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Significance of ST Segment Elevation in Electrocardiograms in Patients with Ruptured Cerebral Aneurysms

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Summary

Twenty-three patients with aneurysmal subarachnoid haemorrhage (SAH), who showed an ST segment elevation in their electrocardiograms (ECG), were examined.

There were 12 males and 11 females, with a mean age of 61 years. The clinical condition on admission was Hunt and Kosnik grade II in four, III in seven, IV in one, and V in 11 patients. Computerized tomography (CT) also revealed many cases of diffuse, thick SAH or intracerebral or intraventricular haematoma. Laboratory examinations including serum electrolyte, pH, and PaO_2 revealed no abnormalities that might have influenced the ECG. Elevation in the levels of myocardial enzymes in serum was observed in two of the nine patients examined, although the elevation was only slight in one of them. Echocardiography, which was performed on several occasions on all patients, and cardiac catheterization, which was performed on eight patients, revealed a reduction in the motion of the left ventricular apex that was synchronous with ST segment elevation. This is the first report about these phenomena. No abnormalities were observed in the coronary artery. The elevated ST segment was normalized within one week in all patients, accompanied by normalization of the apical wall motion recorded on echocardiograms. In four patients, however, T wave inversion accompanied the improvement of the ST segment and was normalized within three months after the onset.

These results suggest that ST segment elevation in the acute stage of SAH reflects transient cardiac dysfunction rather than myocardial injury. In some patients, however, the elevated serum levels of myocardial enzymes or T wave inversion suggested the presence of myocardial injury. Close follow-up seems to be necessary in such cases

Keywords: Aneurysm; cardiac catheterization; electrocardiogram; subarachnoid haemorrhage.

Introduction

Byer *et al.*[2] were the first to report electrocardiographic changes in the acute stage of aneurysmal subarachnoid haemorrhage (SAH). Subsequent clinical[1, 4, 5, 7, 8, 11–14, 17–19, 21] and experimental[9] studies revealed changes such as QT prolongation, T wave abnormalities, ST segment elevation or depression, appearance of the U wave, and arrhythmias. However, the pathophysiology of these changes remains unclarified.

There are few reports on cardiac catheterization of SAH patients in the acute stage. Of the above changes, ST segment elevation is usually interpreted as reflecting transmural myocardial injury. This change is extremely important in treating patients with SAH, because treatment for ruptured cerebral aneurysms can differ greatly, depending on whether ST segment elevation reflects vasospasm of the coronary artery due to SAH-induced autonomic imbalance or incidental acute myocardial infarction caused by organic obstruction of the coronary artery. It is also possible that ST segment elevation reflects a new pathological condition which has not yet been reported. If consciousness is disturbed, patients with such a disease may not complain of headache or chest pain. Before the era of computerized tomography (CT), SAH was sometimes misdiagnosed as myocardial infarction[18].

Thus, in the present study we conducted a retrospective analysis of the clinical significance of ST segment elevation, which is one of the changes observed in the electrocardiogram (ECG) after the onset of SAH.

Clinical Material and Method

The subjects of the present study were 23 patients who had ST segment elevation at the time of admission (Table 1). These 23 patients were 10% of the 226 patients with SAH due to ruptured aneurysms who were treated at our center over a six-year period.

The mean time from onset to admission was 3.5 hours. Eleven patients had a history of hypertension, and two of angina pectoris. In patients with hypertension after admission, blood pressure was controlled by intravenous injection of a calcium antagonist.

