Transcript: Why Do Those With HIV Have More Heart Attacks?

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At the European AIDS Conference in Madrid, Dr. Jens Lundgren explains why HIV and its medications seem to be causing more heart attacks, and tells Peter Staley that there's a lot to be said for traditional preventive strategies. To see the video click here.

Peter Staley: Good morning from Madrid. This is Peter Staley with AIDSmeds.com. We're at the European AIDS Conference. And this morning we're with Dr. Jens Lundgren from Denmark with the University of Copenhagen, and you're also the director of the Copenhagen AIDS Program. Welcome.

Jens Lundgren: Thank you very much. I appreciate it.

PS: You've been on the podium a lot at this conference, and I've enjoyed listening to you. You gave a fascinating talk this morning about cardiovascular risk—heart attacks, basically, with people with HIV. It's something obviously a lot of us are thinking about, especially as we're living longer, getting older, like myself, I'm 46, living with HIV 25 years now. And a lot of us are seeing our cholesterol go up, too, on the drugs. So we're worrying about this. You started by talking about a major study that looked at cardiovascular risk. The D:A:D study. Can you describe that for us?

JL: So what we did was, back in '98, '99, when the protease inhibitors first came on the market, we were concerned whether these drugs were changing the risk of cardiovascular disease. And therefore, we designed a study—it's a relatively large study because still, luckily, the risk of heart attack is very low in HIV patients. So we needed a lot of patients to see whether exposing the patients to these drugs would increase the risk of myocardial infarction. So that was undertaken nearly ten years ago. And now, we are beginning to get fairly solid results suggesting that, indeed, there is an increased risk by using protease inhibitors. And in addition to that—and I think that was really the point I was trying to make—in addition to that drug effect, which I personally think is very important to identify, because I would like as a physician to know the drugs I'm providing to my patients, what effects and side-effects do they actually have? So I think that's a very important research area. But what the studies have also shown is, there are multiple other risk factors in addition to exposure to treatment that influence cardiovascular disease. And essentially, though we were unsure when we started this study, the results show that what we call the traditional risk factors, those that everybody's aware of in the general population—smoking, hypertension, increased cholesterol for that matter—all those risk factors are in operation as well.

PS: People with HIV aren't angels. We have the same problems as everyone else.

JL: Which I think is just a sign of normal health, to some extent. So that's really the point I'm trying to drive home. That this is not only looking at drugs, this is looking at a composite of all the contributing risk factors that you have, and there are now tools to calculate, what is your individual risk of contracting a heart stroke in the next ten years. And I guess my point then is, since we can now calculate that, we can essentially say to many of our patients, Listen, you may have a little bit of increased cholesterol because you're taking these drugs. But your risk of getting a heart attack is so low, so let's essentially just ignore that. On the other hand, we will have patients where we will say, I'm sorry, your risk is now so high, so we really need to aggressively try to reduce that risk. And there are luckily a lot of tools to do that. And that's really where I'd like to see the research attention now focused: on how to prevent myocardial infarctions in HIV patients. Can we just take the interventions, all the steps and whatever else is recommended in the general population, can we just take all that thinking and extrapolate it for HIV patients, or do we need to do something special? And in order to understand that, we need to do research. And I'm not sure that we are there yet. So that's really my main point that I would strongly encourage that we start to focus more on intervention studies to reduce the risk, and I think that's where the next ten years' research should be based.

PS: Now, for the PIs, you mentioned a Boston study. We're kind of getting close to quantifying the increased risk for people who are on therapy that includes a PI, for instance, how much more likely they are to have a heart attack than an HIV-negative person. And that's how much?

