A Case of Myocardial Infarction:
Caused by the Dissecting Aneurysm of the Aorta

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Although many cases of the dissecting aneurysm of the aorta have already been reported, the dissection rarely extends to the ostium of the coronary artery and causes myocardial infarction. Only several cases with such a complication could be found in the literature. The present report describes a case of the dissecting aneurysm of the aorta complicated by acute myocardial infarction.

Case Report

A 60 year old man, college professor, was admitted to the Tokyo University Hospital because of the attack of acute myocardial infarction. Family history revealed that father died of anginal attack. Past history showed nothing contributory except for slight hypertension of 150/90. He had not had the episodes of chest pain. A few days previously he had felt back pain and visited home doctor, whose diagnosis had been intercostal neuralgia. On 27, Sept. '67, he was discussing an important problem from 1:00 p.m. to 5:00 p.m., attending the meeting at the college. He said his opinion just before the meeting was over. Soon after his speech, he developed nausea and left precordial oppressive pain radiating to the left shoulder. He was carried to the medical office of the college. At that time, pulse rate was 90 per minute, regular. Blood pressure was 88/0. Electrocardiogram showed a sinus rhythm with ventricular premature beats, marked left axis deviation and elevated ST segments and peaked T waves in leads I, aVL and V1 through V6 with reciprocal ST, T changes in leads II, III and aVF (Fig. 1). The diagnosis of acute myocardial infarction was made and he was transferred to the Tokyo University Hospital at 6:40 p.m. Physical examination on admission showed a dyspneic, restless man complaining severe chest

![Electrocardiogram](image-url)

*Fig. 1. Electrocardiogram taken at 6:00 p.m. on 27, Sept. showing elevated ST segments and peaked T waves in leads I, aVL and V1 through V6 with reciprocal ST, T changes in leads II, III and aVF.*
pain. Consciousness was clear. Skin was cold with sweating. Face was pale. Pupils were miotic without anisocoria. Light reflexes were prompt. Pulse rate was 140 per minute, weak and irregular. Blood pressure was 80/0. No heart murmur or vascular bruit was audible. Moist rales were heard on the right lung. There was no pretibial edema. Reflexes were normal and no spinal or cerebral signs were observed. Examination of blood revealed a white cell count of 16000. Serum CPK taken 3 hours after the attack was 33.1 units which was slightly elevated. Urinalysis showed a positive test for protein without other abnormal findings. After admission he was put into an oxygen tent. Vasoconstrictive drugs were administered by intravenous drip infusion. Opiates were used to relieve pain. There was, however, no improvement. Electrocardiogram at 10:00 p.m. showed a sinus rhythm at a rate of 125, QRS duration of 0.17 second with left bundle branch block, decreasing R waves from lead V1 to V3 and QS pattern in lead V5. Gradually he developed pulmonary edema and died at 0:35 p.m. on 28, Sept., 19 hours after the attack. His chest X-ray was not taken because of severe condition. But we could get his X-ray taken 6 months previously which showed no abnormal findings except for slight elongation of the aortic arch and enlargement of the left ventricle.

The findings of the postmortem examination were as follows. The ascending aorta showed subadventitial hemorrhage. The longitudinal section of the ascending aorta revealed a horizontal tear of the intima about 3 cm in length, 2 cm above the aortic ring which formed the dissecting aneurysm in the media. The dissection extended to a distance of about 1 cm from the orifice of the left coronary artery and compressed its ostium (Fig. 2). Microscopic examination revealed no findings of idiopathic cystic medionecrosis. The aorta and coronary arteries showed little atherosclerotic changes. Heart weighed 400 gm. Anterior half of the left ventricular wall and septum showed changes of fresh myocardial infarction. Marked congestion of lungs, liver and kidneys were found.

**Discussion**

This is a case of acute myocardial infarction, the pathogenesis of which is the dissecting aneurysm of the aorta which extended to the left coronary artery and compressed its ostium.

Only several cases of myocardial infarction with the same origin could be found until now. In 1938 Weiss reported a forty-year-old male who developed sudden breathlessness and then sharp burning epigastric pain. The chest X-ray showed widening of the aorta. The electrocardiogram taken on admission was interpreted as indicating myocardial disease. The patient died 2 days after admission. The postmortem ex-

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Fig. 2. Transverse section of the left coronary ostium showing compression of its lumen by the dissected media.
(Weigert—van Gieson stain; \( \times 8 \))

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amination revealed a rupture in the intima of
the aorta about 1 cm above the aortic ring which
extended to the orifice of the left coronary ar-
tery and compressed its ostium. The myocar-
dium of the left ventricle showed the signs of
recent myocardial infarction.

