Pectoralis major ruptures: A review of current management

Article in Journal of shoulder and elbow surgery / American Shoulder and Elbow Surgeons ... [et al.] - April 2015
DOI: 10.1016/j.jse.2014.10.024 · Source: PubMed

CITATIONS 18
READS 1,710

4 authors:

Usman Butt
Salford Royal NHS Foundation Trust
15 PUBLICATIONS 154 CITATIONS
SEE PROFILE

Lennard Funk
Wrightington, Wigan and Leigh NHS Foundation Trust
124 PUBLICATIONS 855 CITATIONS
SEE PROFILE

Saurabh Sagar Mehta
University Hospitals of North Midlands, UK
24 PUBLICATIONS 217 CITATIONS
SEE PROFILE

Puneet Monga
Wrightington, Wigan and Leigh NHS Foundation Trust
47 PUBLICATIONS 159 CITATIONS
SEE PROFILE

Some of the authors of this publication are also working on these related projects:

- Textbook of Musculoskeletal Medicine View project
- Rehabilitation after rotator cuff repair View project
Pectoralis major ruptures: a review of current management

Usman Butt, FRCS (Tr & Orth)*, Saurabh Mehta, FRCS (Tr & Orth), Lennard Funk, MSc FRCS(Tr & Orth) FFSEM(UK), Puneet Monga, MSc, DipSportsMed FRCS (Tr & Orth), MD

Upper Limb Unit, Wrightington Hospital, Wigan, Lancashire, UK

Background: Rupture of the pectoralis major tendon is increasing in incidence, with a spike in the number of reported cases in the last decade. This is commonly attributed to an increased interest in health, fitness, and weight training combined occasionally with concomitant use of anabolic steroids. It is essential for the diagnosis to be recognized and for the patient to be referred to a surgeon with expertise in dealing with these injuries so that appropriate and informed care can be implemented.

Methods: Based on a comprehensive review of the literature and expert opinion, we present a review of pectoralis major ruptures, including information pertaining to the anatomy and biomechanics of the musculotendinous unit and how this relates to the injury pattern and management; the clinical diagnosis and indications for additional imaging; and the indications for nonoperative and operative management along with the authors’ preferred technique. A summary of outcomes is presented.

Conclusion: The combination of patient demographics and clinical features frequently yields an accurate diagnosis, but further imaging is helpful. Magnetic resonance imaging with dedicated sequencing is the investigation of choice and can aid in diagnosis, surgical planning, and providing important information about prognosis and outcome. Early surgery is preferable, but good outcomes in the chronic setting are achievable. With a detailed understanding of the anatomy, direct repair to bone is possible with either transosseous or anchor repair techniques in acute and the majority of chronic cases. In chronic cases in which direct repair is not achievable, autograft and allograft reconstruction should be considered.

Level of evidence: Narrative Review.

Keywords: Pectoralis major; tendon; repair; reconstruction
Anatomy and biomechanics

The PM has a complex musculotendinous morphology, an understanding of which is essential for any surgeon considering surgery on this structure. Anatomic studies have demonstrated the presence of 2 heads: the clavicular head, arising from the medial half of the clavicle; and the larger sternal head, arising from the second to sixth ribs, the costal margin of the sternum, and the external oblique aponeurosis. The sternal head is the much larger of the 2, accounting for >80% of the total muscle volume, and can be further subdivided into 7 overlapping segments.10,21,26

The 2 muscle heads converge laterally into a rather broad and flat bilaminate tendon consisting of an anterior layer, formed from the clavicular head and the upper segments of the sternal head coursing inferolaterally, and a posterior layer, formed from the lower segments of the sternal head coursing superolaterally (Fig. 1). Immediately before insertion on the lateral edge of the intertubercular sulcus, the 2 laminae of the tendon fuse.10 In the most recent cadaveric study based on 6 specimens, both the anterior and posterior layers had an average length of 5.4 cm, with respective tendon widths about 1 cm less than their lengths without the “twist” noted in previous studies.10,38 The investing layer of fascia of the PM is continuous with the fascia of the arm and medial intermuscular septum.24