Table 1. *Summary of Clinical Data on Patients with ST Segment Elevation*

Case no.	Age (yrs) Sex	Duration from onset to admission (hrs)	Grade on admission[a]	CT Group[b]	Site of ruptured aneurysm	Operation	Symptomatic vasospasm	Outcome[c]
1	59, M	4.8	II	3	lt. MCA	done	no	GR
2	53, F	24.0	II	3	distal ACA	done	no	GR
3	57, M	10.5	II	3	unknown	not done	no	GR
4	44, M	0.8	II	2	rt. ICA	done	no	GR
5	64, M	1.5	III	4	rt. MCA	done	no	GR
6	47, M	2.2	III	3	rt. MCA	done	yes	GR
7	50, M	2.4	III	3	rt. VA	done	no	GR
8	61, M	0.4	III	3	ACoA	done	no	GR
9	71, F	3.3	III	2	ACoA	done	no	MD
10	45,M	2.5	III	3	rt. VA	done	no	MD
11	80, F	2.2	III	4	ACoA	done	yes	SD
12	48, M	2.2	IV	4	ACoA	done	yes	D (day 13)
13	73, F	0.3	V	4	ACoA	done	no	MD
14	50, M	0.5	V	4	rt. MCA	done	no	SD
15	34, M	0.5	V	4	lt. MCA	done	no	VS
16	87, F	1.1	V	3	not examined	not done	unknown	D (day 9)
17	67, F	0.4	V	3	not examined	not done	unknown	D (day 4)
18	80, F	0.5	V	3	not examined	not done	unknown	D (day 10)
19	79, F	0.7	V	3	non filling	not done	unknown	D (day 3)
20	53, F	0.5	V	3	not examined	not done	unknown	D (day 5)
21	77, F	0.4	V	3	not examined	not done	unknown	D (day 4)
22	50, M	11.0	V	4	ACoA	done	yes	D (day 31)
23	69, F	6.8	V	4	not examined	not done	unknown	D (day 8)

ACoA anterior communicating artery; *ACA* anterior cerebral artery; *ICA* internal carotid artery; *MCA* middle cerebral artery; *VA* vertebral artery.

[a] Clinical condition on admission graded according to Hunt and Kosnik[15].

[b] CT groups defined according to Fisher *et al.*[10].

[c] Outcome measured by the Glasgow Outcome Scale[16]: *GR* good recovery; *MD* moderate disability; *SD* severe disability; *VS* vegetative state; *D* death.

ST segment elevation was regarded as being significant if the elevation was 0.5 mm or more in extremity leads, 3 mm or more in thoracic V 1–2 leads, or 1 mm or more in V 3–6 leads.

Cerebral angiography was performed in 17 patients under adequate sedation. Ruptured aneurysms were confirmed in all but two of them. In one of the latter, the aneurysm was not detected by 4-vessel study, and the other patient showed complete cessation of the intracranial circulation. In the remaining 6 patients, their general condition did not permit angiography, but signs of ruptured aneurysm were detected by CT.

In all patients, the following parameters were determined on admission: clinical grade (according to Hunt and Kosnik[14]); CT group (according to Fisher *et al.*[11]); serum Na, K, and Ca; PaO_2; and pH. In 9 patients, serial determinations of creatinine phosphokinase levels along with myocardial iso-enzyme fractions (CK-MB) were also examined (case nos. 4, 7, 9, 11, 13, 14, 19, 22, 23). Echocardiography was performed every 6 hours during the first 2 days and once a day until discharge in all patients. In 8 patients whose family gave consent, coronary angiography and left ventriculography via the femoral artery were performed by the Seldinger technique simultaneously with cerebral angiography (case nos. 5, 7, 8, 10, 11, 15, 19, 22).

Direct operations for ruptured aneurysms were performed on 15 patients following angiography, after myocardial infarction had been ruled out. No patients showed rerupture or aggravation due to angiography.

Results

1. Clinical Characteristics

Twelve patients were males and 11 were females, with a mean age of 61±14 years (Table 1). The most frequent site of ruptured aneurysms was the anterior communicating artery (six patients), followed by the middle cerebral artery (five patients). The clinical grade (Hunt and Kosnik) on admission was II in four, III in seven, IV in one, and V in 11 patients. Thus, the clinical condition was often severe. As for CT grouping, two patients were assigned to group 2, 13 to group 3, and eight to group 4. This means that diffuse, thick SAH or intracerebral or intraventricular haematoma was often seen in patients with ST segment elevation. Seven patients died rather early after rupture of their aneurysms, and symptomatic vasospasm of the cerebral artery was seen in four (25%) of

Table 2. *Site of ST Segment Elevation in Electrocardiogram*

Case no.	I	II	III	aVR	aVL	aVF	V_1	V_2	V_3	V_4	V_5	V_6
1									○	○	○	
2									○	○	○	
3								○	○	○		
4	○				○					○	○	○
5		○	○			○			○	○	○	○
6	○				○			○	○	○	○	○
7	○				○					○	○	○
8	○				○			○	○	○	○	○
9	○				○				○	○	○	○
10	○				○				○	○	○	○
11	○				○			○	○	○	○	○
12								○	○			
13	○				○			○	○	○	○	
14	○				○						○	○
15		○	○			○		○	○	○	○	○
16								○	○	○		
17	○				○				○	○	○	
18	○				○				○	○	○	
19	○	○							○	○	○	○
20		○	○			○						
21	○				○					○	○	○
22	○				○				○	○	○	○
23							○	○	○	○		

