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Discussion

DR J. J. PUIGBO: I think that Dr Falase's idea of histological studies comparing different areas is a very important approach. This is the approach that we follow in Venezuela in cases of Chagas's, to look for the initial stages of the disease. When I was a medical student we were only seeing the last stages of the disease when it was too late to do anything. I think that the same has happened now in EMF, but now we are identifying earlier cases and can trace it back to the eosinophils. I think that your suggestion on epidemiology is going to be very useful in order to locate the cases. Maybe the seasonal rain in our countries is the problem. The surgical solution that you proposed is also relevant.

DR C. DUBOST: When performing this type of operation, do you make the incision between the right atrium and the right pulmonary artery, or between the left atrial appendage and/or the left pulmonary artery?

PROF. FALASE: Between the right atrium and pulmonary artery.

DR DUBOST: Do you close the tricuspid valve?

PROF. FALASE: No, we do nothing to it, so that it is still incompetent, achieving a better blood flow.

DR DUBOST: Yes, a high degree of pulmonary hypertension could make the operation deleterious. Do you intend to do this type of operation on the left side because you could do the same with a left pulmonary artery?

PROF. FALASE: No, but then we would be running blood through the lungs again.

PROF. J. F. GOODWIN: I would suggest that we are dealing with one syndrome and the syndrome is endomyocardial disease with degranulating eosinophils. I would take issue with you, Professor Falase, however, when you say that it is certain that eosinophils cause endomyocardial disease because I am not sure that that is so. I am sure there is an association between the abnormality of the eosinophil and endomyocardial disease but I am not sure that it is aetiological. I believe that there is probably a different factor in the temperate zone variety compared with the tropical one. I have always been impressed by the possibility of an insect-borne infective agent but that might only be so in Africa and it might be entirely different in Venezuela.

DR C. J. F. SPRY: Like you, I am concerned about the regional distribution of EMF. It appears to be found in people who have poor nutrition and who live in areas where there are large numbers of insects and parasitic diseases. These people would be repeatedly exposed to infective forms of parasitic disease. Such individuals may have a high degree of immunity to the parasites, and this would give rise to marked inflammatory reactions each time they were infected. This might explain why studies which measured the prevalence of parasitic disease, such as filariasis in patients with EMF, did not show any real differences from control subjects.

PROF. FALASE: Filariasis is very common in the rain forest area. I think we should look for an insect which is confined to those areas.

DR DAVIES: Can I ask about the protein content of ascites and pericardial fluids. I have always assumed that it was a transudate, but some work in India indicates that it is an exudate.

DR FALASE: In most cases it is in fact an exudate not a transudate.

DR OLSEN: We must distinguish between aetiology and pathogenesis. I am in total agreement with everyone that we have not yet established the aetiology of endomyocardial disease, but we have more and more persuasive evidence that eosinophils are a key factor in the pathogenesis irrespective of the causes resulting in eosinophilia (filariasis or other parasites). Christopher Spry showed yesterday (Spry, Tai and Davies, 1983) how the granules act and attack the myocardium first and then the endocardium. So the process we described in 1973 (Brockington and Olsen, 1973), retrospectively suggesting that from a myocardial disease endocardial changes follow with thrombus superimposition ending in fibrous tissue, is, I think, now fairly clear.

CHAIRMAN: Clearly Dr Falase's observation on the tropical rain belt is important. We have come round now to suspecting that there may be some aetiological agent and that it may be insect borne, that it is responsible for initiating the process in which we see the degranulated eosinophil. We need, as Christopher Spry has just said, to get at the very early cases and I think it is very important from what we have heard this afternoon to take endomyocardial biopsies in these very early cases, to look in these patients for the degranulating eosinophil and, at the same time to combine with that longitudinal echocardiographic studies.

DR OLSEN: Professor Falase, it may be confusing to say that Löffler's disease leads to endomyocardial fibrosis. I think it is better if one says that the thrombotic stage leads to the fibrotic stage. Some earlier cases have been called Löffler's, indeed also some very early cases have also been called endomyocardial fibrosis. Also, I think the difficulty arises from talking about two diseases. When we set up this multicentre study in December 1978, we suggested that we should drop the term Löffler's endocarditis and talk about endomyocardial disease whether it occurs in the tropics or in the temperate zone. There is a sequential progression from the acute stage to the thrombotic stage to the fibrotic stage and that can happen in both geographical zones.

DR SPRY: One important reason for not using the term fibrosis to describe this disease is that early lesions are not fibrotic.

References

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