Clinical Implications of Electrocardiograms for Patients With Type A Acute Aortic Dissection

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Type A acute aortic dissection (AAD) is a serious cardiovascular emergency requiring urgent surgery. Timely accurate diagnosis is essential, but often challenging, because of the wide spectrum of clinical presentations. In patients with type A AAD, chest pain is the most common symptom; furthermore, ischemic ST-T changes such as ST-segment elevation or depression or negative T waves are frequently observed on presentation ECG. These clinical presentations of type A AAD are difficult to differentiate from those of acute coronary syndrome (ACS), which could lead to delayed diagnosis and treatment of type A AAD or misdiagnosis of ACS followed by inappropriate treatment. Of note, ischemic ST-T changes have been shown to be associated with poor outcomes in patients with type A AAD. Because ECG is simple, inexpensive, noninvasive, readily available, and rapidly interpretable at the time of presentation, risk stratification based on ECG findings is considered very useful clinically. ECG findings of type A AAD thus have clinically important diagnostic, therapeutic, and prognostic implications; however, the relationships among these factors remain poorly understood. We review the prevalence of ECG abnormalities, clinical features associated with such changes, and the prognostic importance in patients with type A AAD.

Key Words: Aortic dissection; ECG; Mortality; ST-segment

Type A acute aortic dissection (AAD) is a cardiovascular emergency, in which serious complications, such as shock, cardiac tamponade, or coronary artery involvement causing acute myocardial ischemia, are initially present or develop subsequently.1–8 Timely accurate diagnosis is essential, but is often challenging because of the wide spectrum of clinical presentations.1,3,9–14 In patients with type A AAD, chest pain is the most common symptom, occurring in 64–82% of patients,1,3,12,14,16 and the most important differential diagnosis is acute coronary syndrome (ACS) because the therapeutic strategy differs considerably.1,11,13,14 ECG is initially performed for diagnosis, but ischemic ST-T changes such as ST-segment elevation or depression or negative T waves are frequently observed on the presentation ECG in patients with type A AAD (Figure 1).1,4,11–19 In addition, cardiac troponin, a well-known diagnostic marker of myocardial damage in ACS,20 is elevated in up to 25% of patients with type A AAD.14 Such clinical presentations of type A AAD are difficult to differentiate from ACS, which can lead to delayed diagnosis and treatment of type A AAD2 or misdiagnosis of ACS followed by inappropriate treatment.1,2,13,16,17,21–23

Of note, ischemic ST-T changes are associated with serious complications, and are significant and independent predictors of poor outcomes in patients with type A AAD.4,15–17,19 Type A AAD carries a very high mortality rate in the absence of surgical treatment, and therefore immediate surgical repair is needed.1,5,7,8,14,18,19 With advances in operative methods, anesthetic techniques, and perioperative management, surgical outcomes continue to improve, but the morbidity and mortality rates remain high.5,8,14,18 Prompt identification of patients at increased risk of death is crucial for the appropriate management of type A AAD. Because ECG is simple, inexpensive, noninvasive, readily available, and rapidly interpretable at the time of presentation, risk stratification based on ECG findings is considered very useful clinically. Thus, the ECG findings of type A AAD have clinically important diagnostic, therapeutic, and prognostic implications; however, the relationships among these factors remain poorly understood. In this review, we discuss the prevalence of ECG abnormalities, clinical features associated with such changes, and the prognostic implications in patients with type A AAD.

