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Chronic Degenerative Changes in the Myocardium Supplied by Bridged Coronary Arteries in Eight Postmortem Samples

S. Tuna Karahan, MD[1]; H. Selçuk Sürücü, MD[2]; Erdal Karaöz PhD[3]

In humans, the coronary arteries course not only subepicardially but also intramyocardially. The intramyocardial course of the coronary artery is reported to lead to acute ischemic heart disease and, as well, it may be symptomless. The aim of this study was to investigate the long-term ischemic effects of bridged arteries on the myocardium, and was carried out on 8 autopsy hearts with myocardial bridges and 2 hearts without myocardial bridges. The samples from the myocardium were examined with light microscopy. In the myocardium supplied by the bridged arteries, it was observed that there was an increase in the intercellular connective tissue, which was rich in collagen bundles, lymphocytes, fibroblasts and macrophages. Compression of the coronary artery by myocardial bridges may cause chronic degenerative changes, which may remain silent for a long time. (*Jpn Circ J* 1998; **62:** 691–694)

Key Words: Ischemia; Myocardial bridge; Myocardium

The coronary arteries that course subepicardially have also been found intramyocardially in autopsy and angiography studies of men[1–4] This condition is much different from the coronary arteries' intramyocardial course before their termination in that the coronary arteries penetrate into the myocardium and later in their course become superficial again and continue subepicardially. Therefore the coronary artery passes under a part of the myocardium called a myocardial bridge.

Individuals with a myocardial bridge show different clinical presentations according to the relationship of the myocardial bridge and the coronary artery under it. There are reports of a wider range of clinical manifestations, ranging from no symptoms to sudden death[5–7] Some researchers claim that the blood supply to myocardium occurs mainly on diastole, so the myocardial bridge will not cause any symptoms[5,8] whereas others accept the diastolic blood supply phenomenon but believe that myocardial ischemia may occur due to systolic narrowing greater than 75% and heart rates over 150 beats/min[9]

The term ischemia, used in studies on myocardial bridges, defines acute ischemic diseases of the heart (angina pectoris, myocardial infarction and sudden cardiac death). In individuals with myocardial bridge, we thought that it is possible for the coronary artery to be compressed for long periods and chronic ischemia to develop in the myocardium supplied by it. In the present study, myocardial samples from autopsies of individuals with a myocardial bridge but without a cardiac cause of death, were evaluated with light microscopy and the presence of ischemic findings was investigated.

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[1]Department of Anatomy, Faculty of Medicine, Ankara University, Ankara, [2]Department of Anatomy, Faculty of Medicine, Hacettepe University, Ankara and [3]Department of Histology, Faculty of Medicine, Süleyman Demirel University, Isparta, Turkey
Mailing address: S. Tuna Karahan, MD, Ankara Üniversitesi Typ Fakültesi, Anatomi Anabilim Daly, Morfoloji Binasy, Syhhiye, Ankara TR-06100, Turkey. E-mail: tkarahan@turk.net

Methods

The materials used were 8 autopsy hearts with myocardial bridges and 2 hearts without myocardial bridges. The individuals, who had been involved in traffic accidents, had died before being brought to the hospital and there were no clinical data indicating any previous cardiac disease. The cause of death for all 10 cases was found to be non-cardiac on autopsy. In addition their histories, as given by their families, did not reveal any possible heart disease. The ages of the individuals were between 32 and 51 years. The myocardial bridges were at different levels of the left anterior descending artery (LAD). The length of the artery coursing under the myocardial bridge ranged from 30 to 42 mm. At the deepest point, the thickness of the myocardial tissue above the bridged coronary artery was between 1.1 and 2.3 mm (Table 1). Myocardial samples were taken from the anterior ventricular wall, which was supplied by the bridged or non-bridged LAD. Full thickness (1×1 cm) myocardial samples were taken around the point where the coronary artery terminated by entering the myocardium. In hearts with myocardial bridges, samples were also taken from the anterior wall of the left ventricle supplied by a section of the LAD proximal to the myocardial bridge. The myocardial samples were blocked in paraffin and sections were prepared transverse and longitudinal to the myocardial fibres. The sections were stained with hematoxylin-eosin and Mallory–Azan techniques, examined and photographed with a Nikon light microscope.

