

DFASM3 – FollowUp 05 Late Pregnancy Crisis Transcript
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OB-GYN consultant Samantha Jennings is leading a tutorial session with residents Morgan Brown, Andrew Minnows and Julia Teider at Massachusetts General Hospital.

SAMANTHA JENNINGS: So Morgan, it is your turn to present a case you found interesting to the rest of the group. What have you got for us today?

MORGAN BROWN: Well, last week I saw a 31-year old woman in the 37th week of pregnancy, Mrs Ellis, and found her case interesting. She presented to the ER with **sudden onset** of severe **bitemporal headache** and **shortness of breath**. She was in **respiratory distress**, which was made worse by **lying flat**, and reported that she felt as if she was **drowning**.

ANDREW MINNOWS: How had her pregnancy progressed up to then?

MORGAN BROWN: Until the onset of these symptoms, her pregnancy had been uncomplicated, with normal BP throughout. At her last prenatal visit, last week, her BP was 120 over 70 and her **heart rate** was 72bpm. Since becoming pregnant, she has gained 17 kg. Mrs Ellis reported having no fever, cough, chest pain, back pain, nausea, vomiting, **diplopia**, visual symptoms, abdominal pain, contractions or **vaginal bleeding** but she did report reduced fetal movements.

JULIA TEIDER: Did she take any medication or **suffer from** other conditions?

MORGAN BROWN: Yes, she had hypothyroidism, for which she took levothyroxine, 150microg daily, and she also had pernicious anemia. She had two previous pregnancies that ended in **spontaneous abortion** in the first trimester. She took 1mg of vitamin B12 intramuscularly monthly and prenatal vitamins. She had no known allergies and she did not smoke.

SAMANTHA JENNINGS: And what did your examination reveal?

MORGAN BROWN: Well, her BP was 180/110 when **supine**, equal in both arms. Her **JVP** was 15 cm H²O. Her heart rate was 120bpm. I examined her heart and found a diffuse but non displaced **apical impulse**, a **summation gallop** and an apical **holosystolic murmur** grade 2/6. She had no edema in her legs. Her respiration was 32 bpm, her O² sat 70% when breathing ambient air. Upon examining her chest, **bibasilar rales**, persisting with cough and deep breathing were revealed. She presented normal reflexes and her optic fundi was normal.

JULIA TEIDER: What about her abdominal examination?

MORGAN BROWN: It was normal, with **mild** contractions every 5 minutes. Her cervix was dilated by 1 cm and 80% effaced, with membranes intact. The fetal heart sounds were normal at 130bpm.

SAMANTHA JENNINGS: So Andrew and Julia what do you make of it all? What would be your next step?

ANDREW MINNOWS: Well I would run some blood tests, **an FBC** as the high blood pressure is worrying and I would want to determine the cause of it.

SAMANTHA JENNINGS: Julia, what do you think were the most likely etiologies for her high blood pressure?

JULIA TEIDER: This late in the pregnancy, with no previous history of high blood pressure, I would say preeclampsia and gestational hypertension.

SAMANTHA JENNINGS: That's right, chronic hypertension in pregnancy is defined as hypertension that is diagnosed before pregnancy or before 20 weeks of gestation but our patient is far past it. Gestational hypertension is the onset of hypertension after 20 weeks of gestation that is not associated with proteinuria. So a blood test would indeed be indicated. Any other tests?

JULIA TEIDER: I think I would do an ECG as well.

MORGAN BROWN: That is exactly what we did **actually** and quite a few of Mrs Ellis's results were abnormal: her white cell count was high at 21,800 per mm³, Hematocrit was also high at 45%, her creatine kinase was at 1,353 U/liter, her troponin I at 23.3 U/liter, the aspartate aminotransferase was at 74 U/liter.

ANDREW MINNOWS: And what about the 24-hour urine sample for **assessment** of protein excretion?

MORGAN BROWN: It could not be obtained in this patient given her emergency situation but we took a **spot urine sample** which showed a creatinine ratio of 4.27.

ANDREW MINNOWS: Well, given this result, I would say it is likely Mrs Ellis's raised blood pressure was due to preeclampsia.

