TRANSIENT SEGMENTAL ASYNERGY OF THE LEFT VENTRICLE OF PATIENTS WITH VARIOUS CLINICAL MANIFESTATIONS POSSIBLY UNRELATED TO THE CORONARY ARTERY DISEASE

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Eight cases of transient reversible segmental asynergy of the left ventricle thought not to be related to coronary artery lesions are reported. Three cases were associated with inflammatory reactions of unknown origin, and one each with lactic acidosis, abdominal surgery, hypoglycemia, tetanus and pneumonia. None of the patients had symptoms suggestive of ischemic heart disease before or after these episodes. Electrocardiograms before these episodes were all normal. Two-dimensional echocardiography was performed to evaluate abnormal electrocardiograms. Coronary angiography was performed in 4 of 8 cases and was normal in all 4 cases; 2 done as emergencies and 2 non-emergencies. Two ergonovine tests were negative. Left ventricular wall motion abnormalities, present mainly at the apex of the left ventricle, returned to normal in 1 to 4 weeks. Giant negative T waves in the chest leads during this recovery period were characteristic electrocardiographic features and normalized in 6 weeks on average.

We believe that these episodes were not related to ischemia due to coronary artery disease, but to some metabolic humoral factors. An excellent prognosis can be expected if these abnormal metabolic circumstances can be resolved.

WHEN myocardial ischemia is not so severe as to induce myocardial infarction, transient reversible wall motion abnormalities have been termed stunned myocardium! In addition to ischemic heart disease, reversible left ventricular dysfunction has been reported in a variety of other conditions such as viral myocarditis, pheochromocytoma and sepsis.

We analyzed 8 cases of transient reversible left ventricular wall motion abnormalities

without history of ischemic heart disease, to clarify the possibility of metabolic impairment as the cause of these abnormalities.

SUBJECTS AND METHODS

We studied 8 cases of reversible left ventricular wall motion abnormalities observed by 2-dimensional echocardiography (2DE) without history of ischemic heart disease. Table I shows a summary of the clinical characteristics of these patients. This group included 3 men and 5 women with a mean age of 66 years. The electrocardiograms (ECG) before these episodes were normal in all pa-

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TABLE I PATIENT CHARACTERISTICS IN THIS STUDY

Case	Age	Sex	CRP (mg/dl)	max. CK (I.U.)	Wall motion abnormality in				Possible causes and comments
					Apex	AS	AL	P	
1. J.I.	73	M	6.3	1100	(+)	(+)	(+)	(-)	tetanus
2. S.O.	67	F	7.0	NI	(+)	(+)	(-)	(-)	abdominal surgery
3. S.T.	63	M	1.7	736	(+)	(-)	(-)	(-)	lactic acidosis
4. M.H.	64	F	6.4	1500	(+)	(+)	(+)	(-)	hypoglycemia
5. S.K.	49	F	4.6	NI	(+)	(-)	(-)	(-)	unknown
6. A.S.	60	F	5.6	NI	(+)	(-)	(-)	(-)	unknown
7. N.M.	78	F	3.2	NI	(+)	(-)	(-)	(-)	unknown
8. S.K.	74	M	14.4	NI	(+)	(+)	(+)	(-)	during bronchoscopy in pneumonia

CRP=C-reactive protein, CK=Creatine kinase, NI=not increased, AS=antero-septal, AL=antero-lateral, P=posterior

Fig.1. Upper panel: Left ventriculography showing large akinetic area in the antero-septal and apical area of the left ventricle. Ejection fraction was 35%. Coronary angiography done during this episode showed no significant stenosis.

Lower panel: M-mode and 2-dimensional echocardiography 8 weeks after this episode showed normal left ventricular motion (ED=end-diastole, ES=end-systole).

tients. The chief illnesses of these patients were not-cardiac and the 2DE was performed to evaluate abnormalities on routine ECG. In order to evaluate left ventricular segmental wall motion abnormalities, the left ventricle was grossly divided into 4 segments by 2DE; antero-septal, antero-lateral and posterior in the short axis view, and apex in the apical view. Coronary angiography was

performed on 4 of the 8 patients; 2 in an emergency to rule out acute myocardial infarction and 2 were performed electively. An ergonovine test was done in both cases of non-emergency coronary angiography. ECG and 2DE were followed serially until the abnormalities normalized. Creatine kinase (CK) was measured serially in all cases. In 5 of the 8 cases, the plasma

Fig.2. Serial electrocardiograms

A QS pattern was seen in V1 through V4 on admission 26 July. Diffuse T-wave abnormalities were seen in all chest leads on 17 Aug. and returned to normal on 17 Oct.

catecholamine level was measured in the chronic phase, and one during the acute episode.