Results

Due to the inadequate fixation of the autopsy material, the histologic sections were not devoid of artifacts, such as shrinking of the cells and dilated intercellular spaces. But

Table 1 The Data of the Cases With Bridged and Nonbridged Coronary Arteries

Case no.	*Sex*	*Age (years)*	*Heart weight (g)*	*Length of the bridged segment of the coronary artery (mm)*	*Thickest point of the myocardial bridge (mm)*
1. bridged	*M*	*39*	*320*	*30*	*2.3*
2. bridged	*M*	*32*	*291*	*33*	*1.2*
3. bridged	*M*	*36*	*308*	*31*	*1.8*
4. bridged	*M*	*34*	*296*	*30*	*1.1*
5. bridged	*M*	*33*	*332*	*34*	*1.5*
6. bridged	*F*	*34*	*285*	*42*	*1.1*
7. bridged	*M*	*51*	*296*	*37*	*1.6*
8. bridged	*F*	*41*	*248*	*35*	*1.7*
9. control	*F*	*44*	*217*	–	–
10. control	*M*	*33*	*319*	–	–

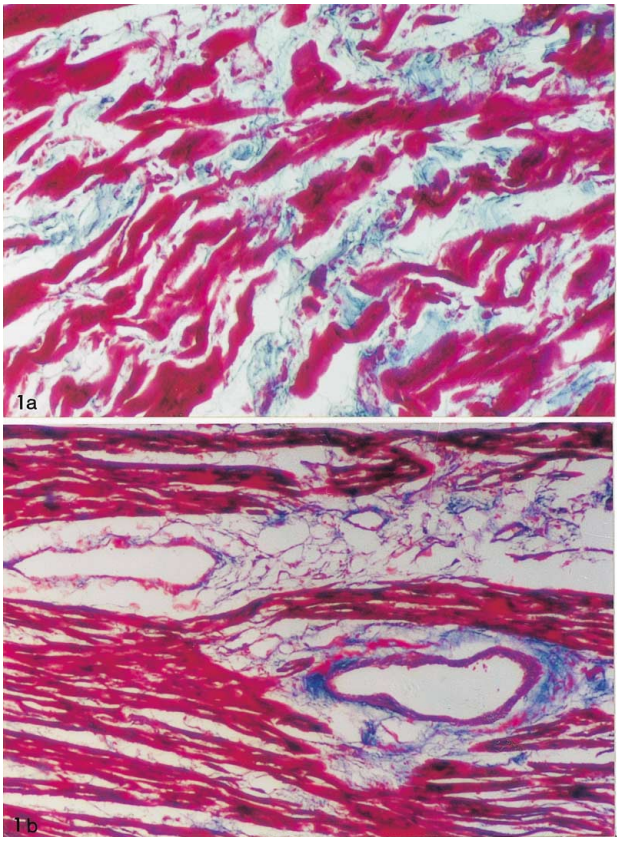

Fig 1. The myocardium supplied by bridged artery. (a) Increased intercellular connective tissue is seen between the myocardial cells (Mallory–Azan, ×399), (b) The connective tissue was especially increased around the blood vessels (Mallory–Azan, ×159).

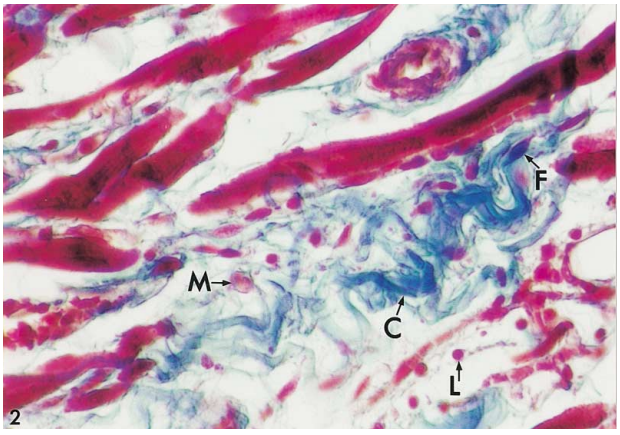

Fig 2. Collagen bundles (C), lymphocytes (L), fibroblast (F) and macrophage (M) in the connective tissue of the myocardium supplied by bridged artery. In addition, the striation of myocardial fibres was not seen (Mallory–Azan, ×823).

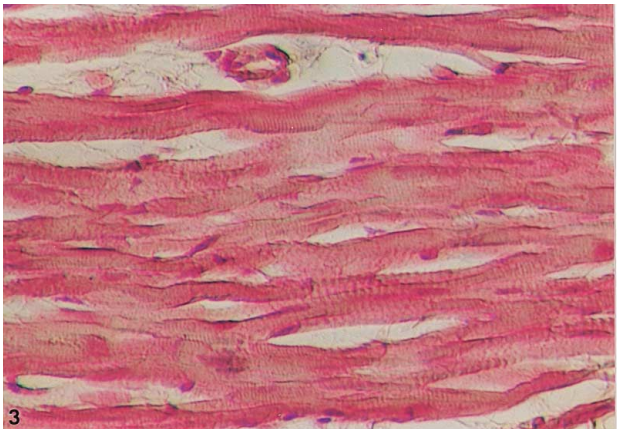

Fig 3. The striation of the myocardial fibres can be observed in the myocardium supplied by non-bridged artery, while the heart has a myocardial bridge on the left anterior descending artery (HE, ×823).

when we compared the myocardium supplied by the bridged arteries with the control groups, we found a profound increase in the intercellular connective tissue (Fig 1a), which was very rich in collagen bundles, lymphocytes, fibroblasts and macrophages (Fig 2), obvious with Mallory–Azan stain. The connective tissue was very prominently increased around the blood vessels, but was increased throughout the whole thickness of the observed areas of myocardium supplied by the bridged artery (Fig 1a,b). No correlation between the pathologic findings and the length and diameter of the bridged arteries was demonstrated as they were close in all the samples (Table 1). In different individuals, including the ones with different lengths of bridged coronary artery, the degree of the connective tissue increase seemed to be similar.