WAINWRIGHT described a similar case in
1944. A forty-two-year old female was sudden-
ly seized with excruciating epigastric pain
which radiated to the chest, right arm and back.
The serial electrocardiogram revealed classical
"coronary T-wave" pattern which was diag-
nostic of recent anterior myocardial infarction.
She died on the eighteenth day. The postmor-
tem examination showed a transverse rupture
in the intima of the aorta about 1 cm above the
sinus of Valsalva, which extended along the left
coronary artery. An extensive fresh infarction
of the myocardium was seen grossly.

A similar case was also reported lately. A
sixty-eight-year-old woman developed severe
dyspnea and tightness across the chest and lost
consciousness. The hemiparesis on the left side
was observed. The left carotid pulse was
stronger than the right side and a short bruit
was audible over the right carotid artery. On
the third hospital day, the electrocardiogram
showed markedly elevated ST segments in leads
II, III and aVF. She died on that day. The postmortem examination revealed a large hori-

tzontal intimal tear about 5 cm above the aortic
ring which extended distally into all the bran-
ches of the arch. The mouth of the right coro-
nary artery was compressed by the dissection
and the heart showed the findings of recent
myocardial infarction.

In the comprehensive review of the dissect-
ing aneurysm of the aorta in 1967, LINDSAY
referred to a case of myocardial infarction, the
cause of which was coronary occlusion by the
dissection. But the details of the clinical course
were unknown.

All these patients developed chest or epiga-
stric pain without the electrocardiographic signs
of myocardial infarction at first. These signs,
however, appeared later and the patients died
of myocardial infarction. Autopsy findings re-
vealed the dissecting aneurysm of the ascending
aorta which extended to the ostium of the coro-
nary artery. The compression of the coronary
ostium was thought to be the cause of myocar-
dial infarction in these cases.

The present case, however, is somewhat dif-
ferent clinically although the autopsy findings
were the same as those cases mentioned above.
The patient developed chest pain and became
seriously ill soon. At that time the electrocar-
diogram showed the typical signs of acute myo-
cardial infarction.

From the clinical diagnostic standpoint, when
the patient develops the dissecting aneurysm at
first and then myocardial infarction, we may
possibly make diagnosis of both diseases clini-
cally. The presence of neurological signs such
as hemiplegia or anterior spinal artery syn-
drome suggests the dissecting aneurysm. But
when the myocardial infarction occurs at the
same time, the diagnosis of the dissecting
aneurysm is thought to be impossible because
the symptoms and signs of the dissecting
aneurysm are masked by those of myocardial
infarction.

The involvement of the coronary artery by
the dissection of the aorta does not always
induce myocardial infarction. These cases were
reported by LAWRENCE and BAYLEY.

The distribution of the dissection in the aorta
indicated the frequent involvement of the ascen-
ding aorta. LINDSAY reported that 65 per
cent of 62 cases of the dissecting aneurysm in-
volved the ascending aorta. In HIRST'S series
of 443 cases, the ascending aorta was involved
in 60 per cent. The most common cause of
death of these cases is hemopericardium induced
by external rupture. Hemothorax and hemo-
mediastinum are complicated very often. But
as a cause of death, myocardial infarction is
very rare. In LINDSAY's cases only one died of
myocardial infarction, the cause of which was
coronary occlusion by the dissection. HIRST referred to 5 cases of coronary artery involve-
ment and SUGAI also referred to one case of
angina pectoris, but further details were un-
known in these cases.

The cause of rarity of complicated coronary
involvement in the dissecting aneurysm of the
aorta is not clear. WAINWRIGHT suggested that
the proximity of the coronary orifices to the
reflection of the pericardium on the aorta might
prevent dissection extending to the coronary

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artery. It is also conceivable that the mechanical forces of blood flow to the wall of the aorta play an important role. The dissection extends more easily to the distal portion and rarely extends against blood flow to involve coronary ostium when it occurs above the aortic ring.

SUMMARY

A case of acute myocardial infarction caused by the dissecting aneurysm of the aorta which extended to the ostium of the left coronary artery was described. As a cause of death of the dissecting aneurysm of the aorta, myocardial infarction is very rare. Several cases of myocardial infarction with the same origin were reviewed.

REFERENCES