Sinus of Valsalva Thrombosis Causing Renal Infarction

Akio Nakata, Hisashi Yoshizawa, Satoshi Hirota and Eisuke Takazakura

Abstract

A 72-year-old man was admitted to our hospital with a renal infarction. On admission, computed tomography (CT) of the abdomen revealed total occlusion of the right renal artery, which was found to be recanalized with residual thrombus 7 days later. Transesophageal echocardiography and chest CT demonstrated crescent-shaped thrombus in the non-coronary sinus of Valsalva without evidence of aneurysm. After coumadin treatment the patient did not experience recurrent episodes of systemic embolization. Five months after the initiation of anticoagulation, transesophageal echocardiography and chest CT demonstrated disappearance of the thrombus. This is a rare case of renal infarction caused by a thrombus in the non-coronary sinus of Valsalva without aneurysm.

Key words: thromboembolism, transesophageal echocardiography, anticoagulation

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Introduction

Sinus of Valsalva thrombosis is a rare condition, especially in the absence of aneurysm. There have been a few reports of sinus of Valsalva thrombosis leading to myocardial infarction, cerebral infarction, and peripheral emboli (1-5). We describe an unusual case of renal infarction caused by a thrombus in the non-coronary sinus of Valsalva without aneurysm.

Case Presentation

A 72-year-old man presented with acute onset of right flank pain and nausea lasting 5 hours. He had smoked 30 cigarettes per day for 20 years. He was not taking any medications. In the emergency room, his temperature was 36.2°C, blood pressure was 164/94 mmHg, and pulse rate was regular at 96 beats/min. A systolic ejection murmur was heard best over the right second intercostal space (Levine II/VI). Upon palpation, the abdomen was flat and soft without tenderness. No abdominal bruits were heard.

Laboratory tests revealed an elevated white blood cell count (11,000 /µl) but the serum C-reactive protein concentration was within normal limits (0.26 mg/dl). Urinalysis revealed proteinuria without hematuria (Table 1). Chest radiography showed no abnormalities. Electrocardiography showed sinus tachycardia at a rate of 117 beats/min. Computed tomography (CT) of the abdomen with intravenous infusion of contrast medium showed no contrast enhancement in the right kidney. The right renal artery was occluded at the ostium (Fig. 1). Based on these findings, the patient was diagnosed with a right renal infarction. The current patient was in sinus rhythm, and with respect to the total occlusion at the ostium of the renal artery, we suspect that dissection of the renal artery rather than thromboembolism was responsible for the occlusion. Furthermore, at the time of diagnosis, as 7 hours or more had already passed from the onset of his symptoms, we did not perform thrombolytic therapy. Seven days later, follow-up CT of the abdomen revealed recanalization of the right renal artery with a residual contrast defect, which was thought to be a thrombus (Fig. 2). Therefore, the etiology of renal infarction was suspected to be thromboembolism. We next sought a source of the thrombus.

Holter electrocardiogram was unremarkable with no evidence of atrial fibrillation. Transthoracic echocardiography showed thickening of the aortic valve, with a pressure gradient of 21 mmHg. However, sufficient evaluation was impossible because of poor image quality, and transesophageal echocardiography (TEE) was required. The TEE revealed a crescent-shaped echodense structure, suggestive of a thrombus, in the non-coronary cusp of the aortic valve (Fig. 3). No other cardiac source of embolism was identified with...
Table 1.  Laboratory Data on Admission

<table>
<thead>
<tr>
<th>Urinalysis</th>
<th>Blood chemistry</th>
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<tbody>
<tr>
<td>pH</td>
<td>8.5</td>
</tr>
<tr>
<td>Prow</td>
<td>(++)</td>
</tr>
<tr>
<td>Glucose</td>
<td>(-)</td>
</tr>
<tr>
<td>OB</td>
<td>(-)</td>
</tr>
<tr>
<td>Hematology</td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>11000 /μl</td>
</tr>
<tr>
<td>Neut</td>
<td>84.3 %</td>
</tr>
<tr>
<td>Ly</td>
<td>10.4 %</td>
</tr>
<tr>
<td>Moco</td>
<td>4.7 %</td>
</tr>
<tr>
<td>Eo</td>
<td>0.5 %</td>
</tr>
<tr>
<td>RBC</td>
<td>4.70 x10^{12} /μl</td>
</tr>
<tr>
<td>Hb</td>
<td>15.4 g/dl</td>
</tr>
<tr>
<td>Hct</td>
<td>44 %</td>
</tr>
<tr>
<td>PLT</td>
<td>26.8 x10^{11} /μl</td>
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</tbody>
</table>