**ECG Findings at Presentation of Type A AAD**

**Normal ECG**

Many previous studies have reported that normal ECG findings or no significant ST-T changes are found in approximately 30% of patients with type A AAD (Table).1,4,7,11,15,17,19

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Figure 1. Representative ECGs of ST-T abnormalities. (A) An example of ST-segment depression/negative T waves. This patient had pericardial effusion on admission, but not coronary artery involvement, which was documented at the time of surgery. (B) An example of ST-segment depression/negative T waves. This patient had cardiac tamponade on admission, but not coronary artery involvement, which was documented at the time of surgery. (C) An example of ST-segment depression/negative T waves. ST-segment elevation was also observed in lead aVR. This patient had cardiac tamponade and moderate aortic regurgitation on admission, and ostial involvement of the left coronary artery, which was documented at the time of surgery. (Reproduced with permission from Kosuge M, et al.) (D) An example of ST-segment elevation. Complete atrioventricular block was present on admission. This patient had ostial involvement of the right coronary artery, which was documented at the time of surgery. (Reproduced with permission from Kosuge M, et al.) (E) An example of ST-segment elevation. ST-segment elevation was also observed in lead aVR. This patient had shock, cardiac tamponade, and moderate aortic regurgitation on admission, and ostial involvement of the left coronary artery, which was documented at the time of surgery.
Left Ventricular Hypertrophy (LVH)

Hypertension is considered the most common risk factor associated with AAD.\textsuperscript{1,4,6,7,11,12,15-18} LVH on ECG is observed in approximately 25% of patients with type A AAD.\textsuperscript{1,2,4,14,16,17} Concomitant LVH makes the ECG diagnosis of myocardial ischemia difficult.\textsuperscript{14} In patients with

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**Table. ECG Findings in Studies of Type A Acute Aortic Dissection**

<table>
<thead>
<tr>
<th>Study</th>
<th>Country</th>
<th>Entry criteria*</th>
<th>No. of patients</th>
<th>ECG findings</th>
<th>Definition of ECG abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapezzi et al (2008)\textsuperscript{12}</td>
<td>Italy</td>
<td>NR</td>
<td>115</td>
<td>ACS-like change: 29%</td>
<td>ACS-like change was defined as the presence of ≥1 of the following findings in ≥2 contiguous leads: ST-segment elevation ≥0.10mV, ST-segment depression ≥0.10mV, and negative T wave ≥0.20mV.</td>
</tr>
<tr>
<td>Chien et al (2013)\textsuperscript{14}</td>
<td>Taiwan</td>
<td>NR</td>
<td>133</td>
<td>Normal: 60% Ischemic ST-T change: 33% Others: 7%</td>
<td>Ischemic change was considered present if ST-segment elevation or depression &gt;0.10mV in at least 2 consecutive leads and others. Others indicated supraventricular or ventricular arrhythmias and atrio-ventricular conduction blocks of various degrees.</td>
</tr>
<tr>
<td>Hirata et al (2010)\textsuperscript{17}</td>
<td>Japan</td>
<td>&lt;12h</td>
<td>159</td>
<td>Normal: 27% Acute change: 50%</td>
<td>Acute changes were defined as a new shift in ST-segment ≥0.10mV or a change in the polarity or the morphology of T wave, compared with previous ECGs. Others indicated atrioventricular block, new atrial fibrillation, premature atrial or ventricular contraction, or sinus bradycardia.</td>
</tr>
<tr>
<td>Biagini et al (2007)\textsuperscript{18}</td>
<td>Italy</td>
<td>NR</td>
<td>164</td>
<td>Normal: 19% ACS-like change: 26%</td>
<td>ACS-like change was defined as the presence of ≥1 of the following findings in ≥2 contiguous leads: ST-segment elevation ≥0.10mV, ST-segment depression ≥0.10mV, and negative T wave ≥0.20mV. ECG findings of ST-segment deviation &lt;0.10mV or negative T wave &lt;0.20mV were classified as nonspecific ST-T abnormalities.</td>
</tr>
<tr>
<td>Kosuge et al (2013)\textsuperscript{19}</td>
<td>Japan</td>
<td>&lt;6h</td>
<td>233</td>
<td>Normal: 30% Ischemic ST-T change: 51% ST-segment elevation: 4% ST-segment depression and T wave abnormalities: 7% Negative T wave: 13% Non-specific ST-T abnormalities: 59% LVH: 29% Right bundle branch block: 7% Left bundle branch block: 2%</td>
<td>ST-segment elevation was considered present if the deviation in leads V2–3 was ≥0.2mV in men aged ≥40 years, ≥0.25mV in men aged &lt;40 years, or ≥0.15mV in women, and the deviation in ≥2 other contiguous leads was ≥0.10mV. ST-segment depression was considered present if the deviation was ≥0.05mV in 2 contiguous leads, and negative T wave was considered present if the depth was ≥0.10mV in 2 contiguous leads.</td>
</tr>
<tr>
<td>Kosuge et al (2015)\textsuperscript{19}</td>
<td>Japan</td>
<td>&lt;12h</td>
<td>409</td>
<td>Normal: 27% Ischemic ST-T change (ST-segment elevation, ST-segment depression, or negative T wave): 52% Bundle branch block/LVH: 19%</td>
<td>Same as in Kosuge et al (2013)\textsuperscript{19}</td>
</tr>
<tr>
<td>Trimarchi et al (2005)\textsuperscript{2}</td>
<td>IRAD (Multicenter)</td>
<td>&lt;14 days</td>
<td>526</td>
<td>Normal: 31% Ischemic ST-T change: 20% Myocardial infarction, new Q waves, or ST deviation: 6% LVH: 22%</td>
<td>NR</td>
</tr>
<tr>
<td>Rampoldi et al (2007)\textsuperscript{4}</td>
<td>IRAD (Multicenter)</td>
<td>&lt;14 days</td>
<td>682</td>
<td>Normal: 30% Ischemic ST-T change: 19% Myocardial infarction, new Q waves, or ST deviation: 6% LVH: 23%</td>
<td>NR</td>
</tr>
</tbody>
</table>

