Delayed Chest Wall Pain Due to Sternal Wire Sutures

Charles E. Eastridge, MD, Saade S. Mahfood, MD, LCdr, William A. Walker, MD, USNR, and F. Hammond Cole, Jr, MD

Division of Thoracic Surgery, Veterans Administration Medical Center, and Department of Surgery, University of Tennessee, Memphis, Tennessee

This report describes 18 patients with disabling chest wall pain due to one or more sternal wire sutures. The pain occurred from 2 to 84 months after a median sternotomy. The pain was described either as sharp and stabbing or as a deep-seated ache. The involved wires had an exaggerated fibrous tissue reaction surrounding the twisted portion. The adjacent noninvolved wires had minimal reaction. In the last 7 patients, serial sections of the fibrous tissue revealed entrapment of one or more sensory nerve fibers. In 6 of the 7 electrical potentials were measured and found to be elevated, indicating wire damage during twisting. Ferroxyl tests confirmed the collection of iron ions at this anodic point as a result of corrosion. Removal of the involved wires and the fibrous tissue surrounding this anodic point relieved the symptoms of pain and tenderness resulting from entrapped sensory nerves.


Anterior chest wall pain that continues or arises weeks after a median sternotomy is usually attributed to incisional trauma, anxiety, or nonspecific musculoskeletal problems. A recent report has described a group of patients thought to have scar-entrapped neuromas causing a neuralgic-type pain that responded to multiple nerve blocks or neurolysis [1]. Others have suggested the pain is due to a hypersensitivity reaction to the nickel contained in stainless steel [2].

In most patients, however, in the absence of a specific cause, analgesics, nonsteroidal antiinflammatory agents, heat, or some form of exercise are prescribed [3, 4].

During the past 18 years we have encountered 18 patients in whom delayed chest wall pain after median sternotomy was due to corrosion of one or more sternal wires used to reapproximate the sternum. The recognition of this cause for pain is important, especially after coronary artery bypass operations, as the pain may be confused with recurrent myocardial ischemia due to graft failure. The purpose of this paper is to present 18 patients who had disabling chest wall pain after median sternotomy in which local inflammation and fibrosis occurred around the twisted portion of the wires.

Material and Methods

Between 1972 and 1990, 18 patients were seen at the Veterans Administration Medical Center, the University of Tennessee Bowld Hospital, and Methodist Hospital in Memphis, TN, and the US Naval Hospital, San Diego, CA, with disabling anterior chest wall pain due to sternal wires used to reapproximate the sternum after median sternotomy. Sixteen patients were men and 2 were women, ranging in age from 31 to 70 years (mean age, 55.5 years). Sixteen had coronary bypass operations and 2 were explored for hemorrhage after gunshot or stab wounds of the heart.

The pain occurred from 2 to 84 months (average, 21.2 months) after a median sternotomy. It was described either as sharp and stabbing or as an ill-defined, deep-seated ache with tenderness over the twisted portion of the wire. The pain was accentuated by pressure over the involved portion of the wire or any movement of the upper extremities that resulted in increased stretch tension of the tissues overlaying the anterior chest wall. It was occasionally described as radiating laterally to the adjacent chest wall, particularly if pressure was applied over the involved wire. Pain that radiated into the neck or anterior shoulder area was associated with involvement of the upper sternal wires and was accentuated by upward motion of the arms.

Before operation, each patient had a physical examination of the sternal area, consisting of palpation of each individual wire for pain or tenderness, posteroanterior and lateral chest roentgenograms, electrocardiogram, and routine laboratory studies. Six patients in this group were suspected of having myocardial ischemia as the cause for the pain. These 6 underwent several tests, which included electrocardiogram, exercise stress test, echocardiogram, and Holter monitoring.

In the 18 patients, a total of 64 wires were removed. There were 34 wires removed from areas that were tender to palpation and 30 from areas that were not tender.

In the last 6 patients studied, the electrical potential of all the wires removed, from both painful and nonpainful areas, was measured and compared with that of two pieces of unused type 316 stainless steel wire. The volt potential, measured in millivolts, was obtained by using a Corning Model 125 pH meter and a modified Corning...
Standard pH electrode. The electrode modification consisted of removing the electrode capsule and fixing alligator clamps to each electrode wire.

The twisted portion of each removed sternal wire was attached to one electrode (anode) and the remainder of the wire, which had been separated, was attached to the other electrode (cathode). The severed portions of the wire were folded in such a manner that the cut ends were not immersed in the conductive solution (normal saline solution). Each measurement was done in fresh electrolyte solution.

A ferroxyl test [5] was done on the wires removed from the last 6 patients in this series. This method employs a pH calorimetric indicator to demonstrate in vitro ionization and polarization in a corrosive cell. The test solution for the corrosive cell consisted of 10% potassium ferricyanide in 3% saline solution in agar. In the test, potassium ferricyanide will react with ionized iron at an anode to form a deep blue precipitate, Prussian blue (Fe₄[Fe(CN)₆]₃). The twisted portion of the wire (anode) was immersed and separated from the nontwisted portion (cathode) in the conductive solution and exposed for a period of 1 week.

