Editorial

Heparin Anticoagulation

Key words: Anticoagulation, unfractionated heparin, low molecular weight heparin

Clin. Cardiol. 2007; 30: 154–155.
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I do not know whether there is any quantitative information on the number of patients receiving heparin. However, if one classifies patients according to their disease states, I think cardiovascular patients have received heparin more frequently than any other group of patients. Most of these patients receive short-term therapy, i.e. patients with

ST-elevation MI, non-ST-elevation MI, unstable angina, pulmonary embolus, patients with cardiopulmonary bypass, patients in atrial fibrillation as a bridge to Coumadin. Most cardiovascular patients who are chronically anticoagulated receive Coumadin.

In this editorial, I will not discuss Coumadin, but will comment on the use of unfractionated heparin and low molecular weight heparin (LMWH). The vast majority of *Clinical Cardiology* readers learned to use unfractionated heparin for their patients many years ago. I include myself in that group. I feel quite comfortable when using this preparation, particularly when it is given intravenously. I often ask medical residents if the patient is anticoagulated. They frequently respond that the patient is heparinized. That is not the answer I want because the activated partial thromboplastin time (aPTT) determines the anticoagulation status of the patient, not the heparin dose administered. When using unfractionated heparin,

it is necessary to measure the aPTT in order to properly and safely anticoagulate the patient. Most hospitals that receive patients with acute coronary syndromes have protocols to initiate and maintain anticoagulation based on aPTT levels. Usually these levels are 1.5 to 2.5 times control. Some think this is an inconvenience and a rather imprecise measurement of anticoagulation status. Despite this, heparin is still the most widely used anticoagulant, but there is an increasing tendency to use LMWH preparations because several randomized studies have demonstrated their efficacy and safety when administered in fixed dosage and without laboratory monitoring. Unfractionated heparin or LMWH (when administered with aspirin) are class I indications for anticoagulation of patients with unstable angina or non-ST-segment elevation myocardial infarction.¹ LMWH (Enoxaparin) is a Class IIa indication in the absence of renal failure and unless CABG is planned within 24 h.

I can think of four reasons for using LMWHs:

1. There is no perceived need for monitoring anticoagulation status
2. There is possibly a smaller risk of bleeding
3. There is possibly a smaller risk of osteoporosis in long-term use
4. Many claim there is a smaller risk of heparin-induced thrombocytopenia (HIT)

Let me take these issues one at a time.

No Need to Monitor Anticoagulation

How can anyone be sure of the anticoagulation status of the individual patient when no parameters are measured in the individual? Data contained in clinical trials, of course, apply to populations. And these populations are generally patients who have no increased risk of bleeding, are not obese and do not have severe renal failure. In addition, children and pregnant women are not included in these trials.

In fact, there are many experts in the field of anticoagulation who feel quite strongly that, during the first trimester of pregnancy, a woman who has a mechanical

prosthetic valve and is concerned about the embryopathy resulting from Coumadin use should receive heparin in high doses or LMWH with measurements of anti-10A activity frequently, i.e. once a week, in order to insure adequate anticoagulation.

Another point is that in clinical trials, not everyone who has received LMWH has done well. No one knows the reason for this, but one possible answer is that they were not properly anticoagulated, despite receiving fixed doses of LMWH. Of course, the same can be said of unfractionated heparin in these very same patients because generally there is not much difference in outcome when either formulation is used.

However, if we are going to measure anti-10A activity in the most dangerous situations, i.e. the pregnant patient with a mechanical valve during the first trimester, why not measure it in everyone, including patients with renal disease, subjects at increased risk of bleeding, children, the very obese, and patients who have a tendency to bleed for whatever reason? While I am on the subject of making measurements, one needs to consider the timing of those measurements in relationship to the dosing of LMWH.

Bleeding Risk is Small

Next I would like to comment on the possibility that LMWH has a small risk of bleeding. As far as I am concerned, any risk of bleeding is unacceptable and I challenge anyone to predict in the individual patient who will bleed and who will not with either unfractionated heparin or LMWH. Obviously, neither preparation should be given to someone who is actively bleeding or who has had a recent intracerebral hemorrhage.

Risk of Osteoporosis is Small

Next, what about the “smaller risk of osteoporosis in long-time use”? I think this is a nonissue for

cardiovascular specialists because I cannot think of a single cardiovascular case that I have taken care of in the last 32 years at the University of Florida to whom I have given chronic heparin therapy. When chronic anticoagulation is required, warfarin is the drug of choice in 2006.

Heparin Induced Thrombocytopenia

The final issue is that unfractionated heparin has a smaller risk of heparin induced thrombocytopenia (HIT). Obviously, this is a major complication of heparin therapy, but HIT is not zero in patients receiving LMWH. In fact, I doubt that anyone reading this editorial would feel comfortable giving LMWH to an individual who developed HIT after receiving unfractionated heparin. Once again, predicting the occurrence of HIT in the individual patient is impossible.

I make the above points not because I am opposed to using LMWH. I think it is a great product, and I think many are using it and using it with great success. However, I do question some of the claims about its superiority to unfractionated heparin. I make no claims that I have a full understanding of the complexities of the coagulation cascade and anticoagulation nuances that expert coagulation hematologists enjoy.

My concerns are based on my own feelings as a clinical cardiologist seeing patients in the acute setting who require urgent anticoagulation.

Reference

1. ACC/AHA Guidelines for the management of patients with unstable angina and Non-ST-segment elevation myocardial infarction. *J Am Coll Cardiol* 2002;40:1366–1374

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