JL: It's approximately a doubling of the risk. And again, the doubling of the risk—if your underlying risk is extraordinarily low, then your doubling of an extraordinarily low risk remains an extraordinarily low risk. On the other hand, a doubling of your risk if your underlying risk is relatively high, that means something. And that's very well exemplified in the Boston study, that in older age patients, their increased risk, in absolute terms, out of 100 patients how many got a myocardial infarction—a heart attack. That number was much higher if you are HIV positive versus HIV negative in older people. So what I'm trying to get at is, it is older HIV patients where we need to start to focus on these issues. And I think compared to how the situation is at the moment, at least in Europe, where we haven't been that aware of that, as physicians, we need to put that much higher on the treatment agenda when we discuss with our patients every three or four months, how's your health, etcetera, that we need to focus more on prevention of cardiovascular risk. And of course patients can do much more themselves. They can stop smoking—unfortunately, fifty percent of European HIV patients, at least, are smokers, and that's obviously a strong risk factor that you can do something about yourself. Your diet, exercise, etcetera—all the general recommendations that you also will say is true for the general population, of course HIV patients can do that as well. And I think they should at least be encouraged to do so.

PS: Now, one thing that's really fascinating, that everyone's buzzing about, is that if you're really scared of a heart attack, and you're on a protease inhibitor, it now turns out the last thing you want to do is stop treatment. Because the SMART trial, with is the largest trial ever done looking at treatment interruptions, taking people off therapy for a time, actually showed an increased heart attack risk for those who stopped therapy. So, yeah, there might be an increased heart attack risk from taking a protease inhibitor, but it's even worse without the drugs?

JL: Here we go—so that's just one example of an intervention that we thought, when SMART was designed, made sense. If the drugs were increasing your risk of a myocardial infarction, well, if you take patients off drugs, it should be less. We turned out to be completely wrong. So we can take that intervention and scratch it from the list of possible interventions. So that's not going to be working, so we need other interventions, we need other ways of reducing the risk. And I think we can do that.

PS: What's the leading theory of why those who stop therapy or are not on therapy—just having HIV by itself is increasing heart attack risk?

JL: I would say that since SMART was patients on therapy who were interrupting therapy, SMART shows that once you interrupt therapy, something happens that increases your cardiovascular risk. It is likely that when you stop treatment, you unleash HIV, so it starts to replicate again, and that results in inflammation, that results in possibly other associated events in the body, circulation activity may be increasing, which then, as a consequence of that, may have either direct influences on the arterial wall and resulting in the disease, or that it will reduce HDL cholesterol, the good cholesterol, and there's actually direct evidence from the SMART study that those who interrupted therapy got depletion of HDL, and that's, at least from a theoretical point of view, not a good thing. So that could well be. But the HDL decline may well have been caused by some sort of inflammatory reaction resulting from interruption of therapy. But I would stress that this is still a work in progress.

PS: HDL declines with HIV infection even before you start therapy, right? So that could be explaining things.

JL: Absolutely. Carl Grunfeld from San Francisco did a hallmark study in 1992 where he observed that untreated patients—of course there was no effective treatment at that point—that those who had progression of the HIV disease had depletion of HDL.

PS: It lowers all cholesterol, but it might be very harmful that you're losing the good cholesterol.

JL: Our current understanding on the different fraction of cholesterol is that you really need to look at the ratio of total to HDL cholesterol, and that ratio should go down. If it goes up, you get an increased risk; if it goes down, you get a reduction in risk. So therefore, it's a ratio between HDL and total cholesterol. And that was clearly shown in the SMART study—that the ratio actually went up in those who interrupted therapy.

PS: Well, it's an area that's going to need a lot more research to figure out, but at least we know the standard interventions, and we need to use those right now.

JL: Absolutely. We've got to take what we know works. And let's start from there, and let's be critical about what else to do. Because HIV patients get a lot of drugs already—let's not forget: don't mess around with antiretroviral therapy. It works. It's helped a lot of patients survive. So don't mess around with that treatment. But we need to think very carefully about how to reduce the risk of cardiovascular disease, but it needs to be in the forefront of our minds. We need to move that area forward.

PS: Thank you very much, and good luck with the rest of your presentations at the conference, and let's get out of this cold. It's my last interview outside, I think.

JL: Thank you.

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