Anomalous origin of the left circumflex coronary artery from the right coronary artery presented as acute inferior myocardial infarction
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Abstract: An anomalously originated left circumflex coronary artery (LCX) from the proximal right coronary artery (RCA) or right sinus of Valsalva is a relatively common however its concurrence with acute myocardial infarction is rare. And all cases with acute MI reported in the literature have been treated with percutaneous coronary intervention. Here, we introduced a very rare case of LCX from the proximal RCA presented with acute inferior MI and treated with intravenous trombolytic therapy.

Keywords: Acute myocardial infarction; Coronary anomaly; The left circumflex artery.

INTRODUCTION
An anomalously originated coronary arteries are detected in about 0.16-1.30% of coronary angiography (CAG) [1-3]. An anomalously originated left circumflex coronary artery (LCX) from the proximal right coronary artery (RCA) or right sinus of Valsalva is a relatively common however its concurrence with acute myocardial infarction is rare. And all cases with acute MI reported in the literature have been treated with percutaneous coronary intervention. Here, we introduced a very rare case of LCX from the proximal RCA presented with acute inferior MI and treated with intravenous trombolytic therapy.

CASE HISTORY
A 42-year-old man without any cardiovascular risk factors except 20 pack-years smoking history admitted to emergency department with acute-onset chest pain of two-hour in duration. Electrocardiography (ECG) on admission revealed ST-segment elevation of more than 2 mm in leads II, III, and aVF (Figure 1).

Since the patient had no contraindication for thrombolytic therapy, he was immediately given 40 mg of tenecteplase intravenously over 5 seconds in accordance with 75-kg subject as indicated. Acetylsalicylic acid, klopidogrel, unfractionated heparin was given according to current guidelines. There was release of chest pain without ST-segment resolution of more than 50% in lead II at 90 minute, thus the patients was referred to our center for rescue percutaneous coronary intervention (PCI). Pre-CAG transthoracic echocardiography examination was normal except mild hypokinetic wall motion at inferior of the left ventricle. Serial cardiac enzyme measurements were in accordance with acute myocardial infarction treated with thrombolytic (Table 1).

Follow-up ECG at 3rd hour revealed complete resolution of ST-segment elevations (Figure 1). Since the patient did not give consent for the CAG, it was cancelled. The patient reported that he used the prescribed medication for only six months after the discharge and then quitted on his will. Two years later, he admitted to our department with troponin-negative chest pain. The patient was electively referred to CAG laboratory. The CAG of the left coronary system revealed normal left anterior descending artery without the LCX while the CAG of the right coronary system, an anomalously originated LCX from the proximal RCA without any obvious lesion on both coronary arteries was demonstrated (Figure 2).

Here, we considered thrombotic occlusion of the anomalous LCX which reperfused following thrombolytic treatment at follow-up. The patient was discharged with acetyl salicylic acid and statin treatment, and under follow-up without any symptom.
Table 1: Follow-up of cardiac enzyme values.

<table>
<thead>
<tr>
<th></th>
<th>CK (IU/L) Normal range: 26-165</th>
<th>CKMB (ng/ml) Normal range: 0.0-4.94</th>
<th>Troponin I (pg/ml) Normal range: 0.0-0.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>55</td>
<td>24</td>
<td>0.1</td>
</tr>
<tr>
<td>6. hour</td>
<td>360</td>
<td>53.8</td>
<td>&gt;25.0</td>
</tr>
<tr>
<td>24. hour</td>
<td>774</td>
<td>124.3</td>
<td>&gt;25.0</td>
</tr>
<tr>
<td>48. hour</td>
<td>231</td>
<td>25.3</td>
<td>13.1</td>
</tr>
<tr>
<td>72. hour</td>
<td>121</td>
<td>6.03</td>
<td>6.7</td>
</tr>
<tr>
<td>96. hour</td>
<td>88</td>
<td>2.3</td>
<td>5.0</td>
</tr>
<tr>
<td>120. hour</td>
<td>39</td>
<td>1.5</td>
<td>2.4</td>
</tr>
</tbody>
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DISCUSSION

Anomalous coronary arteries have been demonstrated in 0.16-1.30% of CAG [1-3]. Whether the presence of LCX anomalies themselves induce atherosclerosis is not clear but 71% of LCX anomalies had a significantly stenosed proximal LCX and 11% had significant atherosclerosis in this artery alone [4, 5]. In our case, the patient applied with acute inferior MI and treated with thrombolytic therapy but without any obvious lesion in the CAG performed two years later. The case was important since the literature has usually reported cases treated with PCI [6]. It is well known that the ostium of anomalous coronary arteries may be difficult and time-consuming to be seated in the diagnostic procedures and also it is essential for prompt management, particularly in subjects undergoing for PCI and coronary surgery [7]. Here, we seated the ostium of the RCA with standard manipulation and right Judkins catheter but the ostium was located more inferoposteriorly. Thrombolytic therapy is gold standard in absence of PCI facility. Its benefit has been proved especially first three hours of acute MI among patients without known contraindications. It can be extremely beneficial in acute MI with high-amount thrombus [8]. Here, tenecteplase with heparin was used. It suggested the underlying mechanism of thrombus-rich abrupt
occlusion of the coronary artery. Since both the RCA and The LCX did not have any obvious lesion, we couldn’t differentiate the involving artery. However, the clinical outcome of the case made the matter meaningless.

CONCLUSION

We introduced the case with the LCX from the proximal RCA presented with acute inferior MI and treated with intravenous trombolytic therapy. Here, we aimed to emphasize the use of thrombolytic in patients with congenital abnormality of the coronary arteries.

REFERENCES