Value of radiographic evidence of calcification of the aortic valve in adults with basal systolic ejection murmurs

I. J. SAREMBOCK, P. J. COMMERFORD, W. BECK

Summary

Severe aortic stenosis is an important, remediable and surgically correctable cause of symptoms and death. Diagnosis in adults and differentiation from minor degrees may be difficult. Aortic valve calcification visible on radiography or fluoroscopy is a sensitive but relatively nonspecific marker for the presence of critical aortic stenosis in patients with angina, dyspnoea or syncope. In 97% of patients with critical aortic stenosis analysed calcification was detected.

Patients and methods

The review comprised all patients over the age of 17 years who underwent cardiac catheterisation at Groote Schuur Hospital during 1981 and 1982. Significant aortic valve calcification was detected. The records of patients who underwent cardiac catheterisation to evaluate the severity of aortic valve stenosis were reviewed. There were 169 patients (97 men, 72 women); ages ranged from 17 to 83 years (mean 54 years); 112 were white (66.7%), 46 were coloured (27.3%), and 11 were black (6.8%).

Clinical evaluation

The results of the clinical evaluation at the time the patients were assessed and referred for cardiac catheterisation were presented. These included the symptoms and their duration, the physical signs, and the presence of left ventricular hypertrophy on the ECG. Special attention was paid to whether or not the clinician had recorded the presence of calcification on the lateral chest radiograph or fluoroscopy. Cardiac catheterisation and fluoroscopy were routinely performed in all patients. The importance of the presence of critical aortic stenosis in the assessment of patients with aortic stenosis in our clinic, this information was always recorded, and fluoroscopy was routinely performed in such patients if no calcium was seen on the lateral chest radiograph.

Catheterisation

Left and right heart cardiac catheterisation was performed in all patients. Critically damped pressure recordings were obtained through fluid-filled catheters using a Statham pressure transducer connected via short, non-elastic plastic tubing to an Electronics for Medicine VR12 recorder system and recorded at a paper speed of 100 mm/s. Left heart catheterisation was performed retrogradely via the femoral or brachial artery approach. Because special attention has been given to the presence of calcification in the assessment of patients with aortic stenosis in our clinic, this information was always recorded, and fluoroscopy was routinely performed in such patients if no calcium was seen on the lateral chest radiograph.

Cardiac Clinic, Department of Medicine, University of Cape Town and Groote Schuur Hospital, Cape Town

I. J. SAREMBOCK, M.B., CHB., F.C.P., F.A.C.C.
P. J. COMMERFORD, M.B., CHB., F.C.P. (S.A.), F.A.C.C.
W. BECK, M.SC., M.B., CH.B., F.R.C.P., F.A.C.C.

Reprint requests to: Professor P. J. Commerford, BHI Cardiac Clinic, Groote Schuur Hospital, Observatory, 7925 RSA.
aortic stenosis (non-CAS) was present in 95 patients. The two groups did not differ significantly with regard to age, symptoms, symptom duration, physical findings or the presence of significant coronary artery disease (Table I).

The ECG pattern of left ventricular hypertrophy was found in 88% of patients with CAS and 66% of those with non-CAS ($P < 0.01$).

Calcification of the aortic valve was visible on fluoroscopy in 97% of patients with CAS and 77% of patients with non-CAS ($P < 0.001$) (Figs 1 and 2). When analysed more carefully to include the density of calcification, this occurred in 66% of cases on lateral chest radiography and 69% on fluoroscopy in those patients with CAS. Dense calcification determined by either method occurred in only 32% of patients with non-CAS ($P < 0.001$). Only 2 elderly black patients (over 50 years) with CAS did not have detectable calcification of the aortic valve (Table II).

### Table I. Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>CAS ($N = 74$)</th>
<th>Non-CAS ($N = 95$)</th>
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</thead>
<tbody>
<tr>
<td><strong>Age (yrs)</strong></td>
<td></td>
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<tr>
<td>No. of patients</td>
<td>61 ± 12</td>
<td>65 ± 17</td>
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<tr>
<td>($&gt;50$ yrs 81%)</td>
<td>($&gt;50$ yrs 67%)</td>
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<tr>
<td><strong>Shortness of breath</strong></td>
<td></td>
<td></td>
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<tr>
<td>No. of patients</td>
<td>65 (88%)</td>
<td>64 (67%)</td>
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<tr>
<td><strong>Angina</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>46 (62%)</td>
<td>63 (66%)</td>
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<tr>
<td><strong>Syncope</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>28 (38%)</td>
<td>29 (31%)</td>
</tr>
<tr>
<td><strong>Symptom duration</strong> (mo.)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>21 ± 13</td>
<td>19 ± 14</td>
</tr>
<tr>
<td><strong>Anacrotic pulse</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>55 (74%)</td>
<td>60 (63%)</td>
</tr>
<tr>
<td><strong>Long systolic murmur</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>65 (88%)</td>
<td>76 (80%)</td>
</tr>
</tbody>
</table>

CAS = critical aortic stenosis.