RESULTS

Wall motion abnormalities were found in the antero-septal and apex in 4 cases and confined to the apex in 4 cases. Peak CK was less than 100 IU in 5 patients, 700 IU in one case, and greater than 1000 IU in 2 cases. Both patients whose CK was more than 1000 IU had had a generalized seizure immediately prior to examination.

Mean C-reactive protein (CRP) level was 6.2 mg/dl. The cause of this inflammatory process was unknown in 3 cases, while one other case was associated with each of lactic acidosis, abdominal surgery, hypoglycemia due to ACTH deficiency, tetanus and pneumonia. Left ventricular wall motion abnormalities all normalized within 1 to 4 weeks. In 6 patients, deep negative T waves were characteristically seen during the course of recovery. In one patient, the ECG was only followed for 2 months; however the

Fig.3. Upper panel: The ECG on 2/14 taken 2 days after surgery showed poor R wave progression and deep symmetric negative T waves in leads V1 through V3. The ECG later normalized.

Lower panel: Transient left ventricular aneurysm (ANE) in the antero-septal area and apex was seen on 2DE.

ECG normalized in 5.6 weeks on average in the other 7 cases. All 4 patients in whom coronary angiography was done had normal coronary arteries; in 2 cases it was done as an emergency procedure. The ergonovine provocation test was performed and was negative in both cases in whom coronary angiogram was done electively.

Case 1 (Fig. 1 & 2)

A 73-year-old man (J.I.) was admitted to our hospital with trismus due to tetanus. An electrocardiogram on admission showed ST segment elevation in all chest leads. 2DE showed severe akinesis in the antero-septal-lateral and apical area of the left ventricle. Emergent coronary angiography was done to rule out acute myocardial infarction and revealed normal coronary arteries. Peak CK was 1100 IU. 2DE study was repeated 8

Fig.4. Coronary angiography done 1 month after the episode was normal, as was the ergonovine test. Left ventriculograms showed normal wall motion.

Fig.5. Upper panel shows a large ventricular aneurysm on 2DE which had resolved in the lower panel (4 chamber view) (ES=end-systole, ED=end-diastole).

weeks after the acute episode and revealed normal left ventricular motion.

Case 2 (Fig. 3 & 4)

An emergency operation for radiation colitis was performed in a 67-year-old woman

Fig. 6. Serial electrocardiograms

An electrocardiogram was normal on 24 June, 1988. Low voltage and ST elevation in V2 through V5 was seen on the day of the episode, 24 Feb. On 15 March, giant negative T waves were seen in the chest leads which normalized 6 weeks after the episode on 22 May.

Fig. 7. Coronary angiography done 4 weeks after the episode was normal.

(S.O.). During the operation an anesthesiologist noticed elevation of the ST segments in I, aVL and modified V5 leads ECG. Serial ECG changes were compatible with acute antero-septal myocardial infarction, although CK values were not elevated. An antero-septal-lateral and apical aneurysm was seen in 2DE and had normalized 4

Fig. 8. 2DE showed a large ventricular aneurysm confined to the apex.

Fig. 9. Serial electrocardiograms:

An ECG on 7/6 was normal. An abnormal ECG on 7/25 prompted the 2DE. Deep negative T waves were seen in V4 to V5 one month after the episode on 8/20.

weeks later. Coronary angiography and left ventriculogram performed 4 weeks after the operation showed no abnormalities. The ergonovine provocation test was negative.

