Medical History

A Swedish report on acute myocardial infarction in 1859

B W JOHANSSON, P NICOL

Myocardial infarction is so common today that we easily forget that it is a disease that was recognised only relatively recently. Yet sudden death that could have been produced by an acute myocardial infarction was described as early as the sixth dynasty, more than 4500 years ago.¹

The term myocardial infarction was introduced in 1896 by the young French physician René Marie in his thesis "*L'infarctus du myocarde et ses conséquences*,"² but did not come into common use until after 1921, when Lian³ published his paper "*Le diagnostic clinique de l'infarctus du myocarde*." In the beginning of this century coronary thrombosis and coronary occlusion were the prevailing terms.

In the beginning of the nineteenth century isolated reports appeared in which the connection between stenosis of the coronary vessels and myocardial changes of the type we call infarction was suggested. It was not until 27 September 1859, however, that the Swedish physician J P Malmsten and the pathologist G W J von Düben gave at a meeting of the Swedish Medical Society the first full clinicopathological description of a myocardial infarction.⁴ It was a remarkable case, "coming very close to modern descriptions of myocardial infarction."⁵ The patient was a 66-year-old man who was taken ill with what we today would call typical retrosternal cardiac pain. He died after about a week. As Leibowitz⁶ states, "the information revealed in the postmortem report by von Düben goes far beyond a ruptured wall, long familiar through Harvey's case of Robert Darcy's postmortem finding in 1649." Von Düben described the clot in some detail, applied the word necrosis (which conforms to modern usage),^{4, 6} and described how the heart wall had become weakened into "a pulposus brittle mass, which at microscopical examination revealed destroyed muscle fibres substituted by a fine-grained, fat-intermingled detritus."⁴ He observed an old and partly canalised thrombus in the left descending coronary artery and concluded his report as follows: "There is no doubt that this already canalised clot had been formed a long time back and had gradually caused the softening described, as it would seem without any symptoms. The period that Doctor Malmsten's account of the illness embraces represents only the rupture; because in this case this did not, as is often the case, occur at a stroke, but over a period, with many smaller interruptions."⁴

Microscopy was von Düben's great contribution to the case. Its use in the study of coronary artery disease came quite late; indeed, von Düben seems to have been one of the pioneers.⁶

As Malmsten and von Düben's report is of such great interest in the history of myocardial infarction and as it is not, so far

as we know, easily accessible in any English translation,* we give now a translation of the complete report. (If the English seems old-fashioned and not entirely clear in places it is because the original Swedish is old-fashioned and in places not entirely clear, and we have tried to maintain the flavour of the original, and not to credit Malmsten and von Düben with observations they did not make and insights they did not have.)

Original 1859 report

Mr Malmsten reported the following case of "*ruptura cordis*." "The patient, 66 years old, lost his wife three months earlier and deeply mourned her death. Fairly fit for his years, although very sensitive to cold, he thought, when he began to feel ill on the Tuesday night, that he must have caught a cold during the day. On the Wednesday he felt cramp-like pains under the sternum, which increased periodically. He felt depressed. He had no fever. He felt better after a 'mustard dough.' When I saw him on Thursday his condition was unchanged. He walked up and down the floor, and complained of pain under the left nipple. His pulse was calm and regular, and on careful examination nothing could be detected in the heart, lungs, or abdomen. The diagnosis was obscure. He was prescribed a teaspoon of castor-oil. A plaster was laid on the side of his thorax, and he was given one teaspoon of a hydrocyanic emulsion two-hourly during the day.

"On the Friday the patient was better. He was told to rest and for anxiety a quarter grain of morphine was given in the evening. The patient rested in bed on Saturday, but dressed and got up on Sunday. On Monday he went out, but became worse in the evening, and I was called again on Tuesday morning. At my visit that morning the patient was resting, but the pains had increased. The pulse was weak and fast. The heart sounds were weak and distant, the 'weak sound' being somewhat increased over the heart. The anxiety was severe, yet the patient could sit up comfortably in bed. As he had not opened his bowels for three days a gentle laxative was prescribed. One hour after I had left, the patient was attacked by severe choking, his chest laboured severely, his face became blue, and, after a quarter of an hour of agony, he died.

"The postmortem was carried out the following day by Professor Düben, who will describe in more detail the heart, which was found to be ruptured. He died thus, in the literal meaning of the term, of 'a broken heart' (in English in the original).