### Table II. ECG, Radiographic and Coronary Angiographic Characteristics

<table>
<thead>
<tr>
<th></th>
<th>CAS ($N = 74$)</th>
<th>Non-CAS ($N = 95$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LVH ± R/C</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>65 (88%)</td>
<td>63 (66%)</td>
</tr>
<tr>
<td><strong>Valve calcification</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>72 (97%)</td>
<td>73 (77%)</td>
</tr>
<tr>
<td><strong>Dense calcification</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>49 (66%)</td>
<td>30 (32%)</td>
</tr>
<tr>
<td><strong>Chest radiograph</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>51 (69%)</td>
<td>30 (32%)</td>
</tr>
<tr>
<td><strong>Fluoroscopy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of patients</td>
<td>49 (66%)</td>
<td>59 (62%)</td>
</tr>
</tbody>
</table>

CAS = critical aortic stenosis; LVH ± R/C = left ventricular hypertrophy ± repolarisation change.

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Fig. 1. Lateral chest radiograph showing dense calcification of the aortic valve.

Fig. 2. Line diagram showing important landmarks for the detection of aortic valve calcification (A = aortic valve calcification; B = bifurcation of the trachea; C = anterior costophrenic angle).
Discussion

The evaluation of adult patients with basal systolic murmurs, which may be due to aortic stenosis, is difficult. In the symptomatic patient with such a murmur who has dyspnoea, angina or syncope, it is important to determine whether the symptoms are due to severe aortic stenosis, which is surgically remediable, or are secondary to coronary artery disease or hypertensive heart disease where the murmur may be produced by a minor, surgically unimportant degree of aortic stenosis. Authorities agree that this assessment is difficult.1,4

The assessment of the character of the pulse, usually considered to be a helpful diagnostic feature, is difficult and even experienced clinicians are often incorrect. This is particularly so in the elderly where the presence of a rigid vascular tree may alter the pulse waveform.8,13 The qualities of the murmurs are notoriously unreliable and clinical assessment is subjective. In general, the more severe the stenosis, the longer the duration of the murmur and the later in systole its peak intensity.13 It is also well known that as left ventricular dysfunction develops and cardiac output falls, the murmur may shorten, soften and even become inaudible.

Ancillary investigations, such as the ECG, may be unhelpful with up to 15% of patients with severe aortic stenosis failing to show the expected pattern of left ventricular hypertrophy and strain.14 This is confirmed in our patients where 12% with CAS did not have a pattern of left ventricular hypertrophy and strain.

Conventional 2-D and M-mode echocardiography can do no more than confirm thickness and possibly calcification of the aortic leaflets and its distribution.6,15 Doppler echocardiography may prove to be more helpful and to date experience in one series using this technique provides evidence of accurate estimation of the gradient across the aortic valve in adults.16

The fact that, firstly, surgical relief of severe aortic stenosis both relieves symptoms and prolongs life17,18 and, secondly, the diagnosis may be difficult to make clinically and confirm non-invasively, leads to cardiac catheterisation being performed to exclude it. Cardiac catheterisation, like all invasive procedures, carries small but distinct risks.20 These risks are increased in advanced age, the presence of diffuse vascular disease or severe coronary artery disease,21 all of which are important factors in the population who may have aortic stenosis. Furthermore, to determine the severity of aortic stenosis, it may be necessary to perform trans-septal catheterisation which carries well-defined risks.22-24

Our study confirms the value of a simple, cheap, non-invasive investigation. Examination of the lateral chest radiograph19-22 or the performance of fluoroscopy2,6 to detect calcification is of value in assessment of the patient with appropriate symptoms and a basal systolic murmur. The presence of heavy calcification of the aortic valve, visible on chest radiography or fluoroscopy, is an important pointer to severe aortic stenosis as the cause of symptoms and should be looked for routinely in such patients. The absence of such calcification does not absolutely exclude aortic stenosis as the cause of the symptoms but does render it unlikely. In addition, heavy calcification also occurred in approximately one-third of patients with non-CAS. Attention to this simple non-invasive procedure may improve diagnostic evaluation of patients with basal systolic murmurs.