The PM is innervated by the medial and lateral pectoral nerves. The medial pectoral nerve (C8-T1) arises from the medial cord of the brachial plexus in the majority of cases. It passes through the pectoralis minor, along the lower border of which it then runs before supplying the inferior portion of the PM.24,26 The lateral pectoral nerve (C5-7), the larger of the 2 nerves, commonly arises from the lateral cord of the plexus before traversing along the upper border of the pectoralis minor muscle. It then passes to the undersurface of the PM muscle along with the pectoral branch of the thoracoacromial artery, supplying the upper two thirds of the PM.24,26 In a recent cadaveric study, the lateral and medial pectoral nerves were found to pierce the pectoral musculature at a mean of 10.1 cm and 8.6 cm, respectively, from the lateral edge of the sternum.25

The PM’s primary role is as an adductor and internal rotator of the arm, although there is a contribution to forward flexion by the clavicular head.26 The muscle is somewhat unusual in that it consists of myofibers of varying lengths.38 This allows differential shortening velocities within the muscle, resulting in the possibility of power production being maximized over a broad range of motion.9,38

In an assessment of individual fiber lengths, Wolfe et al38 found that through an arc of forward flexion, excursion remained consistent, averaging about 19%. However, when the same measurements were done through an arc of 30° extension from neutral, the inferior fibers had an excursion of 40%, twice that of the more superior fibers. Hence, one can see that in the extended bench press position with an eccentrically loaded musculotendinous unit, a biomechanical explanation for the incidence of PM tendon ruptures exists. It is proposed that the tendon fails in a predictable sequence, with the inferior segments of the sternal head failing first, followed by the more superior segments of the sternal head and subsequently the clavicular head.7,38

Etiopathogenesis

The overwhelming majority of cases occur in muscular young adult men aged between 20 and 40 years during bench press, although a number of other demanding activities have been reported as causative, including rugby, wrestling, and boxing, among others.20,23,26,38,41 It is postulated that the male predominance relates to the less elastic nature of male tendons, lower tendon to muscle diameter, and engagement in higher energy activities, although there is no evidence to support this.1 Another less common subset of PM ruptures is in the elderly, thought to be secondary to stiff, atrophic muscles contracting during relatively strenuous activities, such as manual transfers.7

Anabolic steroids are frequently associated with tendon ruptures, including those of the PM. Although the reasons are unclear, studies have suggested that anabolic steroids result in stiffer tendons that absorb less energy and fail with
less elongation and with inferior stress values. On a histologic level, anabolic steroid use is associated with collagen dysplasia, increased vascularization and cellularity, and microdamage of collagen fibers.

Presentation and diagnosis

The diagnosis of a PM rupture is usually apparent from the history and the clinical setting. Frequently, there is a sudden pain at the medial aspect of the upper arm associated with a “pop” felt by the patient. This most commonly occurs with eccentric contraction during bench press. In the acute setting, physical examination may reveal ecchymosis over the anterolateral chest wall and upper arm along with a variable degree of swelling. Loss of the anterior axillary fold with an asymmetric muscle outline that is retracted medially is a useful diagnostic feature (Fig. 2), although these features may be partially obscured acutely as a result of soft tissue swelling. The absence of the anterior axillary fold can be accentuated by abducting the affected arm or with resisted adduction. Testing the power of the muscle is helpful for documentation purposes and as a baseline for comparison after any intervention. This may be carried out clinically or more formally with dynamometry.

The use of plain radiographs in the diagnosis and characterization of PM ruptures is limited. Loss of the PM shadow is a finding that is described but is inconsistent, and its presence or absence should not influence decision-making. In the rare case of a bone avulsion, plain radiographs may be useful. Ultrasound assessment can be a useful adjunct to clinical examination when the diagnosis is in doubt or when there is an unacceptable delay to magnetic resonance imaging (MRI). However, MRI is the investigation of choice for its added value in the characterization of tears and for surgical planning.