\*Time from onset to recording ECG. ACS, acute coronary syndrome; IRAD, International Registry of Acute Aortic Dissection; LVH, left ventricular hypertrophy; NR, not reported.
**Figure 2.** Figure shows the prevalence of ECG patterns in the study group as a whole (233 patients who were admitted within 6 h from symptom onset and underwent emergency surgery for type A AAD), in patients with no complications, and in patients who had any of the following conditions: severe hypertension as defined by a systolic blood pressure of ≥180 mmHg, pericardial effusion, moderate/severe aortic regurgitation, cardiac tamponade, shock, coronary ostial involvement which was documented at the time of surgery, or in-hospital death. (Reproduced with partial modifications with permission from Kosuge M, et al. 15)

**Figure 3.** Figure shows in-hospital mortality in the study group as a whole (409 patients with type A AAD who were admitted within 12 h from symptom onset), in patients who received urgent surgery, and in patients who did not receive urgent surgery. ST-segment elevation in lead aVR was associated with the highest in-hospital mortality, and this association was maintained after patients were classified according to treatment strategy (received surgery or not). In-hospital mortality in patients not receiving surgery who had ST-segment elevation in lead aVR was extremely high. (Reproduced with permission from Kosuge M, et al. 19)
type A AAD, the prognostic effect of LVH on ECG alone has not been studied.

Ischemic ST-T Changes

Ischemic ST-T changes at presentation are associated with delayed diagnosis and treatment of type A AAD or misdiagnosis of ACS followed by inappropriate treatment such as fibrinolytic therapy, anticoagulation or antiplatelet agents, or cardiac catheterization, potentially postponing emergency surgery and leading to catastrophic perioperative complications such as aortic wall rupture or major bleeding.12,13,16,17,21–23 Hanssen et al10 reported that nearly one-third of 133 patients who underwent surgery for type A AAD were initially misdiagnosed as ACS and received antiplatelet therapy at the time of surgery. Dual antiplatelet therapy increased bleeding and transfusion requirements and was associated with increased 30-day mortality rates. One of the factors leading to this misdiagnosis was ischemic ST-T changes on ECG. The diagnosis of type A AAD relies predominantly on the degree of initial clinical suspicion. It should be emphasized that physicians should be aware that ischemic ST-T changes are common in patients with type A AAD, and type A AAD should be included in the differential diagnosis of ACS.