"Concerning the necropsy Professor Düben reported the following: "The pericardial space contained 2 liber† of blood serum and approximately the same amount of very coagulated black blood. Near the apex of the left ventricle, about three-quarters of a thumb from it and half a thumb from the septum was a rupture of the heart-wall half a thumb long, like a slit, and filled with a clot. It went obliquely through the heart's outer muscle layer and pericardium. Its extension inwards could not be followed exactly, partly because of a clot closely applied to the inner surface, partly because of the peculiar nature of the wall. The above-named clot lay as a round disk, of about one-and-a-half thumb's diameter, closely applied to the endocardium and also between the trabeculae and out into the

*We, like Leibowitz,⁶ have been unable to locate a translation that appeared in the Dublin medical press. Perhaps one of our Irish colleagues can help us.

†1 liber = 356.28 g

Section of Cardiology, Department of Internal Medicine, General Hospital, S-214 01 Malmö, Sweden

B W JOHANSSON, MD, MRCP, head physician, section of cardiology

Medical Department, Köping Hospital, S-731 01 Köping

P NICOL, MB, MRCP, consultant physician

wall's cavities, so closely applied that its border could not be determined with certainty and it could not be freed therefrom. The surface of the clot facing the heart chamber was of normal pale red-golden colour, but lost this more and more the further it extended into the heart wall. There it became in patches grey-gold, light-red, and black-brown, in consistency sometimes firm, sometimes "puriform," and merged unnoticeably into the, in colour and consistency completely similar, heart wall. This was over the same area (one-and-a-half thumbs) softened to a pulposus, brittle mass, which at microscopical examination revealed destroyed muscle fibres substituted by a fine-grained, fat-intermingled detritus, but generally with preserved structure. In certain places were embedded blood clots that had come from inside. The nearer the ruptured area, the more easily noticeable became the necrotic change and it so fused with the inner part of the rupture that the extent of this consequently could not be determined with certainty. At examination the left coronary artery was tortuous and, partly because of an age-related atrophy of the heart and partly because of dilatation here and there, its walls were in places thinner and in others thicker than normal. The anterior branch contained an old clot, entirely white in colour and completely canalised in its upper end at least. It started a small distance from the border of the softened area and extended right in towards its centre, where it could no longer be followed and where it assumed a more obturating character and also was found to be puriform.

"There is no doubt that this already canalised clot had been formed a long time back and had gradually caused the softening described, as it would seem without any symptoms. The period that Dr Malmsten's history of the illness embraces represents only the rupture; because in this case, this did not, as is often the case, occur at a stroke, but over a period, with many smaller interruptions."

Other cases

This case of 1859 is not Malmsten's only contribution to the clinical recognition of myocardial infarction; in discussing a case of sudden death in 1861 he mentioned that one should consider thrombosis of a coronary artery.⁶

The report by Malmsten and von Düben remained largely unknown internationally, partly because it was published in Swedish and partly because the title "A case of ruptura cordis" was too general to be noticed as a case of myocardial infarction. Indeed, it probably remained unknown outside Scandinavia until E J Warburg reproduced it and translated it into German in his exhaustive paper of 1930.⁵ This is apparent from, for instance, Adam Hammer's case of 1878,⁷ which is often considered the first time that the disease was recognised as a clinical entity and the diagnosis made at the patient's bedside. Hammer was called into consultation by a doctor in doubt as to the diagnosis in a 34-year-old man (one is surprised at the patient's age) who was in a state of collapse with a very slow pulse.⁸ Hammer considered that the clinical features and course of the case could be explained only by a sudden interruption of the blood supply in one of the coronary vessels secondary to a thrombotic occlusion. When Hammer proposed this theory a colleague exclaimed: "I have never heard of such a diagnosis in my whole life." Hammer answered, "Nor I also" (sic).⁸ Hammer managed to get permission for a necropsy on his patient, which was sometimes difficult in the United States at that time, and this showed a thrombus occluding one of the coronary vessels. Unfortunately, Hammer's report, published in the *Wiener Medizinischer Wochenschrift*,⁷ also attracted little attention in the English-speaking world.