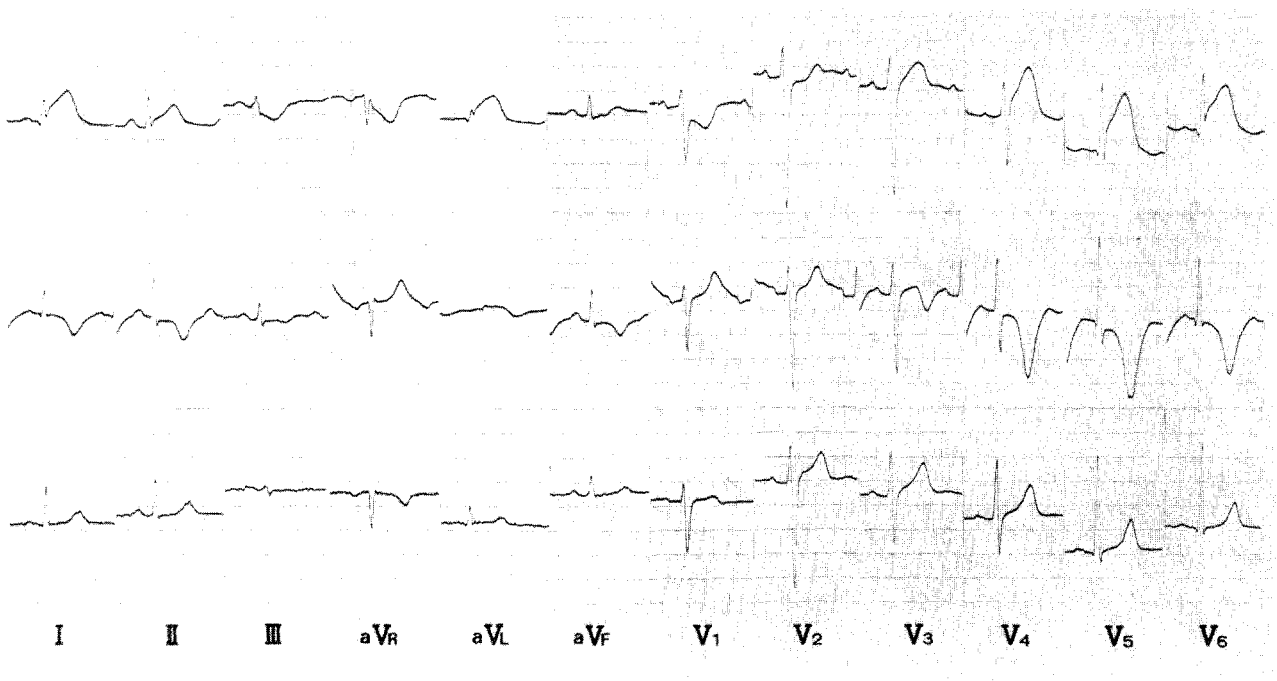

Fig. 1. Serial electrocardiograms (ECG) recorded during the clinical course in case no. 7. Top row: ECG at the time of admission showing ST segment elevation in leads I, aVL, V_4, V_5, and V_6. Middle row:ECG taken on the day following admission showing improvement of the ST segment and T wave inversion in all leads except V_1 and V_2. Bottom row: ECG at three months after the onset showing normalization of the ST segment and T wave