Striation of the myocardial fibres was clearly observed in the normal cardiac samples, but was absent in the samples taken from the myocardium supplied by the bridged arteries. In hearts with myocardial bridges, samples from the myocardium supplied by non-bridged arteries were structurally normal (Fig 3). In all groups the coronary arteries were also examined and no pathological changes were found.

Discussion

There are different opinions about the conditions in which a myocardial bridge would become symptomatic or not. From an optimistic point of view, a myocardial bridge is a normal variant and does not cause any symptoms.[5] Shulte et al demonstrated no ischemic findings in the cadaver of a 71-year-old individual with a left anterior descending artery 16 mm deep in the myocardium along 46% of its length and who had had a heart rate of 136 beats/min.[8] Reig et al reported that a myocardial bridge will not cause any change in the coronary blood flow, because its fibers are structurally different from the normal myocardium.[10] Voelker et al suggested that myocardial bridges are benign and that additional factors, like fast atrial pacing and exercise, will not necessarily cause ischemic heart disease, but they added that larger numbers of patients should be studied to clarify the subject.[11]

Some researchers accept that in strenuous exercise myocardial bridges can cause angina pectoris, myocardial infarction and even sudden death, the reason for this being the decrease in the duration of diastole and diastolic filling of the coronary arteries during exercise.[9,12,13] If the narrowing due to a myocardial bridge is 50% or less, it will not cause any symptoms; if it is 50–75% it will cause ischemic findings but no increase in lactate and if it is 75% or more it will cause ECG changes and an increase in lactate.[13] Different hemodynamic conditions of the coronary arteries, such as sodium nitroprusside, fast atrial stimulation and noradrenaline, are accepted as a possible cause of ischemia in hearts with coronary arteries compressed more than 75% by the myocardial bridge.[14]

Other researchers suggest that coronary vasospasm should be added to any symptoms caused by myocardial bridges, as symptoms can also occur at rest.[15,16] Drugs like nitroglycerine, amiodarone and propanolol, and surgical treatment are suggested for prevention of such a clinical condition.[11,15–17]

The presence of symptoms with a myocardial bridges is also related to the relationship of the muscle fibres forming the myocardial bridge and the coronary artery under it. If the coronary artery is coursing deeply and the bridge fibres are covering it in a helical fashion ischemic symptoms are possible.[18] Myocardial bridges are reported to be more common in hearts with ventricular hypertrophy[19] because of the increased systolic compression.[20] There are other reports that there is no correlation between the heart weight and the occurrence of the myocardial bridge.[21] In the present study the heart weights were in the normal range and our results do not contribute to the relation between the heart weight and the occurrence of myocardial bridges.

The studies on the possible hazards of myocardial bridges are either clinical case reports or radiological investigations[13,15] and the symptoms described are due to acute ischemic heart disease.[3,9] But non-symptomatic myocardial bridges may lead to chronic ischemic heart disease and become symptomatic due to the degree and duration of the compression of the coronary artery. Congestive heart failure may be the first manifestation of chronic ischemic heart disease.[22]

Non-symptomatic chronic ischemic heart disease can be detected by pathologic examination of the myocardium,[22] but no study of the myocardial bridge has defined the microscopic changes of the myocardium supplied by the bridged coronary artery.

In our study the myocardium supplied by a bridged coronary artery was found to have an increased amount of intercellular connective tissue elements compared with the normal myocardium. No scars of previous acute myocardial infarction were observed in our samples, so the increased connective tissue appears to be due to chronic ischemic heart disease caused by the chronic compression of the coronary artery by the myocardial bridge rather than due to an acute myocardial infarction. In different individuals, the connective tissue increase seems to be similar in the myocardia supplied by a bridged artery. Further ultrastructural and morphometric studies in wider series are necessary to quantify the connective tissue increase in different individuals.

When hearts were examined 6 weeks to 13 years after a myocardial infarction, interstitial connective tissue elements like collagen was found to be increased in the interventricular septum independent from the infarct site.[23] Therefore it was necessary to compare the myocardium supplied by the bridged arteries and the myocardium supplied by non-bridged arteries in the same heart. The amount of connective tissue was not increased in myocardium supplied by the non-bridged arteries and these samples were microscopically not different from normal myocardium.

Myocardial bridges may compress the coronary arteries and cause chronic degenerative changes characterized by an increase in the myocardial connective tissue. According to our results, the absence of symptoms in individuals with myocardial bridges does not indicate the absence of microscopic changes of the myocardium.

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