ST-Segment Elevation

ST-segment elevation has been reported to occur in 4–10% of patients with type A AAD.14–17,24 This ECG finding is thus relatively uncommon in patients with type A AAD, but fatal complications caused by fibrinolytic therapy have occurred in patients who received the misdiagnosis of ST-segment elevation acute myocardial infarction.21–23

In type A AAD, the dissected membrane occasionally extends to the coronary ostium (especially of the right coronary artery), causing acute myocardial ischemia associated with acute ischemic ST-T changes on ECG.11–15 In some cases of type A AAD, functional acute coronary artery malperfusion caused by a flap intimal flap with no direct coronary artery involvement has been reported.25 When the coronary artery is totally occluded by such plausible acute mechanisms, the ECG will show ST-segment elevation in patients with type A AAD.11,14,18

Studies assessing the prognostic effect of ST-segment elevation alone in patients with type A AAD are limited.15,17 Kawahito et al24 reported that ST-segment elevation was a significant and independent predictor of in-hospital death in 122 patients who underwent urgent or emergency surgery for type A AAD. However, only 12 patients (10%) had ST-segment elevation in that study. Biagini et al16 reported that ST-segment elevation was not a significant predictor of in-hospital death in 164 patients with type A AAD. In that study, only 7 patients (4%) had ST-segment elevation. Because the numbers of patients with ST-segment elevation were very small in both studies, it was probably difficult to statistically evaluate the prognostic effect of ST-segment elevation alone; instead, the prognostic effect of “ischemic ST-T changes”, including ST-segment depression and negative T waves, was mainly assessed.

ST-Segment Depression/Negative T Waves

ST-segment depression/negative T waves account for most ischemic ST-T changes in type A AAD (Table).15–17 Previous studies have shown that ischemic ST-T changes are common in patients with type A AAD.1,14,11,19 however, the frequencies of such changes vary widely, ranging from 19–52%, probably because of differences in the definitions of ST-T changes and the timing of ECG recording (Table).

Although the underlying reasons for ST-segment depression/negative T waves in the setting of type A AAD remain to be elucidated, several factors might be involved, including acute ischemic ST-T changes caused by AAD itself as described above, hypertension-induced myocardial ischemia,16,18 catecholamine-induced ST-T changes,21 ST-T changes caused directly by acute pericardial effusion,15,17 and global myocardial ischemia caused by cardiac tamponade, clinically significant aortic regurgitation, and shock.14–17 In addition, various coexisting conditions can amplify the effects of such acute factors, including preexisting ST-T abnormalities or underlying coronary artery disease.21 In fact, one patient with type A AAD often had several of these conditions, suggesting that ECG changes are multifactorial and complex.15

Ischemic ST-T changes (encompassing ST-segment elevation, as well as ST-segment depression and negative T waves) on the presentation ECG, which are characterized predominantly by ST-segment depression/negative T waves, are closely associated with poor outcomes in patients with type A AAD.14–16,19 However, the previous ECG studies of patients with type A AAD have used different criteria to define ST-T changes (Table), precluding a meaningful comparison of the results. In addition, the timing of ECG recording differed in those studies (Table), which might influence the prevalence of ECG abnormalities and their clinical effects. In patients with type A AAD, the mortality rate is highest early after symptom onset.4 Therefore, ECG findings in this very acute phase are thought to have important prognostic implications, but this remains to be fully investigated. To clarify this point, we previously studied the relationships of ischemic ST-T changes on the admission ECG to in-hospital death in 233 patients with type A AAD who were admitted within 6 h of symptom onset and underwent emergency surgery.15 In that study, we used globally accepted definitions of ST-T changes to diagnose acute myocardial ischemia on ECG.46 Ischemic ST-T changes were commonly observed in patients with complicated features and in-hospital death (Figure 2),15 and were significant and independent predictors of in-hospital death.

