AN INTERESTING CARDIAC PROBLEM

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THE opportunity has been taken of reporting the following interesting and unusual cardiac case, in which the progressive varying nature of the clinical picture has suggested the possibility of several alternative diagnoses.

Case History.—E. A., a young National Service man of 20 years of age, was admitted to hospital on 14.11.54, having been ill for one week. His first symptoms (7.11.54) were malaise, rigors, a non-productive cough and a sore throat. In the few months prior to admission he had suffered from recurrent sore throats at about monthly intervals. On 10.11.54 he developed pain and swelling in both knees and in his right elbow. Examination showed an ill-looking, flushed and sweating youth, with a temperature of 104° F. He had enlarged and inflamed tonsils. His knees were hot and tender, both showing an effusion and restriction of movement; his right elbow was tender and painful on movement. The sole abnormality found in his cardio-vascular system was a soft, blowing, apical systolic murmur. No rheumatic nodules were found in either upper or lower limbs. Fine crepitations were heard at his left lung base, and scattered sibilant rhonchi throughout both lung fields.

Laboratory investigations at this time showed: Hæmoglobin 95 per cent.= 14.1 g. per cent.; E.S.R. 50 mm. in 1 hour (Westergren); white cell count 12,500/c.mm.—polymorphs 83 per cent., lymphocytes 15 per cent., monocytes 2 per cent. Urine showed numerous pus cells and granular casts, but culture was sterile. The throat swab gave normal throat commensals only.

A diagnosis of acute rheumatic fever was made, and he was given a course of sodium salicylate gr. 150 daily in divided doses for five days, after which the temperature subsided. Five days after admission (19.11.54), although he remained afebrile, crops of minute pustules at the back of his neck, in both axillæ, on his lower abdomen and in both groins were noticed, each pustule showing very little surrounding inflammation. At the same time the pulmonary symptoms had become worse, cough and sputum had increased, and there was more dyspnœa. Examination revealed an acute purulent tonsillitis, medium crepitations throughout both his lungs, and radiologically diffuse broncho-pneumonic consolidation.

Laboratory results (19.11.54): Culture from skin pustules gave a pure growth of *Staphylococcus albus* sensitive to aureomycin; white cell count 8,000/c.mm., with polymorphs 70 per cent., lymphocytes 25 per cent., and eosinophils 2 per cent.; throat swab gave normal throat commensals; cold agglutinins were present in the blood; blood culture was sterile; sputum culture yielded a

pure growth of pneumococci; urine—no protein or other abnormal constituents, and culture was sterile.

In view of the acute purulent tonsillitis, broncho-pneumonia and skin pustules, he was given aureomycin capsules in doses of 1 g. six-hourly. The tonsillitis and broncho-pneumonia improved, but the temperature steadily rose, and on 26.11.54 he suddenly developed some remarkable cardio-vascular signs: his pulse became collapsing in character, his blood pressure being 125/50; a pronounced diastolic thrill was felt along the left border of the sternum, and a very loud (Grade V) "whining," high-pitched, diastolic murmur, throughout early and middle diastole, and fading away in late diastole, was heard. This murmur was very widespread, being audible over the whole of the præcordium and in the neck in the region of the carotid arteries, but was maximal over the third left intercostal space. The apical systolic murmur originally detected was also louder and more diffuse, so that a continuous to-and-fro murmur ("machinery-type") was present at the base of the heart. Other findings included a palpable spleen, but there was no evidence of emboli in any system. The E.S.R. was now elevated to 114 mm./hr., the white cell count showed 9,000/c.mm., with polymorphs 81 per cent., lymphocytes 11 per cent., monocytes 5 per cent., eosinophils 2 per cent., and basophils 1 per cent. Urine examination did not show any red cells in the deposit. Blood culture was again sterile. An electrocardiograph indicated marked right ventricular preponderance and also suggested acute pericarditis.

