ACUTE CALCIFIED SUBACROMIAL OR SUBDELTOID BURSITIS*

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IN recent years considerable interest has been manifested, especially in the English literature, regarding one of the causes for the so-called "painful shoulder," namely, calcified subacromial or subdeltoid bursitis. Like other conditions of unknown exact etiology, many forms of treatment have been proposed, each form having its own advocates, and each form producing satisfactory results. That the condition is not rare, but on the contrary quite common, is shown by the excellent report of Bosworth,[1] who examined 6,061 unselected, apparently healthy persons, and found calcium deposits in either one or both subacromial bursae in 165 (2.7 per cent): the calcium being present in sufficient quantity to be visible by means of fluoroscopy.

Definition.—Chiefly as the result of Codman's[2] epochal work on "The Shoulder," our conception of the term "calcified subdeltoid bursitis" has been immensely clarified. The terms "calcified subacromial" or "subdeltoid bursitis" are used synonymously, and indicate the presence of calcium deposits in the tendons of the short rotator muscles of the shoulder, namely, the supraspinatus, infraspinatus, subscapularis and teres minor, and the overlying serous subdeltoid bursa.

Nomenclature.—The confusion of the earlier writers in labeling this interesting condition is attested by the many descriptive terms given to it by the various authors. Our first recorded description in 1872 was by Duplay, who referred to it as "periarthritis humeroscapularis." Other authors have described the condition under the following captions: periarticular calcifications; painful shoulder; "Duplay's disease"; calcified bursitis; and subacromial calcifications.

ANATOMY

The subacromial bursa lies beneath the deltoid muscle and extends from the upper portion of the muscle to the under surface of the acromium process. It, therefore, separates the greater tuberosity of the humerus from the deltoid muscle, and its floor is in close contact with the tendons of the supraspinatus and infraspinatus muscle. All authors are agreed that there is no communication between the bursa and the shoulder joint. It acts as the gliding mechanism in abduction and rotation of the humerus and, therefore, disappears under the acromium during abduction.

The anatomical studies of the subacromial bursa received their greatest impetus from the observations of Codman and others, who called attention to the floor of the bursa rather than the bursa itself as the seat of pathological changes. The floor of the bursa is closely aligned with the tendons of the short rotators of the humerus, the most important of these being that of the supraspinatus muscle. This muscle originates in the supraspinatus fossa of the scapula and inserts in the uppermost facet of the greater tuberosity of the humerus. Although the tendons of the other short rotators, namely, the infraspinatus, subscapularis, and teres minor are closely fused in the formation of the capsule of the shoulder joint, accurate anatomical studies have clearly shown that it is the tendon of the supraspinatus muscle that is most commonly involved in this condition.

INCIDENCE

My interest in acute calcified subacromial bursitis was aroused when, during the course of a few years, in a small community of 10,000, I was personally able to see eleven such cases: a condition which I had previously thought to be extremely rare. This unwarranted preconception of the rarity of the condition was soon dispelled after reading Bosworth's[1] excellent statistical analysis. Bosworth's studies were made during the course of routine physical examinations on employees and applicants for employment of a large insurance company. This is the most comprehensive statistical study on the subject that I was able to find, not only in regard to the incidence of such calcifications in the general public, but also relative to the possible etiological factors in its causation. His observations covered a large group of presumably normal persons, and the results of these studies are most illuminating.

As stated before, calcium deposits were found in one or both bursae in 165 out of 6,061 examinations, an incidence of 2.7 per cent. All persons examined were office employees, the so-called white collar group, and worked either as typists or clerks. By careful x-ray and fluoroscopic studies, Bosworth was able to determine fairly accurately which of the tendons of the short rotators were involved. In over 50 per cent the deposit was found in the tendon of the supraspinatus. The tendon next most commonly involved was that of the infraspinatus. The right shoulder was involved twice as often as the left.

ETIOLOGICAL FACTORS

There is no known single factor involved in the production of this condition, the present consensus of opinion being that it is a degenerative process, a normal wear and tear, with multiple trivial trauma playing an important rôle.

Trauma.—The relationship of trauma to an acute attack of calcified subacromial bursitis is not well understood. In only a few cases is there a history of a definite single trauma. If a single trauma does immediately precede an attack, it is only an aggravating factor and its relationship is the same as that of a strain, either severe or trivial, to an inguinal hernia, namely, that the predisposing factor and pathological abnormality were already present. The effect of a single trauma is illustrated by the following case report, the only one in my small series in which trauma preceded the attack.

