PDA, which was considered to be caused by suture loosening, and an absence of mass on the aortic valve (Fig. 4). An 8 × 10-mm Cardiofix device (Starway Medical Technology Inc., Beijing, China) was successfully implanted for PDA. The follow-up course was uneventful. Moderate-to-severe hypertension developed, and nephrectomy was performed a year ago.

Discussion

PFs are uncommon, with an incidence of 7%-8% in all primary cardiac tumors. A majority of PFs occur on the left side of the heart and generally involve the heart valves (1, 2, 7). An association of PF with ASD or other CHDs is rare. To date, four cases of PF associated with CHDs have been reported in the literature (Table 1) (3-6).

In this report, we present a potential new syndrome, which may explain some types of PFs associated with CHDs. To our knowledge, there has been no previous report with direct suggestion of the PF as a more prevalent link of CHDs. Further research on PF associated with CHD syndromes is required with a focus on epidemiology, physiological mechanisms, clinical/radiological features, and treatment strategies.

Conclusion

On the basis of the obvious similarities between our cases and those of the other published reports, we propose that a combination of PF and CHDs may represent a recognizable, albeit a rare spectrum of anomalies. We report these cases in the hope that the presence of CHDs will alert the cardiologist to detect a possible PF or vice versa.

References

5. Abad C, De la Rosa P. Right atrial papillary fibroelastoma associated with atrial septal defect. J Heart Valve Dis 2008; 17: 293-6. [CrossRef]

Video 1. Transesophageal echocardiography showed a mass on the aortic valve short axis

Table 1. Cases of papillary fibroelastoma associated with congenital heart diseases

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Age, sex</th>
<th>CHDs and size, mm</th>
<th>PF location and size, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morishita, 2013</td>
<td>76, M</td>
<td>PDA</td>
<td>AoV, 5</td>
</tr>
<tr>
<td>Betigeri, 2011</td>
<td>33, M</td>
<td>AV canal defect (ASD + Cleft mitral)</td>
<td>IVS crest, 15 × 20</td>
</tr>
<tr>
<td>Abad, 2008</td>
<td>60, M</td>
<td>PLSVC, ASD</td>
<td>RA (IAS), 15 × 20</td>
</tr>
<tr>
<td>Watanabe, 1996</td>
<td>64, M</td>
<td>ASD</td>
<td>TV, 11</td>
</tr>
<tr>
<td>Current Study</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient 1</td>
<td>44, F</td>
<td>ASD</td>
<td>AoV, 9</td>
</tr>
<tr>
<td>Patient 2</td>
<td>52, F</td>
<td>ASD, coronary anomaly</td>
<td>AoV, 6</td>
</tr>
<tr>
<td>Patient 3</td>
<td>42, F</td>
<td>PDA</td>
<td>AoV, 11</td>
</tr>
</tbody>
</table>

PDA - patent ductus arteriosus; PF - papillary fibroelastoma; PLSVC - persistent left superior vena cava; RA - right atrium; TV - tricuspid valve.

Bonsai-induced Kounis Syndrome in a young male patient

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Introduction

The use of cannabis and its synthetic derivative, bonsai, has recently increased, and it has become an important health problem (1). Kounis...
syndrome develops by the activation of mast cells, and it is an acute coronary syndrome (ACS) related to allergies, hypersensitivity, anaphylaxis, or anaphylactic reactions (2, 3). Bonsai-induced Kounis syndrome has not been reported in literature. The present study presents the case of a 27-year-old patient who arrived at the emergency clinic with chest pain 6 h after the use of bonsai.

Case Report

A 27-year-old male patient arrived at the emergency clinic with sudden-onset retrosternal pain in the left arm, vomiting, and sweating. The chest pain was characterized by pressure and burning and lasted for 6 h. The patient did not have any known atherosclerosis risk factor and reported bonsai use for the first time in his life. He expressed that he had used a great amount of bonsai 1 h before the onset of chest pain. All vital signs of the patient were stable. His electrocardiographic (ECG) investigation revealed mild bradycardia and ST segment elevations in the inferior derivations (D2, D3, and AVF) (Fig. 1). The patient was referred to the coronary intensive care unit for coronary angiography (CAG). CAG indicated that all coronary arteries were patent (Fig. 2). The fibrinogen and homocysteine levels were increased in our patient, but other analyses could not be performed due to technical limitations.