At this juncture it was considered that the most likely diagnosis was subacute bacterial endocarditis complicating a patent ductus arteriosus. A course of intramuscular crystalline penicillin was therefore instituted on 30.11.54 in daily doses of two million units, to be continued for six weeks. After twelve days of penicillin the patient experienced a sudden sharp pain in the left hypochondrium. No splenic friction rub was heard and no other abnormal physical signs elsewhere were detected. Simultaneously, the temperature rose, being now intermittent in character and persisting for two weeks, after which it gradually subsided. During the course of penicillin therapy the cardio-vascular findings slowly changed, until at the termination of treatment a new set of physical signs was present. The collapsing pulse was very obvious, the blood pressure being 130/50; gross capillary pulsation was seen in the nail beds; the Duroziez phenomenon was present over the femoral arteries; the cardiac apex was displaced to the left, being in the fifth intercostal space 4½ inches from the mid-line; both the pulmonary and aortic second sounds were increased; the to-and-fro murmur at the pulmonary area was gone, and in its stead was a loud, high-pitched, blowing, early diastolic murmur, maximal in the third left intercostal space and conducted down the left border of the sternum. This murmur was typical of aortic regurgitation. The systolic murmur, however, had by this time diminished considerably in intensity. All the physical signs now pointed to marked aortic reflux, and these signs have persisted up to the time of reporting this case.

DISCUSSION

The early history and findings in this case were compatible with a diagnosis of acute rheumatic fever, without any clinical evidence of carditis. The subsequent development, however, of the to-and-fro murmur at the base of the heart, in conjunction with splenomegaly, indicated the need for revision of the diagnosis. The possibility of a subacute bacterial endocarditis was therefore considered. It is very unusual to see this condition occur in a previously clinically normal heart. Bacterial infection is most frequently established in a valve damaged by past rheumatic fever, less often by arteriosclerosis or syphilis. It may also, of course, complicate congenital heart disease. In the present instance, in view of the continuous murmur at the base of the heart, it was believed that a patent ductus arteriosus was the site of the bacteria infection. Inflammation of the joints simulating rheumatic fever occurs in about 25 per cent. of all cases of subacute bacterial endocarditis, and interrecurrent infections, such as broncho-pneumonia, have been described in the disease. The early joint and chest findings would therefore fit in with the diagnosis. In addition, the splenomegaly and the later attack of pain in the left hypochondrium, suggesting splenic infarction, would also help to strengthen this diagnosis. Against this were the following facts: there was no evidence at all of any organic cardiac lesion when the patient was first admitted to hospital, least of all of patent ductus arteriosus; there were no petechiæ anywhere, no finger clubbing, no hæmaturia, either macroscopic or microscopic, no leucocytosis, and repeated blood cultures were sterile. The end-picture of gross aortic regurgitation makes it difficult to accept the diagnosis of patent ductus arteriosus *per se*. There is a possibility, however, that if he has had subacute bacterial endocarditis, it may have been superimposed on a congenitally abnormal aortic valve, as, for example, a bicuspid valve. This may be quite symptomless and give rise to no abnormal physical signs. After the valve had been damaged by the bacterial process, the basal cardiac murmurs could easily have appeared, and it is logical to assume that the final lesion in the heart would be aortic regurgitation.

The final diagnosis that needs to be considered is that of an acute malignant bacterial endocarditis terminating in rupture of a cusp of the aortic valve. The source of infection may well have been the purulent tonsils or the infected lungs. The skin pustules strongly suggest a pyæmic phase in the disease. The most unusual and intense character of the diastolic murmur, resembling the "whine" of a machine, can be attributed to a ruptured aortic valve cusp. This would, of course, lead to well-established aortic regurgitation. Pericarditis is not infrequent in acute septic endocarditis, whereas it is most unusual in subacute bacterial endocarditis. This patient showed electro-cardiographic evidence of recent pericarditis. In acute septic endocarditis, however, the patient is very ill, the clinical picture is primarily one of pyæmia, a substantial leucocytosis is found, and there is almost invariably a positive blood culture. None of these factors was much in evidence in the present case, but it should be borne in mind

that antibiotics may have assisted in producing negative results in the blood culture series and in modifying the clinical picture as a whole.

It will be seen, therefore, that the final diagnosis is still open to question, but the consensus of opinion, after full specialist observation and investigation, is that this patient has had a less severe form of acute ulcerative endocarditis with rupture of one or more of the cusps of the aortic valve.

SUMMARY

An interesting cardiac condition has been reported in which the initial clinical picture suggested acute rheumatic fever. The subsequent progress, it will be seen, indicated in turn subacute bacterial endocarditis superimposed on either a patent ductus arteriosus or a congenitally abnormal aortic valve, or finally acute ulcerative endocarditis with rupture of a cusp of the aortic valve.

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