ON THE GENESIS OF THE INfarCTION OF THE POSTERIOR PAPILLARY MUSCLE OF THE LEFT HEART VENTRICLE

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Observations on the development, morphology and clinical importance of papillary infarction are extremely insufficient and scanty as yet. Following the purposeful studies carried out by Arhangelskii (3) in this respect, Smoliannikov and Naaldachina (6) also deal with this problem. No investigations on the latter have been reported in Bulgaria.

The genesis of heart infarction continues to be a question of controversial opinions despite the extraordinary great number of studies carried out, especially in recent years (2, 4, 5, 7, 8). Of interest in this respect is the study of the papillary muscle infarct, particularly the isolated infarction of the posterior papillary muscle on account of its higher incidence (3) and characteristic features of its blood supply (1, 6).

In the course of necropsy studies, the posterior papillary muscle was cut longitudinally and in instances of infarction, histologically stained with hemalaun-eosin and pikrofluxin after the van Gieson technique.

Investigation is carried out on 587 necropsy preparations over a period of 14 months. Bearing in mind that heart infarction occurs mostly after the 30-year age limit, and predominantly against the background of atherosclerosis and hypertony, our material was studied in this respect as well. Autopsy is performed on 310 cadavers of individuals who died after the 30-year age. The basic affection in 139 of them is disseminated atherosclerosis and in 35 — hypertony. Chiefly the left heart ventricle has been studied.

Of a total of 32 myocardial infarctions, 9 appear to involve the posterior papillary muscle in individuals above 55 years; in four the infarct is isolated and in five — combined with infarction in other localities of the wall of the left heart ventricle (Table 1).

The isolated anatomicalwise infarction of the posterior papillary muscle is with irregular outline, often with a hemorrhagic zone, predominantly situated in the chordal half of the muscle, whereas in combined cases the volume and position of the infarct of papillary muscle is most variable.

Histologically (Fig. 1) fresh necrosis is noted dependent on the duration of the infarct, as well as hemorrhages, leukocytic infiltration, macrophagial reaction and fibrosis formation. The blood vessels are on the whole hyperemized and in isolated instances there is pronounced perivascular edema. In the surroundings of some arterioles strongly pronounced fibrosis is marked — a finding that could be assumed as consequent to coronary sclerosis. Macroscopically as well as histologically, thrombosed vessels are detected only in one case (No 6) with coronary thrombosis (diabetes melititus and generalized atherosclerosis).
Table of the cases with infarction of the posterior left papillary muscle

<table>
<thead>
<tr>
<th>No</th>
<th>Necropsy preparation</th>
<th>Name</th>
<th>Sex</th>
<th>Age</th>
<th>Concise diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>193/1964</td>
<td>S. I. L.</td>
<td>m</td>
<td>55</td>
<td>Rheumatic heart disease. Recent infarction of the posterior papillary muscle</td>
</tr>
<tr>
<td>2</td>
<td>224/1964</td>
<td>M. T.</td>
<td>m</td>
<td>64</td>
<td>Generalized atheromatosis and inveterate cerebral softening. Coronary sclerosis. Recent infarction of the posterior papillary muscle</td>
</tr>
<tr>
<td>4</td>
<td>381/1964</td>
<td>A. H. H.</td>
<td>m</td>
<td>67</td>
<td>Generalized atherosclerosis. Coronary sclerosis. Combination of recent infarction of the septum and of the posterior papillary muscle</td>
</tr>
<tr>
<td>5</td>
<td>382/1964</td>
<td>S. S. A.</td>
<td>m</td>
<td>59</td>
<td>Hypertony. Coronary sclerosis. Combined extensive infarction of the wall and involvement of the posterior papillary muscle</td>
</tr>
<tr>
<td>7</td>
<td>14/1965</td>
<td>M. P. K.</td>
<td>f</td>
<td>62</td>
<td>Pulmonary TBC. Hypertony. Coronary sclerosis. Isolated recent infarction of the posterior papillary muscle</td>
</tr>
<tr>
<td>9</td>
<td>53/1965</td>
<td>S. I. V.</td>
<td>m</td>
<td>64</td>
<td>Generalized atherosclerosis. Coronary sclerosis. Combined infarction inveterated of the wall and recent of the posterior papillary muscle</td>
</tr>
</tbody>
</table>

From clinical viewpoint, clearly outlined and characteristic data are not invariably disclosed in infarctions of the posterior left papillary muscle, combined with infarctions of other areas of the wall of the left ventricle. Two of the isolated infarcts (cases 2 and 7) exhibit a course devoid of clinical manifestations. For the first case followed-up the respective changes were established electrocardiographically.

Insofar genesis is concerned of the posterior papillary muscle infarction, the following considerations could be stressed: our first case with combined rheumatic heart disease, involving the aortic opening (stenosis and insufficiency), with no morphological changes at all in the coronary vessels affords us sufficient ground to assume that a decisive role for the development of the infarction is played by the hemodynamical factor. In case 6 coronary thrombosis is established substantiating the mass infarction with involvement of the posterior papillary muscle as well. In all remaining cases, irregular, unevenly manifested coronary sclerosis is present without severe stenosis and without thrombosis. The circumstance that no coronary thrombosis is detected justifies the assumption, that for the development of infarction of the posterior left papillary muscle neurogenic and vasomotor factors are of utmost importance. Our second case is very indicative in
Fig. 1. The area adjacent to the infarction zone with filled up blood vessels and lymphocytic infiltration (case 1, prot. 193/1931, male 55, hematoxylin-eosin. ×80)

Fig. 2. Infarction zone with hemorrhage (case 3, prot. 225/1961, male 58, hematoxylin-eosin. ×80)
Fig. 3. Thrombosed vessel of the posterior papillary muscle, hemorrhage and necrosis (case 6, prot. 149/1964, female, 62, hematoxylin-eosin, x80)

Fig. 4. Infarction of the posterior papillary muscle with hypertrophied blood vessels (case 8, prot. 23/1965, hematoxylin-eosin, x80)
this respect. It concerns an active political worker who travelled by sea. On the day of passing, he had to attend two receptions and to direct a press-conference. For the remaining cases the vasomotor moments are essential.

The cases herein reported fully support the conception that for the development of myocardial infarction, vasomotor disorders (4, 5, 8) occurring mostly on the basis of already existing, in some cases severe coronary sclerosis, play an essential role. A more detailed clinical-anatomical analysis comprising a greater number of cases reviewed will certainly elucidate the problem of genesis, course, clinical manifestations and outcome of the combined infarction and more particularly of the isolated papillary infarct, whose clinical picture and morphology are far from being sufficiently investigated.

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