A deep blue deposit on the twisted portion of the wire would indicate intense ionization of iron caused by corrosion resulting from damage of the protective chromic oxide coat. The chromic oxide coat is responsible for the corrosion resistance of type 316 stainless steel wire.

Histological studies were done on the fibrous reactive tissue found surrounding the twisted portion of the wires in the last 7 patients studied. Tissue was serially sectioned and stained with hematoxylin and eosin and S-100 immunoperoxidase.

**Results**

In each of the 18 patients, removal of the wires and the adjacent scar tissue resulted in complete relief from the pain and tenderness.

The sternal wires in areas that were tender to palpation exhibited a thick fibrous tissue reaction that surrounded the twisted portion of the wires (Fig 1). Adjacent wires from nontender areas exhibited very little fibrous reaction. Serial sections of the fibrous tissue surrounding the wires causing pain revealed one or more sensory nerve fibers entrapped in scar tissue (Fig 2).

Electrical potential measurements of the wires that caused pain ranged from 63 to 410 mV (mean, 200 mV); the range in the wires not associated with pain or tenderness measured from 5 to 30 mV. The electrical potential between two pieces of unused type 316 stainless steel wire measured less than 10 mV. Ferroxyl testing of the wires with increased electrical potential showed a deep blue discoloration located on the twisted portion of the sternal wire, indicating ionization of iron at this anodic point (Fig 3). The higher the electrical potential, the greater the blue deposit. The ferroxyl test was negative in all wires removed that were not associated with pain and tenderness.

**Comment**

With the introduction of stainless steel wire in the 1930s, it was thought that the inflammatory reaction secondary to electrochemical corrosion and direct chemical attack would be eliminated. Although stainless steel wires are generally well tolerated in body fluids, there are occasions when corrosion does occur [6, 7].

Stainless steel is a general term covering a large number of alloys, all of which have an iron base. Steel, roughly speaking, is a solution of carbon and iron. Resistance to corrosion may be achieved by adding various elements, each of which confers specific properties on the iron. The first and most important of these elements is chromium. The remainder of the elements in 316 stainless steel wire are nickel and molybdenum [8]. The addition of molybdenum to such steel further increases the resistance to corrosion in the presence of chloride ions and organic acids that occur in body fluids.
The resistance of stainless steel to corrosion depends upon the presence of a thin, invisible, but continuous film of chromium oxide measuring approximately $10^{-5}$ mm in thickness that covers the surface and insulates the metal from corrosive electrolytes contained in body fluids [9, 10]. In normal circumstances, this protective film is self-sealing (11). If, at any point in the placement of the wire, the protective chromic oxide coat is damaged to the extent that self-sealing cannot occur, this area becomes anodic to the rest of the wire and a simple voltaic cell results. In body fluids, an electric current will flow from the anodic (damaged) portion to the cathodic (undamaged) portion of the wire.

In regard to the flow of positive electricity, the area from which the current flows into the electrolyte solution (body fluid) suffers electrolytic dissolution or corrosion. The area of the undamaged wire, into which the current flows from the electrolyte solution, receives a deposition of hydrogen ions or metal ions. If oxygen is present on the cathode, it will combine with the discharged hydrogen ions to form water [10]. In tissue iron ions will accumulate in the area adjacent to the anode, stimulating an inflammatory response.

Damage to stainless steel wire may occur during placement, as a result of severe cold working (twisting), or by rough handling of the wire with instruments. Ideally, the instruments used to grasp or to cut the wire should be made of the same metal as the implant or be lined with tungsten carbide to prevent or reduce metal transfer. Fragments of different metals, when transferred to the implanted wire, will become anodic to the implant and a corrosive cell will be established [8].

With an appreciable degree of corrosion, metal particles of disintegration will accumulate in the tissue adjacent to the anode. The tissue responds not only to the degree and rate of corrosion, but to the specific tissue toxicity of the corrosive products. Accordingly, the reaction may vary from a minimal investment of the anodic area with fibrous tissue to various degrees of fibrosis in an attempt to wall off the irritant. The fibrous investment in these patients was found surrounding the twisted portion of the wire. The greater the measured electrical potential, the greater the speed of corrosion, and the greater the fibrous reaction. Because stainless steel is an iron alloy, iron ions are expected to accumulate around the anodic portion of the wire; this may be demonstrated by subjecting the wire to the calorimetric ferroxyl test. It is the iron ions that evoke the inflammatory reaction and the fibrosis. During the process of fibrosis, sensory nerves may become entrapped in the fibrous tissue, resulting in an entrapment neuropathy.

The pain resulting from entrapment neuropathy may at times evoke concern of graft failure or inadequate revascularization in the patient who has had coronary bypass and result in further costly cardiac studies. When nerve entrapment is suspected as the cause of pain, the diagnosis may be confirmed by physical examination of the sternal area. The areas overlying the involved wires are tender to palpation and, with pressure, the pain complaints will be reproduced.

Treatment consists of removal of the wires and the adjacent scar tissue that surrounds the twisted portion. This relieves the symptoms of pain and tenderness resulting from the sensory nerve entrapment.

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References

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J. Kent Trinkle, MD
Cardiothoracic Surgery
University of Texas Health Science Center at San Antonio
7703 Floyd Curl Drive
San Antonio, TX 78284-7841