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ACUTE PERICARDITIS SIMULATING CARDIAC INFARCTION

BY

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Simulation of cardiac infarction by acute pericarditis is well recognized by cardiologists, though references are not numerous (Barnes and Burchell, 1942; Wolff, 1944). Pericarditis in such cases is of unknown aetiology, since an obvious cause would leave no doubt about the diagnosis. It forms a subgroup of the type mentioned by East and Bain (1948), in which "cause and association are quite obscure," and is termed by Peel (1947) "primary or idiopathic," by Dry (1943) "non-specific . . . from which recovery may be complete," and by Andrews *et al.* (1948) "acute idiopathic." The infrequency of specific references seems to justify a report of only two cases. The association of one with a general arterial disease suggests a possible mechanism.

Case 1

A man aged 63 was admitted to the Royal Sussex County Hospital on July 23, 1948, and was diagnosed clinically as a case of temporal arteritis. Biopsy confirmed the diagnosis and relieved a severe headache: the histological report was, "The adventitia is infiltrated with round cells, among which are a few eosinophils and giant cells. There are areas of necrosis in the media. The appearances are typical of temporal arteritis." Though the condition was regarded as still active, with persistent fever, a predominantly polymorph leucocytosis of 11,800, and an erythrocyte sedimentation rate of 31 mm. in the first hour (Wintrobe corrected, normal 0-10), discharge was necessary after a fortnight.

Headaches returned, however, and he was awaiting readmission, when in the early hours of Sept. 21 he was suddenly attacked by a pain under the left costal margin which spread to the lower substernal area. He was admitted 16 hours later, with evident severe substernal pain but negative clinical findings except for fever of 100° F. (37.8° C.) and what was regarded as triple heart rhythm. During the next 24 hours his temperature fell to normal and the pain disappeared, but a pericardial friction rub developed. An electrocardiogram showed changes indicating acute pericarditis, with elevation and deformity of the R-T interval in all leads. This returned practically to normal a fortnight later; no evidence of cardiac infarction was present. He was then discharged, afebrile, but with a raised sedimentation rate, which persisted until he was last seen in February, 1949, when he complained of pains in the occiput, sterno-mastoid, thigh, and deltoid muscles. The heart appeared normal clinically and by electrocardiography and cardioscopy.

Case 2

A man aged 37 was seen on Jan. 19, 1949. Following moderate exposure and mild malaise, and after a heavy meal, he developed epigastric pain while driving his car. This grew

more severe and paroxysmal, extending from the xiphisternum upwards to the suprasternal notch, passing outwards to the pectoral regions and posteriorly to the interscapular region at the level of the sixth dorsal vertebra. Although no abnormal clinical signs of any kind were found, repeated injections of "omnopon" and pethidine were necessary.

By the next morning the patient felt practically recovered, but during this second day fever of 101° F. (38.3° C.) developed, and a loud but painless pericardial friction rub became audible. An electrocardiogram showed the changes of acute pericarditis, with elevation and deformity of the R-T interval in the limb leads and chest leads CR1 to CR7, maximal in II and CR3. Friction disappeared in 24 hours and fever in 48 hours. After a fortnight the heart was clinically and cardioscopically normal, but there was still slight elevation of the R-T interval, maximal in II and CR4. After a further seven weeks, when the patient had been working for a month, slight elevation of R-T in CR4 alone remained.

Comment

Both cases were at some stage, and with justification, diagnosed clinically as cardiac infarction. Apart from differential diagnosis, the general question of their mechanism may be raised. As will be observed from the earlier references, none has been suggested. It is also noted by Wolff (1944) that the prognosis is good, as is implicit in the terms "non-specific," "obscure," or "idiopathic" used by other authorities, since fatalities and necropsies would be likely to lead to some conclusion regarding their cause. A bacterial origin seems unlikely, since duration is brief and sequelae are rare. The cases cannot be regarded as "a chief manifestation of an infective process" (Lewis, 1946), such as acute rheumatism (Evans, 1948).

Another possibility arises, however, when the group simulating cardiac infarction is considered. The most likely simulator of a vascular lesion is another vascular lesion. In Case 1 there is evidence of activity of temporal or giant-cell arteritis, now recognized as a generalized process (Cooke *et al.*, 1946). The possibility has been suggested that this may affect the coronary vessels (Cole, 1948) or other large vessels (B.M.J., 1949). A reasonable explanation of the pericarditis in Case 1 seems to be arteritis of the vessels supplying the pericardium—branches of the internal mammary vessels or descending aorta—producing a pericardial infarction. Pericarditis or subepicardial infarction is known to occur in some cases of coronary occlusion, so that vascular lesions can produce such changes. On the other hand, they are not invariable in coronary disease, so that the subepicardial muscle derives part of its blood supply from elsewhere. The vessels providing the supply may be those damaged by the arteritis in Case 1, and possibly in "idiopathic" Case 2 some chance lesion may also have affected them. The rarity of an obvious general cause may be due to the fact that when there is generalized arterial disease the coronary vessels, exposed to constant and severe stresses in unintermittent response to changes in the internal and external environment, will fail earlier than the relatively sheltered vessels of the pericardium. These, on the other hand, may suffer from such diseases of capricious local incidence as temporal arteritis.

Summary

Two cases of acute pericarditis simulating cardiac infarction are reported, one associated with temporal arteritis. Pericardial infarction is suggested as a responsible mechanism.