Pro; protein, OB: occult blood, Neut; neutrophils, Ly; lymphocytes, Moco; monocytes
Eo; eosinophils, Basso; basophils, RBC; red blood cell, Hct; hematocrit, PLT; platelet
TP; total protein, AST; aspartate aminotransferase, ALT; alanine aminotransferase
LDH; lactate dehydrogenase, ALP; alkaline phosphatase
y-GTP; gamma-glutamyltranspeptidase, T.Bil; total bilirubin, Amy; amylase
CRP; C-reactive protein, ESR; erythrocyte sedimentation rate, BUN; blood urea nitrogen
Cl; creatinine, Na; sodium, K; potassium, Cl; chloride, T.chol; total cholesterol
TG; triglycerides

Figure 1.  CT scan of the abdomen with intravenous infusion of contrast material shows no contrast enhancement in the right kidney. The right renal artery was occluded at its ostium (arrows).

Figure 2.  Follow-up CT of the abdomen was obtained 7 days later (reconstructed scan). The right renal artery is re-canalized with a residual contrast defect (arrow).

Figure 3.  Transesophageal echocardiographic image of the aortic valve showing an echodense structure within the non-coronary sinus of Valsalva (arrows). LA, left atrium; RA, right atrium; L, left coronary cusp; R, right coronary cusp.

Discussion

We describe a case of sinus of Valsalva thrombosis causing renal infarction. The major causes of renal infarction are thromboemboli originating from a mural thrombus in the heart, especially in patients with atrial fibrillation or a prior myocardial infarction (6, 7). Other causes include emboli from vegetations in the setting of infective endocarditis, heart tumors, or dissection of the aorta or renal artery (8, 9). It is well documented that atrial fibrillation is the most important risk factor for peripheral thromboembolism (10). Be-
Figure 4. CT scan of the chest with intravenous infusion of contrast medium. A contrast defect is present in the sinus of Valsalva (arrows).

Figure 5. Transesophageal echocardiographic image of the aortic valve obtained 5 months later. The echodense structure within the sinus of Valsalva has disappeared.

cause the renal arteries are end-arteries, acute occlusion always results in infarction.

The sinus of Valsalva is rarely reported as a source of emboli. Aortic aneurysm is a common site of thrombus formation and the origin of systemic emboli. Aneurysms of the sinus of Valsalva are thought to be sites where blood stagnation can occur. Spontaneous echo contrast in a large, unruptured sinus of Valsalva has been reported (11). Spontaneous echo contrast is known to be associated with embolic events. Therefore, sinus of Valsalva aneurysms are thought to be a potential source of embolization. Furthermore, some cases of unruptured aneurysm of the sinus of Valsalva in which thrombus was identified present with embolization (12, 13).

Christiaens et al (3) reported a case of thrombus in the non-coronary sinus of Valsalva without associated atherosclerotic lesions or aneurysms. In the current case, thickening of the aortic valve with a mild pressure gradient was observed. This might cause turbulent flow in the sinus of Valsalva, resulting in endothelial damage of the aortic wall. Furthermore, the present patient was 73 years old and had a long history of cigarette smoking, which increases the probability of atherosclerosis. Nagata et al reported a case of thrombosis of the left sinus of Valsalva in which slight atheromatous changes were observed only in the affected sinus of Valsalva (1). Therefore, we speculate that there may have been a mild lesion in the sinus of Valsalva which played a role in the formation of the thrombus, although the exact mechanism responsible for thrombus formation in the present case is not known because he did not undergo surgery. In the previous reports of sinus of Valsalva thrombosis, coumadin was prescribed for all of the patients except one patient who died the day after the onset (1-3, 5). In the current case, if brain embolism or a systemic embolism recurs, a poor prognosis will be expected. Therefore, it is necessary to continue coumadin.

Sinus of Valsalva thrombosis is a rare condition; however, it should be included in the list of potential sources of embolism, especially in patients without atrial fibrillation. TEE was valuable in the diagnosis of sinus of Valsalva thrombosis in this case.

References