REPORT OF A CASE

CASE 1.—C. H., age 42, an insurance executive, while playing tennis struck his left shoulder against a brick wall. He was immediately seized with an acute, excruciating pain over the tip of the left humeral tuberosity. Palliative treatment afforded no relief, and operation was performed

* A résumé.

the next day under local anesthesia. The calcified mass was curetted out, with immediate relief. He was able to return to work the next day.

In only seven of Bosworth's forty-one cases of acute calcified bursitis was there a clear-cut history of antecedent trauma, and many were of a trivial nature, such as merely reaching for an object, that our imagination would have to be far-stretched to pin the blame on the trauma.

While acute trauma is only an aggravating factor, the question of repeated and usual trivial trauma must be given more consideration. The rotator tendons and the accompanying bursa are easily pinched between the humerus and acromium during abduction. Meyer,[8] after exhaustive studies on cadavers, and Codman have arrived at the following conclusions regarding the etiology of this condition: The tissues involved in subacromial bursitis are especially affected in what Meyer calls "use destruction." Minute tears are produced in the substance of the tendons. Because of the avascular nature of the tissues, necrosis occurs with degeneration of the fibers and the formation of calcium deposits. An acute inflammatory process occurs which produces tension in the unyielding fibers of the tendon, and finally breaks through and involves the overlying subacromial bursa.

Infection.—The possibility of a focal infection as a cause of this condition is without foundation. Removal of possible foci of infection has no influence on the disease. Cultures from the deposits and the surrounding tissues do not produce any pathogenic organisms, and although associated with severe pain there is no acute inflammatory reaction present.

Occupation.—Codman believes that the constant stretching and tension of the supraspinatus tendon, which occurs in certain occupations, is an important etiological factor. Bosworth's series showed that occupations requiring constant prolonged abduction of the arm foster the formation of calcium deposits. One-third of my cases occurred in persons employed as ranch workers who did a great deal of pruning and thus subjected their supraspinatus tendons to frequent stretching over a long period of time.

SYMPTOMS AND DIAGNOSIS

The symptoms of acute calcified subacromial bursitis are characteristic, and the diagnosis is usually easy. There is no mistaking an "acute" attack—the pain is severe and excruciating. The pathognomonic symptoms and findings are as follows:

1. There is an acute agonizing pain in the shoulder over the greater tuberosity of the humerus. The onset is usually sudden and may awaken the patient out of a sound sleep.

2. Tenderness is the most prominent finding, is exquisite in character, and is always present over the greater tuberosity of the humerus. The shoulder is "as sore as a boil." The point of tenderness and its severity cannot be mistaken for any other condition.

3. Voluntary abduction is impossible and rotation of the shoulder is very painful. There is considerable muscle spasm of the deltoid, and the arm is held rigidly to the side.

4. Roentgen findings are characteristic. The calcareous deposit is readily seen overlying the greater tuberosity of the humerus. It varies in density and thickness and occasionally may be multiple. Caution must be exercised in using the proper roentgenological technique, as the calcium deposit may be overlooked if it is superimposed on the shadow of the humerus or the acromium. Bosworth recommends fluoroscopy in the detection of the calcium deposits. By so doing he is able to put the shoulder within its full range of motion and take a spot film of each deposit.

The acute symptoms usually subside in about two weeks, but only a small minority of the patients are willing "to wear it out." Because of the severe pain and disability, they demand that something be done.

TREATMENT

This condition is sometimes self-limited and a few of the patients will obtain relief, at least temporarily, without any form of therapy. This has happened in two of my patients who had a complete remission of symptoms without any treatment. This fact leads to confusion when one attempts to evaluate the various forms of therapy, and also explains why it is possible for a proponent of a particular form of treatment to claim success for his own method.

The criterion of success in any form of therapy, whether surgical or medical, is, of course, the end-results. However, there are other factors which must be given serious consideration, and these include the length of disability, the period of hospitalization, the immediate relief of pain, the restoration of normal function, and the question of permanency. I am in agreement with the majority of authors that the best treatment for the acute attack is early and prompt incision of the calcified deposit. This is a relatively minor surgical procedure, can be performed under local or general anesthesia or with brachial-block anesthesia. The period of hospitalization is seldom over three or four days. Relief of pain is complete and immediate, with no possibility of recurrence. Normal movement is usually obtained in two to three weeks. However, there are a certain number of patients who will refuse surgical treatment. In these cases one of the other well-recognized forms of treatment may be administered. Although these other forms of treatment produce good and sometimes excellent results in experienced hands, the surgical excision of the calcified mass is the surest and quickest method of obtaining complete relief.