Other cases of myocardial infarction were published by two Russian clinicians from Kiev, Obrastzow and Straschesko, in a little-known Russian journal, *Russkii Vrach*,⁹ and later the same year, 1910, in a German journal.¹⁰

Not until after Herrick's description in 1912¹¹ was myocardial infarction accepted in the United States after an intensive information campaign.¹² In England the medical profession was difficult to convince. Sir Clifford Allbutt, the professor of medicine at Cambridge, discarded as late as 1915 the idea of disease of the coronary vessel producing angina pectoris, and Sir James Mackenzie was still not using the term "coronary

thrombosis" in the third edition (1913) of *Diseases of the Heart*, and referred to angina pectoris as a "symptom complex which exhausted the heart muscle."¹³

It is surprising how long it took the medical profession to accept myocardial infarction. It was not until the typical changes in the electrocardiogram were recognised that it became a more common disease. In 1859 Malmsten and von Düben held their case to be so unusual that it was regarded as worthy of publication: in 1978 the heart infarct register in Malmö, a town with 238 000 inhabitants, reported 770 new cases of acute myocardial infarction.

References

- 1 Bruetsch B. The earliest record of sudden death possibly due to atherosclerotic coronary occlusion. *Circulation* 1959;xx:438-41.
- 2 Marie R. *L'infarctus du myocarde et ses conséquences*. Paris, 1896.
- 3 Lian C. Le diagnostic clinique de l'infarctus du myocarde. *La Medicine* 1921:450.
- 4 Malmsten JP, Düben GWJ von. Fall av ruptura cordis. *Hygiea* 1859;21:629-30.
- 5 Warburg EJ. In: Leibowitz JO. *The history of coronary heart disease*. Los Angeles: University of California Press, 1970:115.
- 6 Leibowitz JO. *The history of coronary heart disease*. Los Angeles: University of California Press, 1970:116-7.
- 7 Hammer A. Ein Fall von thrombotischem Verschlusse einer der Kranzarterien des Herzens. *Wien Med Wochenschr* 1878;28:97-102.
- 8 Baldry PE. *The battle against heart disease*. Cambridge: Cambridge University Press, 1971:113.
- 9 Obrastzow WP, Straschesko ND. In: Leibowitz JO. *The history of coronary heart disease*. Los Angeles: University of California Press, 1970:147. (Original in *Russkii Vrach* (Russian journal).)
- 10 Obrastzow WP, Straschesko ND. Zur Kenntnis der Thrombose der Koronararterien des Herzens. *Zeitschrift für klinische Medizin* 1910;71:116-32.
- 11 Herrick JB. Clinical features of sudden obstruction of the coronary arteries. *JAMA* 1912;59:2015-20.
- 12 Herrick JB. *Memories of eighty years*. Chicago: University of Chicago Press, 1949.
- 13 Allbutt C, Mackenzie J. Cited after Baldry PE. *The battle against heart disease*. Cambridge: Cambridge University Press, 1971:115.

(Accepted 27 January 1982)

A young girl in a third world country had a kidney removed because of uncontrolled bleeding after renal biopsy. She was suspected of having IgA nephropathy, but now appears healthy. What is her prognosis, and should she take any special precautions?

It is always a matter of concern to hear about loss of a kidney after renal biopsy. In some cases this complication can now be better managed using arteriography and embolisation of the bleeding blood vessel.^{1,2} This procedure may control haemorrhage after biopsy and avoid the need for nephrectomy, which is very occasionally necessary to prevent exsanguination. Was the diagnosis of IgA nephropathy fully established with compatible histology on light microscopy and a predominance of IgA on immunofluorescence? When changes of IgA nephropathy are noted it is still not feasible to give a certain prognosis in the absence of more clinical details. If the indications for biopsy were recurrent haematuria separated by periods when the results of urine examination were totally normal a good prognosis can be expected. When patients with other presenting features are classified as having IgA nephropathy the prognosis is less consistent. Some patients do develop renal failure and other complications. If the patient and her urine are now normal, and there were no unusual features on the initial biopsy or in the clinical presentation, it would seem reasonable to consider her to have a good prognosis. No special precautions are needed in this or most other glomerulopathies as none are thought to influence progression or resolution. A very occasional check—for instance, every few years—to make certain that there are no abnormalities in the urine or blood pressure is perhaps advisable.—MARTIN S KNAPP, consultant physician and nephrologist, Nottingham.

¹ Young AE. Therapeutic embolisation. *Br Med J* 1981;283:1144-5.

² Curet P, Roche A, Riche MC, et al. Therapeutic embolisation for traumatic renal haemorrhage. *Nouv Presse Med* 1980;9:2633-6.