RHEUMATIC PERICARDITIS IN EARLY CHILDHOOD

BY

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Acute rheumatism occurring under the age of 3 years is uncommon and only three cases of rheumatic pericarditis with effusion appear to have been reported in this age group, the youngest being a child aged 2\(\frac{1}{2}\) years reported by Ellenberg and Cook (1944). Antell (1935) and Massie and Levine (1939) each report one case, aged 3 years. Killian (1934) mentions seven cases of foetal pericarditis but considered that syphilis was the more likely cause.

In view of the rarity of recorded cases it is felt that the following two might prove to be of interest.

**Case Notes**

**Case 1.** A boy, aged 2\(\frac{1}{2}\) years, was quite well until July, 1949, when he became rather cross and irritable. During September he was believed to injure his right foot but an X-ray examination at the time disclosed no abnormality. He frequently complained of pain in the right foot during the following weeks. His mother noticed that he became ill-looking, pale, and slightly breathless during the week prior to his admission to hospital in October.

On examination, his face was pale and somewhat puffy. The alæ nasi were moving vigorously. The apex beat was palpable in the fifth left intercostal space just inside the mid-clavicular line. The heart sounds appeared normal. There was a well marked pericardial friction rub down the left border of the sternum. The throat was inflamed but the tonsils did not appear enlarged. Temperature was 100-2\(^\circ\) F. and pulse 120. Blood pressure 105/60. Hb 80 per cent (Sahli). Red blood cells 4,480,000 per c.mm., leucocytes 14,600 per c.mm. B.S.R. 47 mm. in the first hour. Urine was normal. Mantoux 1–1000 was negative. X-ray of the chest showed an enlarged cardiac shadow. A culture from a throat swab showed a moderate growth of hämolytic streptococci; no K.I.B. On the third day following admission the child was still breathless and anxious. The apex beat was not easily localized but appeared to be in the fifth intercostal space outside the mid-clavicular line. The heart sounds were distant and the friction rub had disappeared. The area of cardiac dullness was enlarged to percussion and extended beyond the apex beat. There was an impaired percussion note with diminished air entry at the base of the left lung. A further X-ray showed an enlarged cardiac shadow suggestive of a pericardial effusion.

An electrocardiogram taken on the fourth day following admission showed a sinus tachycardia with inverted T waves in leads III and V2; 6 weeks later, T waves were upright in all leads.

In view of the throat infection on admission the patient received 50,000 units of penicillin 4-hourly for 14 days.

Three weeks later the little boy's condition was greatly improved. Temperature had been normal from the sixth day. The signs of pericarditis had disappeared and radiologically the heart shadow had returned to normal. B.S.R. 23 mm. in the first hour. The Mantoux 1–1000 and 1–100 were again negative.

Two months following admission the child appeared well. Clinically the heart was normal apart from a soft localized systolic murmur at the apex. X-ray examination showed a normal heart shadow. B.S.R. 5 mm. in the first hour.
Six months later, the child was free from symptoms and appeared well. Clinically the heart was not enlarged, the sounds were of good quality and there was a soft apical systolic murmur, which was not conducted.

Case 2. A girl, aged 3 years, became listless, feverish, and refused food. She was seen at intervals by the family doctor until 11 days later he noticed that she had developed a cardiac murmur and advised her admission to hospital.

On examination, the child was flushed and very breathless. The apex beat was in the fifth left intercostal outside the mid-clavicular line. There was a well marked friction rub heard over the base of the heart and a loud systolic apical murmur conducted into the axilla. The area of cardiac dullness was enlarged to percussion beyond the apex beat. There was an impaired percussion note and tubular breathing at the base of the left lung. The throat appeared normal. Temperature was 100° F. and pulse 120. Hb 80 per cent (Sahli). Red blood cells 3,030,000 per c.mm., leucocytes 8200 per c.mm. Urine was normal. Mantoux 1–1000 was negative.

She remained extremely ill during the following ten days. No B.S.R. estimation was done as any attempt was strongly resented. In fact it was only with difficulty that a satisfactory fluid intake was maintained owing to the patient's poor condition and the degree of upset produced by the least interference.

Eight days after admission the friction rub had greatly diminished and a diastolic murmur became audible at the second left intercostal space and was conducted down the sternum. The apical systolic murmur remained unchanged. A chest X-ray showed an enlarged heart shadow with atelectasis of the left lower lobe. The cardiac shadow suggested the presence of a pericardial effusion.

After three weeks there was some improvement. The apex beat was in the fifth intercostal space in the mid-clavicular line. There was no clinical evidence of pericarditis. The apical systolic murmur could be heard over a wide area but the diastolic murmur could not be detected. A further X-ray showed an enlarged cardiac shadow but the left lung field was now clear. The Mantoux 1–1000 and 1–100 was again negative. An electrocardiogram taken on three occasions showed no abnormality.

The patient continued to improve fairly quickly and was much less resentful of interference. The systolic murmur became less audible and after 7 weeks no murmurs could be heard. The apex beat had returned to its normal site. The patient was discharged home after 10 weeks.

Ten months after the onset the child appeared very well. The heart appeared normal apart from a faint localized systolic murmur at the apex.

Comment

Two cases of pericardial effusion due to rheumatism have been described. It is interesting that neither of these presented the usual picture of acute rheumatism with joint pains unless the injury to the foot in Case 1 could be discounted and the symptom regarded as rheumatic in origin. Salicylates were refused by both patients and this was in keeping with the findings of Ellenberg and Cook (1944). In both the presence of signs at the base of the left lung in an acute illness might suggest pneumonia if the cardiac condition was overlooked. Tuberculosis was considered as a possible aetiological factor in both cases but a consistently negative Mantoux reaction, the absence of any pulmonary lesion, and the course of the illness were against this diagnosis. A paracentesis was not carried out as it did not appear justified and the leucocyte count was not suggestive of pus in the pericardium.

REFERENCES