atenolol, methyldopa, slow-release nifedipine, and an antiemetic. Her renal function remained normal. Proteinuria was recorded sporadically but never reached more than 0.4 g/24 hours. After a normal delivery at 39 weeks (a healthy daughter, 2450 g, loth—26th percentile for gestational age), her symptoms again remitted; by 4 weeks postpartum the blood pressure was 130/80mm Hg while she was taking atenolol, and she felt entirely well. Renal function was normal and proteinuria had disappeared.

CONCLUSION

Pre-eclampsia usually progresses through three distinct stages: hypertension alone, proteinuric hypertension without symptoms, and proteinuric hypertension with symptoms. Finally, eclampsia ensues. Characteristically, the speed of the progression accelerates so that the symptomatic third stage may last only a few hours and, at most, a few days. A woman with symptomatic pre-eclampsia therefore must be delivered without delay regardless of gestational maturity. At presentation this patient appeared to have proteinuric pre-eclampsia without symptoms .which shortly afterwards was associated with severe headaches and vomiting—the typical pre-eclamptic symptoms. Thus an argument could have been made for urgent delivery. But several of her features were so atypical that we believed the diagnosis of pre-eclampsia could be excluded with reasonable certainty and that the pregnancy could be continued safely.

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