Case 4 (Fig. 5, 6 & 7)

A 64-year-old female (M.H.) was admitted to our hospital for loss of consciousness due to hypoglycemia caused by ACTH de-

iciency. As her ECG showed ST segment elevation in all chest leads, 2DE was done revealing a large ventricular aneurysm in the antero-septal and apical regions of the left ventricle. Peak CK was 1500 IU with a MB fraction of 3%. Coronary angiogram done 4 weeks after the episode was normal and the ergonovine provocation test was negative.

Case 7 (Fig. 8 & 9)

A 75-year-old female (N.M.) had some inflammatory reaction and her electrocardiogram showed ST-T changes. 2DE showed akinesis confined to the left ventricular apex. The motion abnormality had normalized 8 weeks later and giant negative T waves in the chest leads normalized in the course of recovery. Serum CK was not increased.

DISCUSSION

The present study group exhibited the following clinical characteristics. 1) There was no history suggestive of ischemic heart disease either before or after these episodes. 2) Abnormal segmental left ventricular motion, not coincident with coronary artery disease in 4 of 8 cases, normalized 1 to 4 weeks after these episodes in all cases. 3) The characteristic giant negative T waves in the chest leads were consistent with subendocardial ischemia during the recovery phase of stunned myocardium, but CK levels did not indicate myocardial infarction. The CK in 2 cases whose maximal CK was 1000 IU, was thought to be derived from skeletal muscle due to a prior convulsion. 4) All 4 cases in whom coronary angiography was done had normal coronary angiograms. The ergonovine test was done in 2 cases and was negative in both. 5) Some transient inflammatory process which was cleared in a few days was present during all of these episodes.

Renkin et al reported that persistently inverted T waves in the setting of unstable angina were associated with hypokinesis of anterior wall which was reversible 8 months after percutaneous transluminal coronary angioplasty of the left anterior descending artery? Severe coronary lesion is mandatory in this situation. In 4 out of the 8 patients in this study, these abnormalities of left ventricular wall motion could be explained by severe lesions of the left anterior descending

artery. Thrombus formation in the normal coronary artery is possible in these cases reported herein, although it is usually superimposed on atheromatous plaque. The ergonovine test is highly sensitive to the presence of vasospastic angina. While silent vasospasm of the coronary artery or complete recanalization of the occluded coronary artery cannot be ruled out completely, ischemic heart disease was not thought to be the cause of these reversible left ventricular wall motion abnormalities.

Segmental asynergy has often been seen in patients with myocarditis^{3,4}. In cases 5–7, increased CRP might have been associated with wall motion abnormalities confined to the left ventricular apex. The presence of silent viral myocarditis is possible, however viral myocarditis is usually associated with chest symptoms.

Pheochromocytoma has been associated with reversible left ventricular wall motion abnormalities unrelated to the coronary artery. The level of catecholamines is usually extraordinarily high in those cases, and catecholamine myocarditis has been documented⁵. In 5 of the 8 patients, the basal plasma catecholamine level was measured and was within the normal range. In case 1, the level during the episode was approximately 10 times the normal level, but may not have been severe enough to induce left ventricular dysfunction. Tetanus, hypoglycemia and other metabolic stimuli can also induce release of catecholamines from the adrenal gland. These levels usually increase left ventricular wall motion and were not high enough to induce wall motion abnormalities⁶.

Subarachnoid hemorrhage is associated with giant negative T waves in the course of recovery. In half of the victims, 2DE showed reversible segmental left ventricular wall motion abnormalities? The causes of this have been discussed and excessive secretion of catecholamine is thought to be one of the causes.

Reversible left ventricular dysfunction has been observed in a significant proportion of patients with septic shock⁸. This left ventricular dysfunction is thought not to be associated with decreased coronary blood flow, but rather to a metabolic humoral factor⁹.

Similar observations are reported as associated with renal failure!¹⁰ Some humoral factors have been suggested in these situations, rather than coronary events, as identified by administration of cocaine in the experimental dog model¹¹ and interferon alpha in AIDS!¹²

We believe that the transient reversible left ventricular wall motion abnormalities seen in this study group were associated with some metabolic impairment stated above, and thought not to be related to the coronary artery. The heart can be a target organ of some inflammatory processes by circulating some metabolite, and an excellent prognosis can be expected if the abnormal metabolic circumstances can be resolved.

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