Dysphagia due to left atrial enlargement after mitral Starr valve replacement

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A patient is described who developed dysphagia after a Starr mitral valve replacement. The onset of dysphagia was associated with shift of the oesophagus from right to left, suggesting that this position of the oesophagus may be important in the development of dysphagia due to enlarged left atrium.

Dysphagia due to left atrial enlargement has become well recognized, but such dysphagia after mitral Starr valve replacement has not previously been described. The patient described here may throw light on the reason why these cases of enlarged left atrium develop dysphagia, a complication that has been shown to be due not merely to the size of the left atrium.

Case report
A woman of 60 developed increasing dyspnoea from the age of 35 until she was 44, at which time she was found to have mitral stenosis, and a closed mitral valvotomy was performed. Initially she was greatly improved, but her dyspnoea gradually returned and by the time of admission her effort intolerance was much increased and was accompanied by orthopnoea, paroxysmal nocturnal dyspnoea, and angina. On examination she had the signs of mitral stenosis and incompetence - a moderately loud pansystolic murmur and an opening snap o-o8 sec after the second sound followed by a long mitral diastolic murmur. The electrocardiogram showed atrial fibrillation with a normal electrical axis and no evidence of ventricular hypertrophy.

On the plain chest x-ray there was moderate

\[\text{FIG. 1} \ (a) \text{ Preoperative penetrated posteroanterior chest x-ray showing moderate (Grade 2) enlargement of the left atrium.} \ (b) \text{ Postoperative x-ray showing that the left atrium has not increased in size.}\]
enlargement of the ventricular mass, pulmonary artery, and right atrium. The penetrated film showed moderate enlargement of the left atrium (Fig. 1a), and a barium swallow showed indentation of the oesophagus by the enlarged left atrium, the oesophagus being displaced to the right of the midline (Fig. 2a and b). At cardiac catheterization the pulmonary artery pressure was 50/35 mmHg with a mean of 40 mmHg and a mean indirect left atrial pressure of 30 mmHg. At operation a median sternotomy was performed with the patient lying on her back; the clinical and x-ray findings were confirmed and the mitral valve was excised and replaced with a Starr prosthesis.

After operation she made a good recovery from the cardiac point of view, but she had difficulty in swallowing solids which appeared to stick at the level of the manubrium, but had no trouble with liquids. She was being given oral slow release potassium, both slow K and Sandoz K 4 times a day. On barium swallow the oesophagus was seen to be obstructed with a flattened, narrowed section where the left atrium was pressing on it. The position of the oesophagus had changed - it was now displaced to the left of the midline (Fig. 3a), in spite of the fact that no surgical manoeuvre had been carried out outside the pericardium. An oesophagoscopy was performed and pieces of solid food were removed, the oesophagus itself showing only slight inflammation of the mucous membrane at the site of the obstruction. The chest x-ray showed no increase in the size of the left atrium (Fig. 1b). Her dysphagia was slightly improved and she was sent out for convalescence. She quickly reverted to being unable to swallow at all, and six weeks later she was readmitted. Barium swallow now showed a definite stricture, which had not been present two months previously, at the upper level of the segment compressed by the left atrium where the oesophagus was crossing the vertebral column (Fig. 3b). On tipping the patient, there was no evidence of gastro-oesophageal reflux.

Attempts to dilate the stricture with mercury bougies were unsuccessful, and she required two subsequent dilatations at oesophagoscopy before she could swallow. The cause of the stricture was not clear, but it had developed since operation and was exactly at the point where the oesophagus passed between the vertebral column and left atrium. Biopsy showed squamous epithelium below the stricture.
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Discussion

Despite the frequency of mitral valve disease with enlarged left atrium, dysphagia is only rarely a complication. It is not simply a matter of size, as in some cases of the largest left atria recorded dysphagia does not occur (Tinney, Schmidt, and Smith, 1943).

There must therefore be other contributory factors. Lee, Freeman, and Olson (1968) have suggested the presence of cardiac failure as one such factor, and in their case report, and several others of dysphagia due to left atrial enlargement, cardiac failure was present. After it had been successfully treated medically, dysphagia disappeared (Dines and Anderson, 1966; Parsonnet, Bernstein, and Martland, 1946; Tinney et al., 1943; Bloomfield, 1940).

Lee and his co-workers considered atrial fibrillation to be a contributory factor also, as all the cases recorded have been in atrial fibrillation.

The position of the oesophagus may be a factor in the development of dysphagia. Usually an enlarged left atrium displaces the oesophagus posteriorly and to the right of the midline (Tinney et al., 1943; Daley and Franks, 1949), but occasionally it is deviated to the left. Daley and Franks (1949) recorded 15 cases of massive dilatation of the left auricle. In only 3 of them was the oesophagus displaced to the left and all 3 developed dysphagia. Of 20 published cases of dysphagia due to left atrial enlargement, the oesophagus was displaced to the left in 6, to the right in 3, and in the other 11 the position was not stated (Table).

<table>
<thead>
<tr>
<th>TABLE 20 recorded cases of dysphagia due to left atrial enlargement showing side to which oesophagus was deviated</th>
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<tbody>
<tr>
<td><strong>No. of cases</strong></td>
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<tr>
<td>------------------</td>
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<tr>
<td>Bedford (1927)</td>
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<tr>
<td>Bishop and Babey (1936)</td>
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<td>Bloomfield (1940)</td>
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<tr>
<td>Daley and Franks (1949)</td>
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<td>Dines and Anderson (1966)</td>
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<td>Lee et al. (1968)</td>
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<td>Le Roux and Williams (1969)</td>
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<tr>
<td>Newton and Levine (1942)</td>
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<td>Nichols and Ostrum (1932)</td>
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<tr>
<td>Parsonnet et al. (1946)</td>
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<tr>
<td>Rösler and Weiss (1925)</td>
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<tr>
<td>Shaw (1924)</td>
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<td>Tinney et al. (1943)</td>
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In the patient described here the oesophagus was initially to the right of the midline, but when dysphagia developed after operation it was found to have become displaced to the left. There was no increase in the size of the left atrium on chest x-ray examination, so it can only be assumed that it was the change in position of the oesophagus which brought on the dysphagia and the development of the stricture. The left is perhaps a less favourable side for the oesophagus as it becomes compressed as it crosses between the aorta and the vertebral bodies, whereas on the right there is more room for it to expand.

This patient developed a benign stricture at the point where the oesophagus passed between the left atrium and vertebral column. The oesophagus was lined with squamous epithelium throughout, which excluded a columnar lined oesophagus with reflux of gastric contents as the cause. Presumably the stricture was secondary to the irritation of slow release potassium and food held up at this point.

References


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