Case reports


Atrial septal defect and calcification of the tricuspid valve

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A patient with atrial septal defect and calcification of the tricuspid valve is presented. The calcification was first detected by fluoroscopy and its presence was confirmed during cardiac surgery. To our knowledge, this combination has not been reported previously. Possible factors contributing to the calcification are ageing, excessive blood flow, and raised right ventricular pressure.

Calcification of the tricuspid valve is rare. The purpose of this paper is to report a patient with an ostium secundum atrial septal defect and calcification of the tricuspid valve. We have been unable to find a similar case reported in English.

Case report

A 44-year-old man was admitted to Barnes Hospital in August 1968 for evaluation of a congenital heart lesion. A heart murmur had been noted since early childhood, and he was deferred from military service at age 18 years for this reason. There was no history of rheumatic fever. In his third decade, dyspnoea with heavy exertion began. In January 1968, rapid irregular heart action, orthopnoea, and increased dyspnoea when walking were noted. Atrial flutter was converted to sinus rhythm by electrical counter-shock. Digitalis, diuretics, and quinidine were administered with only slight relief of his dyspnoea. On admission to Barnes Hospital in August 1968, he could walk only half a block and was sleeping on two pillows. The patient's 15-year-old daughter is known to have an atrial septal defect.

On physical examination, the apical impulse was felt 2 cm. lateral to the mid-clavicular line in the fifth left intercostal space. There was a moderate left parasternal heave. A widely fixed split of the second heart sound was heard at the second left interspace, with accentuation of the pulmonary component. A grade 2/6 short systolic ejection murmur was present along the left sternal border and at the second left interspace.

Haemoglobin was 14.7 g./100 ml. and serology was negative.

The electrocardiogram showed right axis deviation, complete right bundle-branch block, frequent atrial and ventricular premature contractions, and digitalis effect.

The chest x-ray showed enlargement of the right ventricle and atrium and pulmonary hyperaemia. There was no evidence of left atrial enlargement and no calcification was observed.

Cardiac catheterization results are recorded in the Table. A left-to-right shunt was detected at the atrial level with a pulmonary/systemic flow ratio of 2.5/1, as determined by the Fick method. Pulmonary artery pressure was slightly raised. A surprising finding on fluoroscopy was a heavy ring of calcification in the region of the tricuspid valve (Fig.). Angiography showed a large right atrium, right ventricle, and pulmonary artery. There was no mitral insufficiency during a left ventricular angiogram.

Operation was performed using cardiopulmonary bypass. A 2.5 x 2.5 cm. secundum type atrial septal defect was closed with a running evertting 4-0 Tefdek suture. The everted margins of the defect were then over-sewn with the same suture material. The tricuspid valve contained moderately heavy calcification extending from the entire fibrous ring into the valve leaflets, but not involving the free edge. Calcification was most obvious in the posterior leaflet. The valve leaflets were not distorted and the valve appeared to be competent. The chordae tendineae and papillary muscles were grossly normal.

The post-operative course was complicated by a right lower lobe pneumonia which responded to antibiotics. One week after operation numbness developed suddenly in the right foot, with loss of pulses below the popliteal artery. A clot

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## Table

Cardiac catheterization data

<table>
<thead>
<tr>
<th>Site</th>
<th>Oxygen saturation (%)</th>
<th>Systolic (mm Hg)</th>
<th>Mean (mm Hg)</th>
<th>Diastolic (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>57</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>67</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right atrium (mid)</td>
<td>88</td>
<td>35</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Right ventricle after angiocardiogram</td>
<td>80</td>
<td>49</td>
<td>11</td>
<td></td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>80</td>
<td>35</td>
<td>13</td>
<td>20</td>
</tr>
<tr>
<td>Left atrium</td>
<td>95</td>
<td>92</td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

was removed from the popliteal artery by catheter embolectomy through the right femoral artery. Subsequent convalescence was uneventful. An episode of atrial flutter with 2:1 AV block necessitated readmission three days after discharge. Electrical conversion was performed successfully and he has remained well since.

**FIG. A** Left anterior oblique projection showing heavy calcification (arrow) in the region of the tricuspid valve. Note that the area of calcification is anterior to the mitral valve and inferior to the aortic valve region.

**FIG. B** A magnified view of the tricuspid valve calcification (arrows) in the left anterior oblique projection.

### Discussion

Calcification of the tricuspid valve is unusual in any type of heart disease (Cooley and Schreiber, 1967; J. E. Edwards, 1969, personal communication; J. L. Titus, 1969, personal communication). Though pathological abnormality of the tricuspid valve occurs in as many as 22 per cent of patients with rheumatic heart disease (Cooke and White, 1941), calcification is rarely present (Tillotson and Steinberg, 1962; Epstein, 1940; Hollman, 1957). Starr, Herr, and Wood (1966) found no gross calcification in 31 tricuspid valves removed at operation from patients with rheumatic heart disease.

Tricuspid valve calcification is also rare in congenital heart disease. Hollman (1957) described calcification in the tricuspid valve in a 46-year-old man with severe pulmonary valve stenosis. He suggested that the calcification could have been due to congenital tri-
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cuspid valve disease or possibly to the long-
continued high closing pressure to which the
valve was subjected. Edwards et al. (1965)
have observed fibrosis of the tricuspid valve
and thickening of the chordae tendineae in
pulmonary stenosis with intact ventricular
septum with survival beyond infancy. Focal
calcification was present in the tricuspid valve
though not of a degree to be visualized by
fluoroscopy. Sautter, Emanuel, and Doege
(1965) described irregularly distributed calci-
cum in a stenotic tricuspid valve in a 75-
year-old woman who died from tetralogy
of Fallot. The calcification was attributed to
high pressure in the right ventricle.

Two experienced cardiac pathologists have
not observed calcification of the tricuspid valve
in atrial septal defect (J. E. Edwards, 1969,
personal communication; J. L. Titus, 1969,
personal communication), and we have
been unable to find a published reference to
this association. In his review of atrial septal
defect, Roesler (1934) reported that the AV
valves and chordae tendineae showed thickening
in 48 of 62 cases. Involvement was primar-
ily of the mitral valve though isolated
involvement of the tricuspid valve was also
reported. He did not describe calcification.

The number of reported cases of tricuspid valve
calcification is too small to allow any
surmise regarding the cause of the calculca-
tion. In our case the only possible factors
seem to be ageing and excessive blood flow
across the tricuspid valve with slightly raised
right ventricular pressure. McMillan and
Lev (1964a), in a study of the effect of ageing
on the heart valves, noted thickening of the
aortic and mitral valve leaflets in the fourth
decade and atherosclerotic plaques in both
valves slightly later. These changes progres-
sed with ageing and were occasionally associ-
ated with calcification. Alterations in the
tricuspid and pulmonary valves were much
less obvious and no atherosclerotic plaques
or calcifications were seen. These ageing
changes in heart valves were thought to be
secondary to blood flow and pressure tension
relations.

The arterial embolus occurring in our
patients during the early post-operative period
warrants comment. Embolic complications of
atrial septal defect repairs have been recently
reported by Hawe et al. (1969) to occur with
moderate frequency especially in patients
older than 40 and in those in whom a pros-
thesis is used to close the defect. These
authors recommended that older adult patients
should receive anticoagulant therapy for an
indefinite period after repair of an atrial septal
defect.

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