ST-Segment Elevation in Lead aVR

Previous studies assessing the clinical significance of the admission ECG in patients with type A AAD have focused on ischemic ST-T changes.1,4,12,15,18 however, lead aVR was not considered in those studies. Evidence indicating the importance of lead aVR, especially ST-segment elevation in this lead, has recently accumulated in the setting of ACS.28,39 ST-segment elevation in lead aVR has been shown to be associated with poorer outcomes in patients with ST-segment elevation acute myocardial infarction or non-ST-segment elevation ACS.28,29,31,34,39 However, the prognostic significance of ST-segment elevation in lead aVR has yet to be assessed in the setting of type A AAD.

We previously investigated the prevalence of ST-segment elevation in lead aVR, the clinical features associated with such changes, and the in-hospital prognostic effect in 409 patients with type A AAD who were admitted within 12 h of symptom onset.19 ST-segment elevation in lead aVR was observed in 12% of these subjects and was strongly associated with serious conditions such as shock, cardiac tamponade, or coronary ostial involvement (especially of the left coronary artery), as well as higher in-hospital mortality rate (Figure 3).19 In a multivariate analysis,
ST-segment elevation in lead aVR was the strongest predictor of in-hospital death, followed by no surgical treatment. Our study demonstrated that ST-segment elevation in lead aVR was a significant and strong prognostic marker in patients with type A AAD, whereas isolated ST-T changes in the 11 leads excluding lead aVR were not. To our knowledge, that was the first study to demonstrate the prognostic significance of ST-segment elevation in lead aVR in type A AAD. In clinical practice, lead aVR is often ignored on 12-lead ECG assessment, but our findings emphasize its importance in early risk stratification for type A AAD.

Possible Mechanism of ST-Segment Elevation in Lead aVR

In ST-segment elevation acute myocardial infarction, ST-segment elevation in lead aVR can be caused by transmural ischemia in the basal septum, often resulting from obstruction of the left main or the proximal left anterior descending coronary artery with involvement of the first septal branch. In non-ST-segment elevation ACS, lead aVR looks into the left ventricular cavity from the right shoulder and is therefore referred to as a cavity lead, and ST-segment elevation in this lead might reflect global subendocardial ischemia of the left ventricle, often associated with left main or 3- vessel disease. Thus, in the setting of ACS, ST-segment elevation in lead aVR reflects acute myocardial ischemia caused by severe coronary artery disease as described above, regardless of ST changes in the other 11 leads.

In the setting of type A AAD, the mechanisms underlying ST-segment elevation in lead aVR are unclear. However, acute myocardial ischemia under certain limited conditions is thought to provoke ST-segment elevation in lead aVR. If the left coronary artery ostium is completely obstructed, ST-segment elevation might occur in lead aVR because of transmural ischemia in the basal septum. In patients with coronary ostial involvement (especially of the left coronary artery) associated with critical stenosis but not complete occlusion, global subendocardial ischemia of the left ventricle can also potentially cause ST-segment elevation in lead aVR. Furthermore, even in the absence of coronary artery involvement in the setting of type A AAD, serious conditions such as shock or cardiac tamponade can cause subendocardial ischemia of the left ventricle, and global subendocardial ischemia might lead to ST-segment elevation in lead aVR. Under any of these conditions, patients with type A AAD who have ST-segment elevation in lead aVR are considered to have a very poor prognosis. Although growing evidence indicates the importance of lead aVR in identifying high-risk patients with ACS, our data extend these findings to patients with type A AAD.

Conclusions

ECG findings have critically important diagnostic, therapeutic, and prognostic implications in patients with type A AAD. We hope that our review will contribute to the optimal use of ECG findings in clinical practice.

Disclosures

The authors have no conflicts of interest to declare.

References