The following methods are available for the treatment of this condition:

1. *Surgical Excision by Open Operation.*—This is the method of choice used by the majority of orthopedic surgeons. Using either local, general, or brachial-block anesthesia, an incision is made directly over the involved bursa. After bluntly

separating the fibers of the deltoid muscle, the distended, glistening bursa comes into view. The wall of the bursa is incised and the thick, white, gritty, putty-like mass is gently curetted out. The curettage will also remove the calcified material in the tendon of the underlying supraspinatus muscle or other rotator tendons. The procedure takes only a few minutes. The wound is closed without drainage and the arm is immobilized for a few days in a sling. The relief of pain is immediate and complete. Drainage and suppuration of the wound do not occur. Active and passive motion are encouraged and full restoration of function is complete within two or three weeks. The reaction of the patient is one of extreme satisfaction and gratification for the relief from pain. I have seen no recurrence of even the slightest symptom. The excellent results obtained with minimal time loss compare most favorably and far surpass the other forms of treatment.

2. *The Injection of Procaine Into the Bursa.*—Haggart and Allen [4] were able to report good results by the injection of 20 cubic centimeters of two per cent procaine hydrochloride directly into the bursa and shoulder joint. Soon after reading their report, I had the opportunity to attempt this procedure on one case, with unfavorable results. Relief of pain was immediate, but within forty-five minutes after the effects of the procaine had worn off, the pain returned with sudden violence, so that open incision was performed with immediate relief.

However, in cases of acute bursitis without calcification, I was able to obtain good results by the injection of procaine. Haggart and Allen explain the relief of pain by improvement in the local circulation, thus causing absorption of the calcified deposit and probably a rupture of the calcified material from the tendons into the bursa.

3. *High Voltage Roentgen Therapy.*—The technique used by Chapman [5] is as follows: 200 k.v., 2 mm. cu., 1 mm. al., 250 r in air. A series of four or five treatments are given twice a week followed by a rest of two weeks, and a second series of two or four treatments only if indicated. Results were not constant, some cases giving striking results and apparently similar cases proving failures. It is apparent from a study of Chapman's fifty-four cases that few were of the acute, fulminating type, as only fifteen were in sufficient pain to disturb sleep and only seven were taking analgesics.

Although the use of roentgen therapy seems easy for the patient and is relatively inexpensive compared with the 100 per cent prompt cures obtained by surgery, Chapman's results were not impressive, as attested by the following figures:

22 per cent obtained no relief
10 per cent obtained slight relief
46 per cent obtained marked, but incomplete relief
22 per cent obtained complete relief.

Compared with the immediate relief obtained by surgery, in many cases roentgen therapy did not give relief for two weeks, while in others relief was not obtained for a month or more. Chapman claimed no superiority or priority for his method, but has evaluated in a clear, concise, and logical manner the results of this form of therapy.

4. *Physiotherapy.*—All forms of physiotherapy have been used, principally baking and diathermy. In my opinion, they have been merely palliative in the milder cases and have aggravated the pain in the fulminating cases. If relief does occur, it is usually partial or temporary with subsequent recurrent attacks to be expected. However, physiotherapy is a useful adjunct in the postoperative prevention of adhesions and in securing earlier mobility of the arm.

5. *Simple Needling of the Bursa.*—The simple withdrawal of a small amount of fluid from the bursa, with relief of pain, was first reported by Alanson Weeks of San Francisco in 1908. It is obvious that the withdrawal of fluid from the bursa will reduce tension and eliminate pain, but it seems illogical to suppose that such a procedure will remove the irritating calcium deposit with the constant threat of subsequent attacks.

6. *Multiple Needling of the Bursa.*—This method has been described by Weeks and Delprat.[6] It can be performed under local or gas anesthesia. It will relieve pressure from the bursa and will allow some of the calcified mass, if soft and semifluid, to escape from the bursa into the surrounding tissue. The relief from pain is not as prompt and complete as following surgical incision, and the entire calcified mass cannot be removed. Weeks and Delprat believe, however, after a study of forty patients in which relief was obtained by this method, that the calcium will be absorbed unless it has organized into bony hardness.

7. *Bursal Irrigations.*—By inserting two large needles into the bursa and forcing normal saline solution through one needle to flow out the other, Patterson and Darrach [7] obtained good results in sixty-three patients. The soft calcified specks were thus able to be washed out by the fluid under pressure.

8. *The Ammonium Chloride Treatment.*—The latest form of therapy for this condition is that advocated by Dick, Hunt and Ferry [8] in 1941. They report excellent results, and their treatment consists of

(*a*) Relatively large doses of ammonium chloride.
(*b*) Rest of the diseased part.
(*c*) Physical therapy.
(*d*) Removal of foci of infection.