The detection of aortic valve calcification on lateral chest radiography is difficult and we believe it is under-reported. Attention to anatomic landmarks (Fig. 2) will aid in identification. When indicated, specific instructions to the radiologist may be of assistance (Fig. 1). Fluoroscopy is helpful in the differentiation of aortic valve calcification from mitral annular and coronary artery calcification.

It is worth noting that the demonstration of non-CAS does not mean that the condition will not progress in the future. In addition, significantly symptomatic patients in this group may require surgery. The progression of aortic stenosis is unpredictable and patients shown to have non-CAS should be carefully followed-up at regular intervals and may require repeat cardiac catheterisation.

It is important to note that a minority (7%) of the patients in this analysis were black. This is in keeping with a clinical impression that isolated aortic stenosis is uncommon in adult black patients at our hospital. Equally important is the fact that the 2 patients with CAS who did not show significant calcification on radiography or fluoroscopy were black. This may reflect different pathogenetic processes or different propensities to dysplastic calcification in different racial groups and emphasises the importance of considering this factor when dealing with individual patients.

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REFERENCES

The clinical efficacy of a second-generation α-glucosidase inhibitor in non-insulin-dependent diabetic patients

M. A. K. OMAR, M. A. SEEDAT, I. HILLEBRAND

Summary

In a placebo-controlled double-blind randomised group comparison, the tolerability and metabolic effect of emiglitate, a new second-generation α-glucosidase inhibitor, was evaluated in 10 Indians with non-insulin-dependent diabetes mellitus being treated with sulphonylureas. Patients on emiglitate showed a decrease in postprandial plasma glucose levels (means 10 ± 0.94; 10.1 ± 0.97 mmol/l) compared with the levels in the run-in period (mean 11.4 ± 1 mmol/l) but the difference was not significant. However, the emiglitate group showed a significantly greater decrease while on the drug compared with the placebo group at the end of the 1st week (P < 0.01). The Hb A1 levels in those on emiglitate decreased significantly a week after cessation of therapy (mean 7 ± 0.81% compared with the run-in levels (mean 9.4 ± 1.79%; P < 0.02). The drug was well tolerated and caused no haematological or biochemical abnormalities.


The use of enzyme inhibitors to influence carbohydrate digestion has now become established as a therapeutic principle in the treatment of diabetes mellitus. Recently, emiglitate, a new α-glucosidase inhibitor with the chemical structure of a 1-deoxynojirimycin, has been found to reduce the postprandial rise in blood glucose and serum insulin levels in both rats and healthy volunteers (I. Hillebrand — unpublished data).

Diabetes Clinic, Department of Medicine, University of Natal, Durban
M. A. K. OMAR, M.D., F.C.P. (S.A.), M.R.C.P.
M. A. SEEDAT, M.R.C.P.
Bayer (Pty) Ltd, Wuppertal, Federal Republic of Germany
I. HILLEBRAND, M.D.

The metabolic effect and tolerability of emiglitate was investigated in a group of Indian non-insulin-dependent diabetes mellitus (NIDDM) patients being treated with sulphonylureas.

Patients and methods

Ten patients (5 males and 5 females) with NIDDM diagnosed on the basis of the revised World Health Organisation diagnostic criteria were selected for the study. Their mean age was 49 years (range 32-58 years) and the mean duration of NIDDM was 7.5 years (range 2-15 years). Each patient was on dietary therapy plus a sulphonylurea preparation (tolbutamide, glibenclamide or glimepiride).

The study was designed as a double-blind placebo-controlled, randomised group comparison. After a run-in period of 14 days, half the patients were put on placebo and half on emiglitate (taken once daily with the first bite of breakfast). Both placebo and emiglitate were given to patients as scored 20 mg tablets identical in size, shape and colour. The patients continued taking their usual sulphonylureas and were on similar diets during the study period. After 2 weeks on placebo or emiglitate all patients received a further 2-week course of placebo.

Plasma glucose levels (measured 2 hours after breakfast) and Hb A1 levels were recorded at the end of the run-in period and thereafter at weekly intervals, when enquiries were made concerning each patient’s well-being, and his/her weight, plasma glucose levels in patients receiving emiglitate were not significant over the run-in period or weeks 1 and 2. During the placebo period, from day 14 to day 28, the glucose levels rose, but the differences fell short of statistical significance. In the placebo group the plasma glucose levels throughout the study period showed no significant changes compared with the run-in levels.