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ADDENDUM

Since completing the above a further case has been observed and the article by Harrison and Wood (*Brit. Heart J.*, 1949, 11, 205) has been published. Their Fig. 9 illustrates development of anastomoses via pericardial vessels in coronary artery disease, by which means the blood supply of subepicardial muscle is presumably maintained. Increase of such pericardial anastomoses by talcum insufflation of the pericardium provides a rational therapy in coronary disease and has been performed on three of my cases, without mortality and with some symptomatic relief.

Medical Memorandum

Gangrenous Intussusception

This note records the successful resection of a gangrenous ileo-ileal intussusception in an infant of just under six months, on the third day of his illness.

CASE REPORT

The following pre-operative notes were kindly supplied by Dr. Michael Cutler:

"I first saw the patient at twelve noon on a Saturday, when the mother stated he seemed 'grievy' and not his usual self. He had twice vomited, first at 9 a.m. that morning and again about 11 a.m. After the operation the mother was again questioned and agreed that the child had an attack of screaming, with drawing up of the legs and pallor, at or about the time of the vomiting. It would appear the attack was neither prolonged nor violent, as the mother, an intelligent witness, thought it unworthy of mention when first questioned.

"The first examination was completely negative, except that the child looked rather pale. The bowels had not been open since the previous evening. I attributed the pallor to a severe infection, and fully expected diarrhoea and pyrexia to appear in a few hours. I instructed the mother to give 8 fl. oz. (227 ml.) feeds of half normal strength three-hourly. I saw the child again that evening. By then he had much improved: the pallor had gone and there was no more vomiting. For the second time I sought in vain for an abdominal tumour. The child slept all through Saturday night, and by Sunday morning the mother thought he had recovered. The failure of the bowels to act was attributed to the small feeds and vomiting. I again carefully examined the patient, but could find nothing wrong. I instructed the mother to continue diluted feeds and announced that I thought the trouble was nearly over. I left the child for twenty-four hours, making my next call on Monday morning. One glance at the child as I entered the room was enough—he was sweating, pale, and obviously very ill. The mother told me he had been vomiting severely since Sunday at 9 p.m. and there was a foul odour to the vomitus. I again examined the child and found no abdominal tumour, but the other signs were now conclusive."

This is a case of intussusception unrecognized until the third day. The failure in diagnosis is attributed to absence of the usual physical sign of blood per rectum, and to the striking absence of pain after the first day. There was only

one attack of screaming, and that was brief. The vomiting was not profuse until 9 p.m. on the second day. The apparent recovery early on the second day also contributed to the difficulty of diagnosis. The case exemplifies the need for vigilance, as emphasized by Morrison and Court (1948).

On admission, the child's temperature was 100.4° F. (38° C.), pulse 144, respiration 28. He looked pale and ill. The abdomen was resistant to palpation, but when the patient relaxed a firm, sausage-shaped mass could be felt lying across the right lower quadrant, fixed with the axis to the right and upwards. This mass was deep in the abdomen and did not harden with the pains. This failure to harden was explained by the subsequent finding of a gangrenous intussusception.

Operation.—Dr. A. H. Grace administered ethyl chloride and open ether. Pre-operatively a catheter was inserted into the stomach and a quantity of greenish fluid aspirated; the tube was kept in place during the operation. A right paramedian mid-abdominal incision was made, and revealed some clear free fluid. An irreducible, gangrenous intussusception of the small intestine was found. This was resected between clamps, and an end-to-end anastomosis was performed with two rows of interrupted catgut. A small oil-silk drain was inserted into the pelvis and left protruding from the wound, which was closed in layers.

The first day after operation the child was very restless, and vomited a considerable amount of green fluid. A catheter was passed into the stomach for hourly aspiration. He was passing urine normally. Intravenous glucose-saline was administered, and he was given 50,000 units of penicillin four-hourly for nine days. During this time the evening temperature reached 100–101° F. (37.8–38.3° C.). He received hypodermic injections of "nepenthe" 1 minim (0.06 ml.) as required, and 3 ml. of water hourly. On the second day after operation a quantity of stale and bright red blood was passed per rectum, and a little greenish fluid was withdrawn from the stomach. Plasma (200 ml.) was administered intravenously, followed by 200 ml. of Group O blood and normal saline.

On the third day about 200 ml. of gastric contents were withdrawn by hourly aspiration. This was a considerable increase, but the patient appeared to be much better in the afternoon. The intravenous drip was then discontinued and the bowels were opened with a rectal saline.

On the fourth day the patient was much better. Gastric suction was discontinued and he was given glucose by mouth. He was passing urine and yellow, loose motions, and he smiled for the first time since the operation.

On the fifth day he was well, and took small amounts of milk and glucose solution.

On the sixth day the wound discharged during the night. The child vomited one feed, and the bowels were opened. In the afternoon a faecal fistula formed, which closed after five days. Sulphamezathine (sulphadimidine) was given for four days, the dosage totalling 3.25 g. After this the patient became apyrexial, and his progress has since been uneventful.

Acknowledgment is due to Dr. J. McMurray for directing the post-operative treatment with untiring skill, to Dr. M. Graham, and to Sister I. A. Vincent and the nursing staff of the Royal East Sussex Hospital for their loving care, and to Mrs. W. Beaufre for her help in the arrangement of this account.

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Professor Frits W. Went, designer and director of the new Earhart Plant Research Laboratory in California, said that for the first time in the history of experimental biology it was now possible to grow plants under a wide variety of rigidly controllable conditions. All possible climatic factors, such as light, temperature, humidity, and gas content of the air, are simultaneously and independently controllable in the laboratory, which is to be used for experimental work by the California Institute of Technology.