A standard shoulder MRI study will not be sufficient to fully identify or to characterize a PM tear, as most sequences will not extend caudally enough to include the tendinous insertion. A dedicated sequence is required with axial slices extending superiorly from the quadrilateral space and inferiorly to the deltoid tuberosity along with coronal oblique cuts. For a thorough assessment, it is suggested that T1, T2, and proton density images should be obtained, although it is the T2-weighted axial images that provide the most useful information, particularly in the acute setting. In the presence of a tear, the normally low signal intensity tendon is absent from a point 1 to 1.5 cm inferior to the quadrilateral space and 1 cm superior to the origin of the lateral head triceps. The retracted stump can be traced and visualized with abnormally high signal intensity adjacent to it, which may be better highlighted with fat saturation sequences. T1-weighted images are more helpful in identification of chronic tears.

Classification

The traditional descriptive classification system laid out by Tietjen divides PM injuries into 3 principal categories ranging from a contusion through partial to complete tears. Complete tears are further subdivided into the anatomic location, whether that is the muscle origin, muscle belly, musculotendinous junction, or tendinous insertion. A more contemporaneous classification has been proposed that takes note of the importance of the chronicity of the tear, the location, and the thickness and width of the tear. Certainly, the chronicity and location of the injury are crucial to operative planning and can drastically alter the nature of planned intervention, particularly with regard to whether an anatomic repair is feasible or whether graft reconstruction may be required. With regard to the extent of the tear, the authors of the classification suggest that normal tendon thickness should be 4 mm thick (2 mm per layer) and 4 cm wide so that intraoperative estimation of any remaining tendon against these estimations will provide an accurate classification of partial or incomplete tears. There are no data regarding intraobserver or interobserver reliability to support this classification, and its applicability and practicality are debatable. Nevertheless, it does draw attention to the importance attached to understanding the anatomy of the PM in approaching this type of surgery. In the senior author’s experience, the key determinants to surgical planning are the chronicity of the injury and its location along the muscle-tendon unit.

Management and outcomes

Nonoperative management of PM ruptures tends to be reserved for the lower demand and elderly subgroup of patients or those not wishing to undergo surgery. It may also be appropriate to treat certain partial tears and tears of the muscle belly in this way. Initially, the affected limb...
<table>
<thead>
<tr>
<th>First author</th>
<th>Publication date</th>
<th>Case number</th>
<th>Mean age</th>
<th>Mean follow-up</th>
<th>Surgical management</th>
<th>Outcome</th>
<th>Conservative management</th>
<th>Outcome</th>
<th>Specific complication</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schepsis</td>
<td>2000</td>
<td>17</td>
<td>29</td>
<td>28</td>
<td>13 (7 chronic)</td>
<td>16 (96%) satisfied</td>
<td>4</td>
<td>51% patients satisfied</td>
<td>NR</td>
<td>No significant difference in outcome of acute and chronic repairs</td>
</tr>
<tr>
<td>Hanna</td>
<td>2001</td>
<td>22</td>
<td>30.9</td>
<td>21.6</td>
<td>12</td>
<td>6 group I* 3 group II*</td>
<td>10</td>
<td>1 group I* 7 group II*</td>
<td>NR</td>
<td>—</td>
</tr>
<tr>
<td>Aärimaa</td>
<td>2004</td>
<td>33</td>
<td>28</td>
<td>52</td>
<td>33 (18 chronic)*</td>
<td>13 excellent 17 good 3 fair</td>
<td>NA</td>
<td>NA</td>
<td>NR</td>
<td>—</td>
</tr>
<tr>
<td>Zvijac</td>
<td>2006</td>
<td>27</td>
<td>31.6</td>
<td>12.3</td>
<td>19</td>
<td>18 excellent 1 poor 3 fair</td>
<td>8</td>
<td>1 poor</td>
<td>NR</td>
<td>—</td>
</tr>
<tr>
<td>Kakwani</td>
<td>2007</td>
<td>13</td>
<td>28.6</td>
<td>23.6</td>
<td>13</td>
<td>6 excellent 6 good 1 poor</td>
<td>NA</td>
<td>NA</td>
<td>1 traumatic rerupture 1 postoperative hematoma (surgery required) 11 returned to preinjury sporting level Active-duty military population Better outcome with acute repair All returned to active duty</td>
<td></td>
</tr>
<tr>
<td>Antosh</td>
<td>2009</td>
<td>14</td>
<td>31.4</td>
<td>NR</td>
<td>14 (6 chronic)</td>
<td>5 excellent 5 good 5 average 1 poor</td>
<td>NA</td>
<td>NA</td>
<td>NR</td>
<td>—</td>
</tr>
<tr>
<td>He</td>
<td>2010</td>
<td>9</td>
<td>32</td>
<td>80.4</td>
<td>9 (2 chronic)</td>
<td>3 excellent 5 good 1 fair</td>
<td>NA</td>
<td>NA</td>
<td>None</td>
<td>—</td>
</tr>
<tr>
<td>Shah</td>
<td>2010</td>
<td>10</td>
<td>33.9</td>
<td>20.3</td>
<td>10</td>
<td>8 satisfied 1 moderately satisfied 1 unsatisfied</td>
<td>NA</td>
<td>NA</td>
<td>1 wound infection</td>
<td>Elite athletes No difference between acute and delayed repair</td>
</tr>
<tr>
<td>Garrigues</td>
<td>2012</td>
<td>34</td>
<td>24</td>
<td>33</td>
<td>24 (3 chronic, 2 allograft reconstructions)</td>
<td>14 excellent 4 good 1 fair/poor</td>
<td>NA</td>
<td>NA</td>
<td>1 keloid scar 1 infection (surgery not required) 1 numbness in ulnar nerve distribution resolved; 1 ulnar nerve numbness and weakness resolved, 1 persistent radicular pain nonresolving</td>
<td></td>
</tr>
</tbody>
</table>
is rested in a sling (arm in adduction and internal rotation) supplemented with cryotherapy for swelling control and analgesia as required. Passive exercises can begin immediately as tolerated, followed by active assisted and active exercises during the course of the subsequent 6 weeks. After this, resistance therapy can be implemented and unrestricted activity allowed at 2 to 3 months.24,26