Discussion

To our knowledge, this patient is the first bonsai-induced Kounis syndrome case in literature. Kounis syndrome, in other words allergic MI, has two types depending on the pathophysiology, or the presence of coronary artery disease. In type I, patients exhibit coronary vaso spasms induced by allergic mediators such as histamine, thromboxane, and leukotrienes without the presence of atherosclerosis risk factors or coronary artery disease. In type II, ACS develops due to coronary vasospasms, plaque erosion, or plaque rupture induced by these mediators in patients with atherosclerotic coronary artery disease. Recently, the fact that there are eosinophil and mast cells in the thrombus material excised from some patients in whom stent thrombosis developed after stent implantation with drug release makes us consider hypersensitivity reactions in these patients. This situation is accepted as the type III variant of Kounis Syndrome (4). With these findings, our case is in accordance with the type I variant of Kounis syndrome.

Cardiovascular and psychological problems are frequently reported to be associated with the use of bonsai. The main pathophysiology of Kounis syndrome is the release of many allergic mediators as a result of mast cell activation induced by allergic stimulants. It has been demonstrated in experimental studies that some endogenous cannabinoid suppress inflammation by decreasing mast cell activation via receptors; however, some endogenous cannabinoids trigger mast cell activation independent from receptors (5). In our patient, coronary arteries were revealed to be completely patent, and this may cause us to consider that a coronary vasospasm was the reason that was caused via mediators released by the bonsai-induced activation of mast cells. The main cardiovascular effects are coronary vasoconstriction, increase in the synthesis of tissue factor, thrombocyte activation, dysrhythmia development induced by various mechanisms, and plaque erosion (6, 7). In a patient considered to have Kounis syndrome, in addition to appropriate ACS management, the determination of serum histamine, specific IgE antibodies, and complement proteins and investigation of eosinophilia aid in the diagnosis (2). Leukocyte, eosinophil, and total IgE levels were increased in our patient, but other analyses could not be performed due to technical limitations.

Conclusion

We hoped to emphasize the consideration of the use of bonsai-type synthetic drugs in a young patient with acute MI signs but without any risk factors.

References

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Bilateral pulmonary vein stenting for pulmonary vein obstruction after surgical correction of total abnormal pulmonary venous connection

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Introduction

Pulmonary vein stenosis (PVS), either acquired or congenital, is a rare condition that can lead to worsening pulmonary hypertension and cardiac failure in children, and it is frequently lethal. The condition is often progressive and is associated with poor survival (1, 2). Pulmonary vein stenting is an option for acute symptomatic relief and significant improvements in diameter, peak PA pressure/systemic pressure ratio, and trans-stenotic gradient (3). Here we report bilateral PVS in an 11-month-old girl after total anomalous pulmonary venous connection (TAPVC) repair who was successfully treated with bilateral stent implantation.

Case Report

An 8-month-old girl weighing 6 kg was referred to our hospital for surgery. Her initial diagnoses were right atrial isomerism, dextrocardia, unbalanced complete atrioventricular septal defect, double outlet right ventricle, severe pulmonary stenosis, and supracardiac non-obstructive TAPVC. She underwent Glenn anastomosis with TAPVC repair when she was 9 months old. Two months after the surgery, she was referred to our clinic because of cyanosis, respiratory distress, hypoxia, and severe upper extremity and palpebral edema. On admission, she was gasping with bradycardia and severe metabolic acidosis. She was immediately admitted to the pediatric cardiac intensive care; endotracheal intubation and inotropic support were started. Her oxygen saturation level was in the low 70s with 100% oxygen supplement. Her echocardiography revealed pulmonary venous obstruction, Glenn dysfunction, and pulmonary hypertension. Anti-pulmonary hypertensive treatment was added to her treatment. An emergent computed tomography angiography (Fig. 1a) showed severe bilateral pulmonary venous stenosis at the junction of the collector sac and pulmonary vein.

Pre-intervention | Gradient* | Post-intervention | Gradient
---|---|---|---
PA | 33/13 (mean 26) | 26/19 (mean 22) |
RPV | 25/23 (mean 24) | 11 | 17/16 (mean 17) | 2 |
LPV | 34/26 (mean 27) | 14 | 19/15 (mean 17) | 2 |
CA | mean 13 | mean 15 |

*Gradients were between the veins and atrium
CA - common atrium; LPV - left pulmonary vein; PA - pulmonary artery; RPV - right pulmonary vein; pressures are in mm Hg.