UC Irvine
UC Irvine Previously Published Works

Title
Are Muscular Bridges Capable of Causing Myocardial Infarction?

Permalink
https://escholarship.org/uc/item/49d8h2qb

Journal
Texas Heart Institute Journal, 33(4)

Author
Kern, Morton J.

Publication Date
2006

Peer reviewed

eScholarship.org Powered by the California Digital Library
University of California

CORRESPONDENCE

Are Muscular Bridges Capable of Causing Myocardial Infarction?

To the Editor:

I read with interest the case report titled "Daughter, You Broke My Heart: Accidental Thrombosis at a Muscular Bridge" by Leon, Salazar, Moreira, and Angelini (*Tex Heart Inst J* 2006;33:380-2), which describes an acute anterior wall myocardial infarction due to a thrombotic occlusion of the left anterior descending artery proximal to a muscular bridge. The authors attribute the occlusion to the production, by extreme exertion, of high stress at the muscular bridge and to a consequent thrombotic occlusion more proximally.

I greatly enjoyed reading this article, which so simply and beautifully described the incidence, function, and potential complications of muscular bridging. I comment because I believe, due to the atherosclerotic plaque that was evident proximally, that the muscular bridge probably had little direct effect on the thrombus formation. I think that the underlying plaque, proximal to the muscle bridge, was activated in part as a consequence of the extreme stress this individual experienced and that the muscular bridge was an incidental finding. It is noteworthy that the left ventricular ejection fraction was preserved, most likely through collateralization that probably was recruited to some degree by intermittent coronary occlusion of the atherosclerotic lesion rather than by the muscular bridging. As is well known by Dr. Angelini and his colleagues, diastolic flow is so rarely impaired by a muscular bridge that this alone would not likely justify collateral recruitment.

In addition to its prompting of a review of this uncommon condition, the paper by Leon and associates is an excellent and scholarly example of the case report as a brief exploration into a mechanism that in this instance might have contributed to myocardial infarction in an unfortunate individual. As an aside, I hope that the patient's daughter had a complete recovery.

Morton J. Kern, MD, FSCAI, FAHA, FACC,
Professor of Medicine,
Associate Chief Cardiology,
Department of Cardiology,
University of California, Irvine,
Orange, California

This letter was referred to the authors, who reply in this manner:

We acknowledge Dr. Kern's constructive input on the subject of the cause-and-effect relationship between muscular bridging and myocardial infarction, as we discussed it in our recent case report.[1] The question that he raises is quite fundamental, and the following facts may shed some light upon it in specific application to our patient:

- Our patient was a well trained, regularly active individual who had previously completed several marathon runs, during the course of which he had been totally asymptomatic.
- The described event, which we interpreted as acute myocardial infarction, was a defined episode, with severe chest pain, cold perspiration, and dyspnea, during extreme exertion and mental stress.
- The electrocardiogram documented an anteroseptal acute infarction (non-transmural).
- The proximal left anterior descending artery (LAD) was found at delayed catheterization to be totally occluded (following persistent, de novo angina, which had appeared only after the original event).
- A guidewire pass resulted in clear, albeit partial, recanalization of the LAD (Fig. 1B in the original paper).
- The balloon pre-dilation yielded full expansion at very low pressure (unusual in a case of atherosclerotic plaque), which suggested that the occlusion was due mainly to thrombus. The authors cannot say whether an underlying plaque rupture had occurred.
- No evidence of atherosclerotic disease was present in other locations, and no calcium was visible in the long, occluded segment.

Although we certainly accept the possibility that our patient's myocardial bridge had nothing to do with the event, we still favor our hypothesis that it did. Even if the muscular bridge was not by itself the cause of preexistent effort-related ischemia and of the development of a collateral circulation, it could have triggered coronary spasm at the time of extreme adrenergic discharge and mechanical stimulation. Prolonged spasm could have resulted in proximal clotting.

