Heart Valves in Pregnancy Require Trade-Offs

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SNOWMASS, COLO. — There is no ideal solution when it comes to managing anticoagulation in the pregnant patient with a mechanical heart valve, Dr. Carole A. Warnes stressed at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

"This is not the same as getting your patient through noncardiac surgery. It's very different. The blood is stickier than at any other time you'll have to manage a mechanical valve," cautioned Dr. Warnes, professor of medicine at the Mayo Clinic, Rochester, Minn.

Other normal physiologic changes in pregnancy that increase the risk of thromboembolic events in patients with mitral or aortic valve prostheses include a nearly 50% increase in circulating blood volume, accompanied by a 30% rise in cardiac output and a 10-20 beat-per-minute increase in resting heart rate. And uterine contractions can trigger sudden jumps in systolic and diastolic blood pressure.

What makes managing thromboembolic risk in these patients so challenging is the need to trade off maternal versus fetal risk.

Unfractionated heparin doesn't cross the placenta. It is often considered safer for the fetus than warfarin in pregnancy. But unfractionated heparin is a poor anticoagulant in pregnancy. The response to the standard dosage varies widely because of the background increases in factor VIII and fibrinogen, so the risk of a thrombosed valve or other thromboembolic event with prolonged heparin is about 10%. The maternal hemorrhage risk is also increased.

Warfarin is far more effective than unfractionated heparin at preventing valve thrombosis in pregnancy. However, it crosses the placenta, and fetal exposure during gestational weeks 6-9 can result in warfarin embryopathy. The risk is about 6%, but might be dose dependent.

Whatever strategy is used, a daily baby aspirin in the second and third trimesters is safe and probably beneficial.

DR. WARNES

"The fetal risk is probably not as high with warfarin as you might think, but for medicolegal reasons you probably want to avoid it in most circumstances," said Dr. Warnes at the conference, which was cosponsored by the American College of Cardiology.

Some advocate low-molecular-weight heparin throughout pregnancy as the best approach, but Dr. Warnes is leery. The supporting data are limited. Moreover, she has seen thromboembolic complications occur even when LMWH dosing was guided by monitoring of factor Xa levels rather than relying on fixed-dose therapy.

The most popular management strategy in the United States entails a switch from warfarin to unfractionated heparin as soon as pregnancy is diagnosed, with a switch back to warfarin at 13 weeks' gestation, after the risk of embryopathy is over. This is followed by another switch back to heparin at about 35 weeks in anticipation of delivery, because the fetus can't safely pass through the birth canal while anticoagulated. The heparin is stopped for as short a time as possible around delivery. Heparin is resumed 6-12 hours post partum, because that's still a high-risk period for valve thrombosis.

If this strategy is used, it's important to give heparin at an adequate intensity. This means maintaining the activated partial thromboplastin time at greater than twice control. If factor Xa monitoring is used, aim for 0.35-0.7 U/mL of anti–factor Xa, Dr. Warnes urged.

The highest-risk situation in pregnancy in terms of thromboembolism involves a tilting disc prosthesis in the mitral position. This is a situation in which continued use of warfarin throughout pregnancy is a reasonable strategy until the switch to intravenous heparin at week 35, even though the Physicians Desk Reference lists warfarin as contraindicated in pregnancy, she said.

Warfarin throughout pregnancy is a particularly attractive strategy in a high-risk woman who was well controlled on the anticoagulant at 5 mg/day or less prior to pregnancy, which might lessen the risk of warfarin embryopathy.

Whatever anticoagulation strategy is used in pregnancy, a daily baby aspirin during the second and third trimesters is safe and probably beneficial. It should be used routinely, said Dr. Warnes. ■

Careful β-Blocker Use a Possibility in Pregnancy

SNOWMASS, COLO. — Don't hesitate to continue β-blocker therapy throughout pregnancy when the situation calls for it, Dr. Carole A. Warnes urged at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

"In practice I have been using β-blockers in pregnancy for 30 years. I've never had a significant problem with a baby after the mother has had a β-blocker," said Dr. Warnes, professor of medicine at the Mayo Clinic, Rochester, Minn.

"Do we worry about the growth of the fetus? Yes, and it needs to be monitored. At the time of delivery the baby may be bradycardic or may have hypo- glycemia, but we can deal with that very easily. So for the woman who needs a β-blocker—for example, a patient with hypertrophic cardiomyopathy, or perhaps hypertension with a dilated aorta—we can use them and use them safely. And if it's better for the mother to continue, then we do so," she asserted at the conference, which was cosponsored by the American College of Cardiology.

There are four key principles to keep in mind when prescribing cardiovascular drugs in pregnancy: Stick to those with a long safety record, use the lowest effective dose and for the shortest duration, avoid multidrug regimens, and steer clear of agents labeled category D or X by the Food and Drug Administration, Dr. Warnes advised.

In addition to many of the β-blockers, other cardiovascular drugs that Dr. Warnes listed as being relatively safe during pregnancy include calcium channel blockers, digoxin, procainamide, methyldopa, hydralazine, and furosemide.

Agents that are not safe during pregnancy include statins, ACE inhibitors, angiotensin receptor blockers, phenytoin, and folic acid antagonists, including some antibiotics, she noted. ■

Focus on Prevention In Acute Pericarditis

SNOWMASS, COLO. — Avoiding corticosteroids in treating acute pericarditis is the best way to prevent development of chronic relapsing pericarditis, Dr. Rick A. Nishimura said at a conference sponsored by the Society for Cardiovascular Angiography and Interventions.

"[Chronic relapsing pericarditis] is a terrible disease. It's incredibly debilitating and incredibly difficult to treat. The best treatment is to not ever give steroids in the first place for your typical viral pericarditis," said Dr. Nishimura, professor of medicine at Mayo Medical School, Rochester, Minn.

Chronic relapsing pericarditis most often follows treatment of an episode of acute pericarditis using a several-week burst of prednisone followed by a quick taper. Patients experience multiple recurrences of pericardial pain and an elevated erythrocyte sedimentation rate (ESR) whenever the prednisone dosage drops below, say, 15 mg/ day.

Acute pericarditis is an inflammation of the pericardium, typically from an upper respiratory tract infection or other viral infection. A burst of prednisone is a popular therapy because it's the fastest way to get rid of the pericardial pain, but the safest and best therapy is high-dose aspirin or an NSAID for at least a month, followed by a slow taper, he said at the conference cosponsored by the American College of Cardiology.

Treatment options for chronic relapsing pericarditis are limited. The patient can receive high-dose aspirin while a very slow taper off prednisone is attempted. If that's unsuccessful, there is some anecdotal support for off-label use of rituximab (Rituxan), the B-cell–depleting rheumatoid arthritis drug. "The alternative is complete pericardiectomy—that's open-heart surgery for a patient who started out with a simple acute pericarditis."

Acute pericarditis is diagnosed on the basis of pericardial pain, presence of a precordial friction rub, elevated ESR indicative of acute inflammation, and ECG findings of diffuse ST elevation and PR depression without pathologic Q waves. "You don't need an [ECG] to diagnose pericarditis," he noted. ■