Theirs is the first mention made of this form of therapy. The ammonium chloride is administered in doses of one gram or more four times a day, preferably in enteric-coated tablets. The ammonium chloride is used to lower the hydrogen ion concentration of the blood and lymph. They claim rapid disappearance of pain and, when not too dense, absorption of the calcified deposit, due to the mild acidosis produced by the ammonium chloride.

Rest of the diseased part is obtained by the use of a sling or by resting the arm on a pillow.

Physical therapy is used with care in the form of diathermy or infra-red baking and all possible foci of infection, such as apical dental infections, infected tonsils, etc., are sought for and eliminated. From a study of their case histories it requires on an average of two weeks for the pain to be relieved.

9. *Immobilization and Analgesics.*—Some patients prefer "to tough it out," and will consent to no active treatment. Immobilization of the arm by means of a sling and the use of analgesics by mouth resulted in the relief of pain in two of my patients. Their calcium deposit is still present and recurrences are always possible.

The relief of pain without any active form of treatment can at times be expected, as Bosworth [1] was able to observe the disappearance of the calcium deposit in a few patients without treatment in any form.

AUTHOR'S MATERIAL

Eleven cases were observed and treated in this series: four (36 per cent) were males and seven (64 per cent) were females. Ages ranged from 33 to 56 years, the average age being 46 years. Seven patients submitted to operation and obtained immediate and complete relief. All of these patients had tried palliative treatment without benefit. One patient refused treatment and left town. It was learned that she submitted to surgical operation elsewhere a few weeks later. Three patients obtained relief of pain after a period of two to three weeks following immobilization and the use of analgesics, but they suffered during that time severe and excruciating pain which only subsided gradually.

SUMMARY AND CONCLUSIONS

1. The possibility of calcified subacromial bursitis must always be considered when dealing with the acute painful shoulder.

2. Diagnosis can always be made with certainty by fluoroscopy and x-ray.

3. Repeated trivial trauma is probably the most important etiological factor.

4. Focal infection probably plays no part in the production of the calcium deposit.

5. The condition has its inception in the tendons of the short rotators of the humerus, especially the supraspinatus, and the subacromial bursa is secondarily involved.

6. The calcium deposit may be present for a considerable length of time without the production of acute symptoms.

7. The removal of the calcium deposit by surgical incision is the treatment of choice in the majority of cases. This is especially true in the acute fulminating type. Relief is immediate, complete, and permanent.

8. Other methods of treatment, as outlined above, have also proved successful.

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MEDICAL EPONYM

Pardee's Sign

Harold E. B. Pardee (b. 1886) describes "An Electrocardiographic Sign of the Coronary Artery Obstruction" in the *Archives of Internal Medicine* (26:244-257, 1920). A portion of the article follows:

". . . It is hoped to show that obstruction of a branch of coronary artery is followed by a sign which is characteristic of this condition and is really recognizable in the human electrocardiogram.

". . . The characteristic changes appearing a day or two after the obstruction are as follows: The QRS group is usually notched in at least two leads, and usually shows left ventricular preponderance. The T wave does not start from the zero level of the record in either Lead 1 or Lead 3, though, perhaps, from a level not far removed from it, and in this lead quickly turns away from its starting point in a sharp curve, without the short, straight stretch which is so evident in normal records preceding the peak of the T wave. The T wave is usually of larger size than customary and accordingly shows a somewhat sharper peak. The T wave is usually turned downward in Lead 2 and in one other lead. Not all of these changes are to be found in every record, but enough of them are present to give it a characteristic appearance."—R. W. B., in *New England Journal of Medicine.*

MEDICAL EPONYM

Kümmell's Disease

Professor Hermann Kümmell (1852–1937) described this condition in a paper, entitled "Die rareficirende Ostitis der Wirbelkörper [Rarefactive Ostitis of the Vertebral Bodies]," published in the *Verhandlungen der Gesellschaft deutscher Naturforscher und Aertze* (64:282-285, 1892). A portion of the translation follows:

". . . To sum up, we are dealing with an injury, often of trifling nature, that affects the vertebral column either directly or indirectly, and so far as the immediate effect is concerned, leaves no sign of its occurrence. After months of complete health, there begins a rarefactive process in the vertebras, which finally results in atrophy of their substance. Suppuration does not occur in this pathologic process, as in tuberculous spondylitis, nor is there any thickening of the bony mass, as in syphilitic processes, or bony deposition and change, as in arthritis deformans. . . .

"The diagnosis of the condition, after what has been said, offers no great difficulties; and if we consider the most important factors—the effect of trauma in an otherwise healthy person, transitory pains and gibbus formation later, with the accompanying local and referred discomfort—confusion with tuberculous spondylitis or other disease of the vertebras is easily avoided."—R. W. B., in *New England Journal of Medicine.*