SAMANTHA JENNINGS: Yes. A spot urine sample showing a high protein: creatinine ratio is reasonably predictive of proteinuria as measured with a 24-hour urine collection. We can conclude that Mrs Ellis seemed to have preeclampsia, but there were other abnormalities to explain. What did her ECG show?

MORGAN BROWN: Poor R-wave progression and **nonspecific ST-T wave** changes.

SAMANTHA JENNINGS: Julia, what do you make of that?

JULIA TEIDER: I think these findings are nonspecific and may be variants of normal results but do raise some concern about ischemic, **myopathic, metabolic, or infiltrative disease** of the myocardium. Since the patient's presentation **suggests heart failure**, I would recommend chest radiography, with abdominal **shielding** to minimize fetal radiation exposure.

SAMANTHA JENNINGS: Very nice! Andrew?

ANDREW MINNOWS: I would obtain a transthoracic echocardiogram to **rule out** peripartum cardiomyopathy and other forms of cardiac disease, especially if the chest radiograph shows pulmonary edema.

SAMANTHA JENNINGS: OK, good. I would add that the patient's hypertension and sudden onset of severe headache suggest rupture of a cerebral **aneurysm**, which is more common during pregnancy. This may be related to hormone-induced vascular **remodeling**. To exclude this possibility, it would be reasonable to perform a CT of the head.

MORGAN BROWN: We also raised the possibility of aortic dissection at the time but the absence of chest or back pain in this patient made a diagnosis of aortic dissection unlikely.

JULIA TEIDER: What were the test results?

MORGAN BROWN: Well, the X-ray showed that the extent of the pulmonary vascular congestion in Mrs Ellis was greater than what may normally develop in late pregnancy. The echocardiogram showed that the patient had global left ventricular dysfunction, with moderate left ventricular dilatation and a left ventricular ejection fraction of 20%. She also had moderate **mitral regurgitation** but her CT scan of the head was normal.

SAMANTHA JENNINGS: What do you think would be the most likely condition to explain Mrs Ellis's depressed systolic function?

ANDREW MINNOWS: It could be **acute MI** but although the patient's troponin level is high, her electrocardiogram is not suggestive of myocardial infarction.

SAMANTHA JENNINGS: Yes, I agree.

ANDREW MINNOWS: I can think of acute rheumatic fever.

SAMANTHA JENNINGS: That is less likely in the absence of fever and arthralgias.

JULIA TEIDER: I don't think it could be amniotic-fluid embolism because of the presence of hypertension, rather than cardiogenic shock and hypotension.

SAMANTHA JENNINGS: Good.

ANDREW MINNOWS: Systemic lupus erythematosus may cause cardiomyopathy, but this patient has no other symptoms to suggest lupus so I don't think that's it. So that leaves me with peripartum cardiomyopathy.

SAMANTHA JENNINGS: Indeed, and remember that hypertension, especially preeclampsia which this patient also appears to have, is a risk factor for peripartum cardiomyopathy. In one study, preeclampsia was present in 30% of patients with peripartum cardiomyopathy.

JULIA TEIDER: Am I right in remembering that **subsequent** pregnancies may be **hazardous** for patients with peripartum cardiomyopathy?

SAMANTHA JENNINGS: That's correct. Now if the working diagnosis was pulmonary edema as a result of severe preeclampsia, complicated by peripartum cardiomyopathy, how would you have stabilized the patient's condition?

JULIA TEIDER: Hum, I would give her intravenous labetalol, hydralazine, furosemide, and administer bilevel positive airway pressure.

SAMANTHA JENNINGS: Morgan, is that what you did? And what happened to the patient after you administered the treatment?

MORGAN BROWN: Yes, that is the treatment plan we followed. We also administered magnesium sulfate and the patient had a good clinical response to treatment. Post partum, her leukocytosis and proteinuria **resolved**, and the **previously** elevated levels of serum creatine kinase and troponin normalized. Another echocardiogram obtained before the patient was **discharged** revealed **mild** improvement in left ventricular function and an estimated ejection fraction of 30 to 35%. The patient was discharged with prescriptions for digoxin, lisinopril, and metoprolol succinate and was **scheduled** for a **follow-up** visit in the cardiology department two weeks later, which would be next week. At the time of discharge, Mrs Ellis felt physically well, her blood pressure was 95/60 mm Hg, and her heart rate was 99 beats per minute.