Cases similar to ours have been published, as mentioned in our article, and we ourselves have had occasion to demonstrate, in the catheterization laboratory, coronary spasm at the level of a muscular bridge, in a patient with spontaneous Prinzmetal angina who had ST elevation during a treadmill test.[2]

Again, in our judgment, atherosclerotic disease—frequently observed proximal to an LAD muscular bridge—is surely a possible explanation of the acute event, but

it is not more likely in our patient than is our "primary clot just proximal to the myocardial bridge" hypothesis. We agree with Dr. Kern that clot formation was probably a dynamic event in our patient, with multiple episodes of spontaneous resolution and recurrence. Such behavior is likely the reason for the preserved left ventricular function. Ischemic preconditioning probably played a role. But again, this behavior also supports the hypothesis of a "primary" thrombosis more than that of a critical, chronic atherosclerotic lesion, with terminal thrombosis.

The authors would like to thank Dr. Kern for his interest and to add, in answer to his kind enquiry, that the daughter of our patient experienced complete recovery from her head trauma and coma.

Fernando Leon, MD,
Hector Salazar, MD,
Wendel Moreira, MD,
Paolo Angelini, MD,
Department of Cardiology,
Texas Heart Institute at
St. Luke's Episcopal Hospital,
Houston

References

1. Leon F, Salazar H, Moreira W, Angelini P. Daughter, you broke my heart: accidental thrombosis at a muscular bridge. Tex Heart Inst J 2006;33:380-2.
2. Angelini P. Coronary artery anomalies. A comprehensive approach. Philadelphia: Lippincott Williams & Wilkins; 1999. p. 147-50.

Use of Double Wires in Sternal Closure: A Useful Technique

To the Editor:

We would like to share with your readers our simple technique for sternotomy closure in obese patients (body mass index, >30).

Using #7 stainless steel wires, we usually apply 2 to 3 simple wires through the manubrium. Three double wires are placed through the body of the sternum. Double wires are placed peristernally. Using the forehand technique, we pass a simple wire peristernally through the 3rd intercostal space. The needle is reversed and is passed through the same intercostal space and then through the opposite space (Fig. 1A). Next, the 2 free ends of the wire are twisted around each other to form a loop (Fig. 1B). We usually place 3 double wires. The last wire through the sternum is usually a figure 8 in the area of the xiphisternum. To close the sternum, we cross all of the wires. A large twister is then engaged through the loops of one of the double wires (Fig. 1C). The wire is pulled up vertically until it is tight. Using a rotary movement of the wrist along with a vertical pull on the wire, we twist the wire tightly until twists are formed up to the sternum.

There are several advantages to this method. Commercially available wires like the Myo wires cost much more than the simple wires that we use. Our method requires only the use of a large twister with a large hook; even a simple bone hook can be used for this purpose. When the hook is used correctly, there is little chance of breaking the wire, and the result is a very rigid fixation of the sternum (Fig. 2). The only problem with this method is occasional bleeding from the chest wall consequent to the double penetration of the intercostal space with the needle. Such bleeding is easily controlled with a peristernal stitch using a Vicryl suture (Ethicon Inc., a Johnson & Johnson company; Somerville, NJ).

Kiessling and colleagues[1] studied the outcome of sternal closure in obese patients. Those researchers compared the standard methods with a double-wire technique, using commercially available wires, and found a substantially lower incidence of sternal dehiscence in patients in whom the double-wire method was used.

In patients with a fragile sternum, as from osteoporosis, we use simple wires or we supplement sternal closure with parasternal weaving.[2] Moreover, placing an additional wire at the lower end of the sternum is im-