Although there are infrequent reports of higher demand patients being successfully managed nonoperatively,17 the literature overwhelmingly supports surgical treatment for active individuals.1,7,24,30 The literature, however, is based largely on case reports, small series, and systematic reviews of these small series. There is a lack of high-quality trials. Outcomes in the literature are presented heterogeneously, although the criteria developed by Bak et al,4 grouping outcomes as excellent, good, fair, and poor, are the most frequently adopted. A summary of the more recent studies is presented in Table I.

The chronicity of a tear may have an impact on the repair strategy, but a good outcome is still achievable, and chronicity should not be a barrier to surgical management, even years from the original time of injury.1,7,24,26,30 The definition of a chronic tear varies between reports,1,20,30 although it is possible that there may be a degree of muscle belly retraction even by 3 weeks. Techniques of medial fascial release performed through a separate incision have been described in an effort to mobilize the myotendinous unit sufficiently to allow primary repair,2 although a reconstruction rather than repair may be more appropriate if sufficient mobilization of the tendon is not possible through the primary incision. In performing any fascial release and clearance of adhesions, care must be maintained to avoid injury to the pectoral nerves.

Autograft and allograft reconstructions are described alternative techniques in the chronic setting when primary repair is not possible and should always be considered and available before a chronic tear of the PM is explored. Autograft options include hamstring, fascia lata, and bone patellar tendon.6,15,29,34,40 Allograft reconstruction with Achilles tendon is the authors’ preferred option as it avoids donor site morbidity, has excellent loading characteristics, has favorable physical dimensions for this indication, and has a good reported outcome.39

Repair techniques vary considerably in the literature, specifically with regard to the fixation method. Reported methods include transosseous suture fixation with the possible addition of a bone trough, anchor fixation, and cortical button fixation. Attachment of an avulsed tendon to the clavpectoral fascia has also been described.19 When the rupture occurs at the muscular/musculotendinous portion, direct repair to a tendon stump has been reported successfully.28 Sherman et al13 compared transosseous sutures, suture anchors, and a cortical button technique, noting no significant difference between fixation devices with regard to cyclic loading or load to failure properties. Ultimate failure of the constructs occurred at the suture-tendon
interface as one might expect. Rabuck et al\textsuperscript{27} reported a bone trough technique to provide the strongest repair construct for PM repair. They did, however, encounter one proximal humerus fracture during testing in their cadaveric study. This complication has a precedent in the clinical literature.\textsuperscript{35} Given the small numbers, heterogeneous techniques, and lack of controlled trials, it is impossible to meaningfully determine the best fixation technique, but all have been reported to be successful.