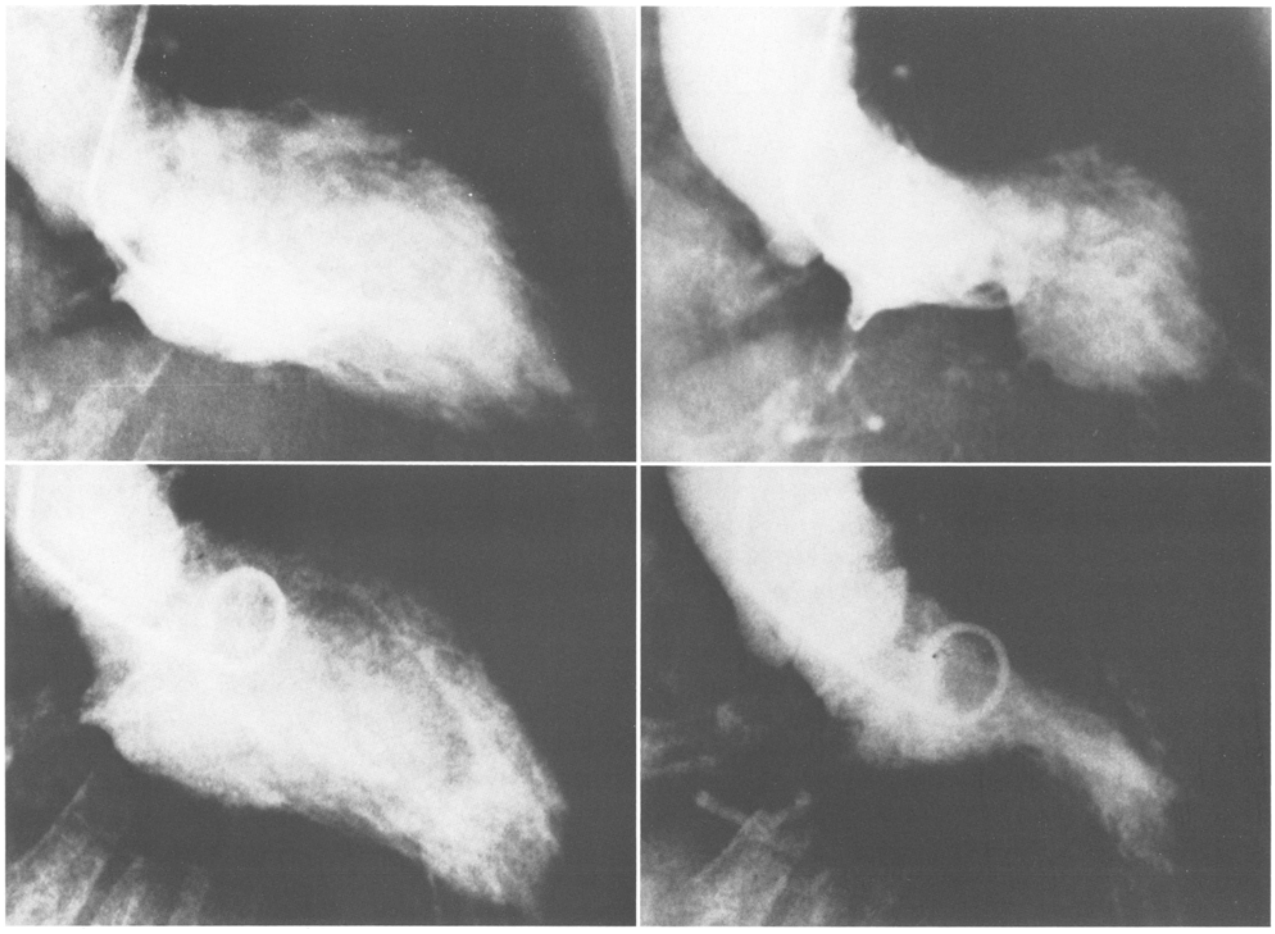

Fig. 2. Left ventriculography at the time of admission (upper left, diastolic; upper right, systolic) and at day 18 (lower left, diastolic; lower right, systolic) in case no. 11. Upper right showing akinesis of left ventricular apex and lower right showing normalization of apical motion

the remaining 16 patients, two of whom died later. Of the nine patients who died, eight died of primary brain damage due to SAH and one died of cerebral vasospasm. Eight of the nine who died belonged to grade V.

2. Laboratory Examination

Electrolyte levels on admission were as follows: Na, 139 ± 3.7 mmol/l; K, 3.7 ± 0.6 mmol/l; Ca, 8.7 ± 0.5 mg/dl; and the pH value was 7.4 ± 0.1. Although PaO_2 was under 60 mmHg in 2 patients on admission, their hypoxia improved just after oxygen inhalation. When serial determinations of serum CK-MB were examined in nine patients, the value of this parameter rose during the course of follow-up in two of them. Of these two patients, one (case no. 22) showed an elevation in this parameter to 192 U/l (normal value is under 10 U/l) at 24 hours, although it returned to the normal range by 72 hours. The other patient (case no. 7) showed a peak level of 43 U/l. This parameter continued to be within the normal range in the remaining seven patients.

3. Electrocardiograms

Table 2 shows changes in the ECG. Signs of injury of the lateral wall or anteroseptal region of the left ventricle were often observed. ST segment elevation was normalized within 24 hours in 14 patients, within three days in five, and within a week in the other patients. In 4 patients (case nos. 7, 11, 13, 17), T wave inversion accompanied normalization of the ST segment, but returned to normal within a week after the onset in two patients and after three months in one (Fig. 1). Another patient could not be judged in this regard because she died on day 4.