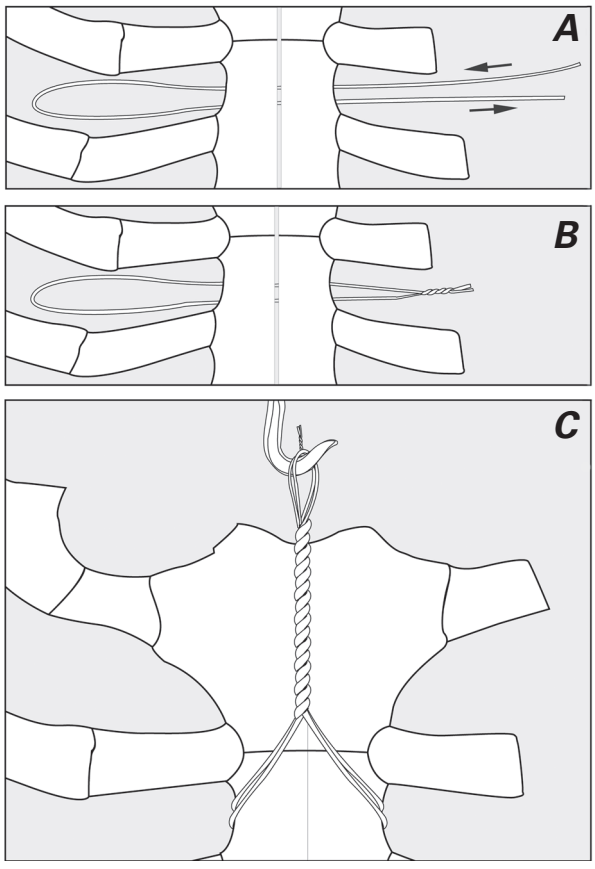

Fig. 1 *Technique of placing a double wire.*

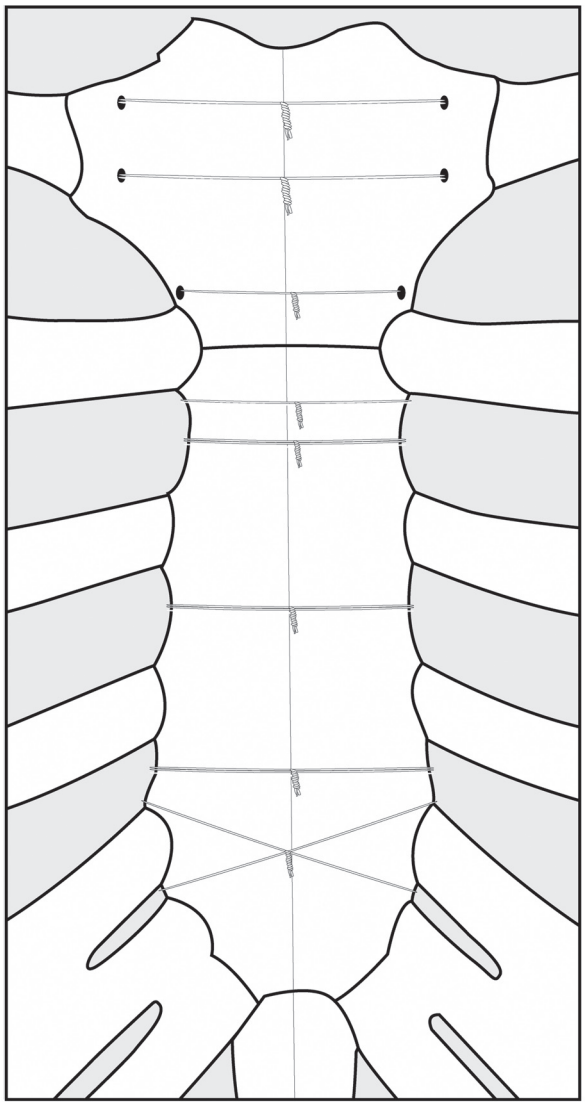

Fig. 2 *Completed sternal closure.*

portant, because this improves sternal stability against the distraction forces that affect the wound during the postoperative period.[3]

The reported rate of postoperative sternal dehiscence, a significant cause of death and morbidity, is as high as 8%.[4] In our surgical unit, our method of wiring has substantially reduced the rate of sternal dehiscence in all patients, including high-risk obese patients, to approximately 1%. We have used this method routinely for over 10 years and have not encountered sternal complication in any of the patients thus treated. We highly recommend this technique for sternal closure in obese patients.

Pankaj Saxena, MCh, DNB,
Igor E. Konstantinov, MD, PhD, and
Mark A.J. Newman, MBBS, FRACS,
Department of Cardiothoracic Surgery,
Sir Charles Gairdner Hospital,
Nedlands, Perth, Australia

References

1. Kiessling AH, Isgro F, Weisse U, Moltner A, Saggau W, Boldt J. Advanced sternal closure to prevent dehiscence in obese patients. Ann Thorac Surg 2005;80:1537-9.
2. Robicsek F, Daugherty HK, Cook JW. The prevention and treatment of sternum separation following open-heart surgery. J Thorac Cardiovasc Surg 1977;73:267-8.
3. Dasika UK, Trumble DR, Magovern JA. Lower sternal reinforcement improves the stability of sternal closure. Ann Thorac Surg 2003;75:1618-21.
4. Okies JE, Phillips SJ. Letter: Sternal approximation. Ann Thorac Surg 1974;17:423-5.