Significance of Persistent R-ST Elevation After Acute Myocardial Infarction

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It has been stated that R-ST segment elevation persisting more than six months after myocardial infarction is suggestive of ventricular aneurysm (Parkinson, Bedford, and Thomson, 1938; Nordenfeldt, 1939; Myers, Klein, and Hiratzka, 1948; Rosenberg and Messinger, 1949; Ford and Levine, 1951; Moyer and Hiller, 1951; Douglas, Sferazza, and Marici, 1962). Other authors maintain that there is no typical electrocardiographic finding associated with aneurysm (Sigler and Schneider, 1935; Schlichter, Hellerstein, and Katz, 1954; Abrams *et al.*, 1963). In the earlier descriptions of post-infarction cardiac aneurysm, R-ST elevation was noted but not discussed (Dressler and Pfeiffer, 1940), or was present and not commented upon (Kahn, 1922; Winternitz, 1934; Eliaser and Konigsberg, 1939).

During the past three years 23 patients have been seen in whom the electrocardiogram showed R-ST elevation more than six months after myocardial infarction. This paper reports an attempt to determine the significance of this persistent abnormality.

SUBJECTS AND METHODS

Studies were made on 23 patients of both sexes who had previously sustained a myocardial infarction and in whom the electrocardiogram showed persistent R-ST elevation. In all but one patient, the persistent R-ST elevation occurred in the anterior praecordial leads. In the exception the abnormality occurred in the inferior leads, III and aVF. Examinations were also made on 61 patients in whom the R-ST segment had returned to the iso-electric line after a rise associated with infarction.

More than six months had elapsed after the infarction before the investigations to be reported were undertaken.

Electrocardiograms were recorded on a Cambridge direct-writing machine; 12 lead tracings were recorded routinely.

Cardiac fluoroscopy was carried out on all patients by a method already described (Groden and James, 1968).

At the time of examination the electrocardiogram finding was not known to the radiologist. Particular attention was paid to the presence of abnormal contour, abnormal pulsation, and pleuro-pericardial adhesion, and the findings were recorded on oblique and lateral radiographs.

Abnormal contour (bulge) was defined as a localized prominence of the exterior surface of the heart. *Abnormal pulsation* was defined as an area of diminished or paradoxical pulsation compared with adjacent heart muscle. Since the assessment was made at fluoroscopy it is likely that such abnormalities occur more often than we have been able to record. *Pleuro-pericardial adhesion* was defined as a tent-shaped opacity with the base contiguous with the heart shadow.

RESULTS

Persistent R-ST Segment Elevation Group (23 patients)

(i) *Bulges.* These were found in 7 patients; some were obvious, and others minimal, being seen only in tangential projection. Five occurred on the anterior surface of the heart, and one on the inferior border. The position of all of these bulges corresponded to the electrocardiographic site of the infarct.

Two of these patients have since died, and necropsy revealed an aneurysm in each corresponding to the bulge detected radiologically. In one other case the diagnosis was confirmed at operation (Groden, James, and McDicken, 1968).

(ii) *Abnormal Pulsation.* This was seen in one patient. It occurred on the anterior border and its position corresponded with the electrocardiographic site of the infarct.

(iii) *Pleuro-pericardial Adhesion.* This abnormality was seen in 5 patients. It occurred on the anterior surface of the heart and corresponded to the electrocardiographic site of the infarct in every patient.

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No radiological abnormality was detected in 10 patients (Table).

TABLE

ABNORMALITIES IN 23 PATIENTS WITH PERSISTENT R-ST SEGMENT ELEVATION AND IN 61 PATIENTS WITH ISO-ELECTRIC R-ST SEGMENTS AFTER MYOCARDIAL INFARCTION

	Persistent R-ST elevation	R-ST iso-electric
Bulges	7	10
Abnormal pulsation	1	4
Pleuro-pericardial adhesion	5	8
No abnormality	10	39
Total	23	61

Note: None of these differences is significant at $p=0.05$ level (simple χ^2 test).

Iso-electric R-ST Segment Group (61 patients)

(i) *Bulges*. These were seen in 10 patients in this group; 8 were on the anterior surface and 2 on the inferior; and 7 corresponded to the electrocardiographic site of the infarct.

(ii) *Abnormal Pulsation*. This was observed in 4 patients; in 3 it was seen on the anterior border of the heart, and in the other patient it occurred at the apex; and 3 corresponded with the electrocardiographic site of the infarct.

(iii) *Pleuro-pericardial Adhesion*. This was found in 8 patients; 7 times on the anterior surface of the heart and once on the inferior surface. In 7 patients it corresponded to the electrocardiographic site of the infarct.

No radiological abnormality was detected in 39 patients (Table).

DISCUSSION

We agree with Holmes and MacFadyen (1964) that a bulge indicates an aneurysm. An area of abnormal pulsation may also represent an aneurysm, even in the absence of bulging of the contour of the heart.

In 13 patients a pleuro-pericardial adhesion was seen. Transmural myocardial infarction gives rise to pericarditis, and it is probably the end result of this process that is recorded. Moyer and Hiller (1951) and Holmes and MacFadyen (1964) have suggested that this may conceal a ventricular bulge on a radiograph. Our own observations in patients with post-infarction cardiac aneurysm tend to support this view.

Almost half of our patients with persistent R-ST elevation showed no radiological abnormality. It is possible that with more refined methods of

investigation, such as cinefluoroscopy, abnormalities would have been detected. Both Nordenfeldt (1939) and Moyer and Hiller (1951) have suggested that if the electrocardiogram remains unchanged for a long period after myocardial infarction, either chronic aneurysm or extensive fibrosis on the anterior wall of the left ventricle should be suspected. It is, therefore, possible that in some of the 10 patients in whom no radiological abnormality was shown there was extensive fibrosis of the anterior heart wall without actual aneurysm.

Several explanations of persistent R-ST elevation have been made. Ford and Levine (1951) put forward two suggestions: one is that there is an area of ischaemic subendocardial muscle present in a cardiac aneurysm, which an electrode within the ventricular cavity would detect as a persistent current of injury, and which is detected on the anterior surface of the heart through the "aneurysmal window"; the other is that the electrocardiographic abnormality represents the pericarditis which is present in 7 out of 10 hearts with aneurysm. Another explanation proposed by Moyer and Hiller (1951) is that the R-ST elevation is a result of hypertrophy of the surrounding ventricular muscle.

SUMMARY

Radiological studies were made for evidence of aneurysm on 23 patients who had sustained a myocardial infarction more than six months previously and in whom the electrocardiogram showed persistent R-ST segment elevation, and these patients were compared with 61 patients who had sustained an infarction and in whom the R-ST segment had returned to the iso-electric line. Approximately 1 patient in 3 who had persistent R-ST elevation showed a bulge (or aneurysm), and 1 patient in 6 showed a similar abnormality in the absence of R-ST elevation. This difference is not statistically significant.

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