4. Echocardiography and Cardiac Catheterization

In all patients, echocardiography was performed on several occasions. During ST elevation, the basal

part of the left ventricle was hyperkinetic, while the apex was hypokinetic or akinetic. These abnormalities disappeared as the ST segment became normalized. Cardiac catheterization, performed in 8 patients, did not reveal any spasm or obstruction of the coronary artery. Like echocardiography, left ventriculography visualized a hyperkinetic basal part and a hypokinetic or akinetic apex. In one case (case no. 11), a follow-up left ventriculogram was obtained, and the normalization of the left ventricular motion was recognized (Fig. 2).

Discussion

In the present study, vasospasm or obstruction of the coronary artery did not seem to play any role in the ST segment elevation in patients with SAH. Instead, we found that hypokinesis or akinesis of the left ventricular apex was involved in the elevation. In other words, the present study disclosed that ST segment elevation is accompanied by a reduction of motion at the apex, although the wall motion in the basal part of the left ventricle is enhanced during the elevation. This abnormal motion at the apex disappeared as the ST segment became normalized, suggesting that ST segment elevation is caused by this abnormal motion. As far as we know, there is no previous report about these phenomena. Although Pollick *et al*[21]. reported the time course of post-SAH left ventricular wall motion, as examined by echocardiography, their study was not confined to ST segment elevation; and they did not pay attention to wall motion in the basal part or apex of the heart. ST segment elevation is usually thought to reflect a transmural myocardial injury in the corresponding region. Indeed, myocardial injury is often found at autopsy of patients who had died after SAH[3, 12, 13]. In the present study, myocardial injury appeared to be absent clinically except for those showing an elevated CK-MB or T wave inversion. Although Yuki *et al.*[22] regarded such ECG abnormalities as representing stunned myocardium due to a coronary vasospasm, this interpretation does not seem to be valid in view of the present findings. They also performed coronary angiography, but it was done at 30 days after the onset. ECG abnormalities in the acute stage of SAH are usually attributed to autonomic imbalance, in particular to direct autonomic discharge to the heart or to increased levels of circulating and tissue catecholamines[8, 9]. It has been suggested that stimulation of the hypothalamus by SAH elevates serum catecholamines and excites the sympathetic nerves, leading to the onset of myocardial necrosis if stimulation becomes excessive[8]. However, if humoral factors such as catecholamines are mainly involved in this condition, it is unlikely that wall motion would differ between different regions of the heart, as seen in our patients. And these regions also did not correspond with the territory of the coronary arteries. Although the blood supply from the endocardial side cannot be identified, these phenomena cannot be explained in terms of the blood supply. Regarding the regulation of the heart by the sympathetic nerves, Pierpont *et al.*[20] found in dogs that the level of norepinephrine in the left ventricular myocardium was lower at the apex than in the basal part. They interpreted this finding as reflecting a difference in sympathetic nerve distribution between different sites of the heart. Although control of the heart by the central nervous system is not defined in detail, it is possible that our results relate to this finding.

The incidence of ST segment elevation in the acute stage of SAH was reported to be 0.8% (1/120) by Di Pasquale *et al.*[5] and to be 12.5% (6/48) by Kreus *et al.*[17]. In the present study, it was 10% (23/226). Thus, the incidence of ST segment elevation in the acute stage of SAH varies appreciably among reports. This difference may be attributed to differences in the populations studied. Our study included many patients in the very acute stage and in the severe state. The sites of ruptured aneurysms are reported to have no association with the onset of arrhythmias[1].

The present study suggests that in patients with ST segment elevation, the sites of ruptured aneurysms are dominant in the anterior communicating artery and middle cerebral artery.

Mortality was high in this study. This is because our institution (a critical care medical center) accepts many patients who arrive in a poor condition. Some investigators suggested a poor prognosis in patients showing ST segment elevation on the grounds that the incidence of cerebral vasospasm is high in such patients[4]. In the present study, however, no correlation was observed between ST segment elevation and cerebral vasospasm. Cardiac dysfunction did not appear to have adversely affected the prognosis in any of our patients. However, further studies are needed regarding the influence of cardiac dysfunction on the intracranial environment. An important finding from the present study is that ST segment elevation was sometimes observed even when the clinical condition was not poor and SAH was mild as judged on the CT scan. Electrocardiographic abnormalities such as ST

segment elevation require particular attention in patients with acute SAH.

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