**Operative technique—authors’ preference**

The preoperative imaging is reviewed with particular attention to the degree of retraction and presence of any intact portion, usually the clavicular head. We use both dynamic ultrasound and MRI in most cases.

The patient is placed in the supine position under general anesthesia. An arm holder (TRIMANO; Arthrex, Naples, FL, USA) is used to allow easy positioning of the arm. Intravenous antibiotics are given at induction, and the skin is prepared with antiseptic paint. The surgical site is draped with the arm free and the axilla excluded with povidone-iodine (Betadine)–impregnated occlusive drapes.

A skin crease oblique incision is made in the deltopectoral groove. We avoid axillary incisions because of the risk of deep infection. In cases of rupture of both the clavicular and sternal heads, dissection is undertaken medial to the deltoid, preserving the cephalic vein. When the clavicular head is intact, the torn or ruptured sternal head is found underneath the clavicular portion, and one needs to dissect medial to the clavicular head to identify it. In chronic cases, a “zone of injury” is sought. This is a result of the resolved hematoma where scarred adherent tissue encloses the torn tendon/muscle and denotes the area of injury.
The retracted tendon is identified and mobilized over stay sutures. In chronic cases, the tendon may be adherent to the chest wall, and mobilization with blunt dissection is undertaken. Dissection and mobilization should extend as far medial as the sternum, both superficial and deep to the PM, with particular care to avoid injury to the medial and lateral pectoral neurovascular bundles.31

The PM footprint at the lateral crista of the intertubercular groove is lateral to the long head of the biceps. The footprint will be more obvious in cases in which the anterior tendinous layer (comprising mainly the clavicular head) is intact; but in these instances, it is important to repair the posterior layer to its correct site just superior and posterior to the intact anterior layer. The footprint is prepared by superficial decortication. Three bone anchors are placed in a triangular configuration, the middle being somewhat more medial than the superior and inferior anchors. One limb of the suture from an individual anchor is passed through the PM tendon with a cruciate stitch; the contralateral limb is then pulled through to parachute the tendon down onto the footprint.31 Standard surgical knots are tied down to secure the repair. The steps can be viewed pictorially in Figures 3 and 4.

The safe range of motion (before the repair is on tension) is determined before wound closure by taking the arm through gentle range of motion, and this is documented. Postoperatively, the patient’s arm is rested in a sling.31 Active hand, wrist, and elbow exercises are allowed immediately. Shoulder motion is initially restricted to passive motion within a safe range determined at surgery (usually external rotation to neutral and forward elevation to 60°). Between 3 and 6 weeks, active assisted motion is instituted with the physiotherapist, progressing to active motion after this time. Rehabilitation is individualized on the basis of the chronicity, size, and location of the tear; quality of tendon and repair; and patient factors. Figure 5 demonstrates the postoperative appearances after a successful repair.

**Conclusion**

Rupture of the PM is an uncommon condition. It occurs mainly in the male population with eccentric loads during bench press or similar setting with the arm in an extended position. In either the acute or chronic setting, the patient’s demographics and history combined with the pathognomonic feature of loss of the anterior axillary fold are diagnostic. MRI with dedicated sequencing is the investigation of choice and can aid in diagnosis, surgical planning, and prognostication. Early surgery within 3 weeks is preferable, but good outcomes in the chronic setting are achievable. With a detailed understanding of the anatomy, direct repair to bone is possible with either transosseous or anchor repair techniques in acute and the majority of chronic cases. In chronic cases in which direct repair is not achievable, autograft or allograft reconstruction should be considered.

**Disclaimer**

The authors, their immediate families, and any research foundation with which they are affiliated have not received any financial payments or other benefits from any commercial entity related to the subject of this article.

**References**


