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Original Article

The management of secondary frozen shoulder after anterior shoulder dislocation – The results of manipulation under anaesthesia and injection

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ABSTRACT

Introduction: Patients with secondary frozen shoulder following anterior dislocation were treated with manipulation under anaesthesia (MUA) and injection.

Methods: Ten patients included in study. Oxford Shoulder Scores (OSS), range of motion (ROM) and need for any further treatment measured.

Results: Mean follow-up of 93 weeks. OSS and ROM improved in all patients. Three patients required repeat MUA. Two patients developed recurrent instability.

Discussion: Secondary frozen shoulder may be more recalcitrant. Recurrent instability is a risk following anterior shoulder dislocation. It is feasible that by performing an MUA to maximise mobility, stability may be sacrificed. It should be performed with caution.

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1. Introduction

Patients with frozen shoulder or adhesive capsulitis characteristically present with a painful reduction in active and passive range of motion. The majority of these cases have no identifiable aetiology and are categorised as primary or idiopathic frozen shoulder affecting 2% of the population. Those cases of frozen shoulder with an identifiable non-traumatic (OA, rotator cuff tendinopathy, calcific tendinitis) or traumatic (fracture, dislocation, soft tissue injury) shoulder pathology are categorised as having secondary frozen shoulder.¹

Shoulder dislocations affect approximately 1.7% of the population and are most frequently secondary to trauma, with

over 95% being anterior dislocations.² It is known that there is a bimodal age and sex distribution with peak incidence in men aged 20–30 years and in women aged 61–80 years.³ Sequelae of dislocations may include rotator cuff tear or brachial plexus injury. However, recurrent shoulder instability due to a structural injury such as a Hill Sachs, Bankart lesion or distended capsule can be particularly problematic and may require surgical stabilisation.

Following traumatic anterior dislocation of the shoulder, the majority of patients will develop some post-traumatic stiffness that usually resolves on its own. However, an unknown but presumed small percentage of patients may go on to develop secondary frozen shoulder, resulting in pain and stiffness, delaying recovery. This may occur as a

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consequence of a period of shoulder immobilisation although the true precipitating cause is not fully understood. It is believed that this group of patients do not have the same natural history as primary idiopathic frozen shoulder; namely a three phase response of freezing, frozen and thawing which could take several months to resolve.⁴ The pathophysiology of post-traumatic secondary frozen shoulder is believed to be related, but a slightly different entity to primary frozen shoulder. There may be additional stiffness due to extrinsic contracture of the rotator cuff and obliteration of the normal sub-deltoid tissue planes. It may therefore be more recalcitrant to conservative treatment and more likely to require intervention as the chance of spontaneous resolution is less for these types of frozen shoulder.¹

To our knowledge, no studies to date have documented the natural history of patients with secondary frozen shoulder from a previous shoulder dislocation. We were faced with a group of patients who had developed secondary frozen shoulder following an anterior shoulder dislocation and failed conservative treatment by physiotherapy. Although not infrequently seen clinically, the literature provides little guidance on their management.

Manipulation under anaesthetic (MUA) and injection is an established treatment method for frozen shoulder.^{5,6} Having previously found that treatment with an MUA and injection in primary frozen shoulder results in good outcomes and is safe,⁵ we treated this group of patients similarly. There was however concern in this group of patients that the frozen shoulder or contracted capsule may help to provide some stability to the shoulder and that MUA and injection may actually induce recurrent instability of the shoulder joint by releasing the contracted capsule.

The purpose of this study is to present an observational study of a small series of patients with frozen shoulder secondary to anterior dislocation who were treated with MUA and injection.

2. Methods

We performed a retrospective review of a prospectively collected, single-surgeon, consecutive series of MUA for and injection for frozen shoulder in the frozen phase over a thirteen-year period from January 1999 and May 2012. The notes were independently reviewed by two of the authors (WJCT, DAW). Institutional ethical approval was obtained. Frozen shoulder was defined as a history of painful shoulder with resultant restricted elevation and external rotation in the presence of normal radiographs. All patients who presented with symptoms and signs of frozen shoulder following documented traumatic anterior dislocation in the previous six months were included in the study. All cases were first time dislocations that were treated initially with simple reduction under sedation and early mobilisation (within two weeks). All patients had failed initial conservative treatment with physiotherapy and had persistent shoulder stiffness and pain. Our exclusion criteria included previous recurrent dislocations and patients who were unfit for a general anaesthetic. All patients had pre-operative glenohumeral radiographs (antero-posterior and Y-views) taken and were listed for MUA as soon

as the inclusion criteria were met. The time from dislocation was recorded at the time of initial consultation.

The procedure was performed as a day case and involved positioning the patient supine on a trolley in the anaesthetic room. A general anaesthetic was then administered. With one hand stabilising the scapula, the range of glenohumeral motion was recorded. The surgeon's other hand supported the proximal humerus and with a short lever arm to reduce the risk of iatrogenic injury, the shoulder was manipulated sequentially through a range of abduction, forward flexion, external rotation, cross body adduction and internal rotation. The final range of motion (ROM) was recorded and 10 ml 0.5% bupivacaine and 80 mg of depo-medrone were injected into the glenohumeral joint via the direct anterior approach.

All patients were offered a rehabilitation programme that commenced on the day after surgery and were permitted to resume normal activity as soon as possible. They were asked to carry out a self-exercise programme of pendular exercises and wall climbing movements. Patients were then assessed at follow-up by the lead author (DAW). Outcome measures included the Oxford Shoulder Score (OSS)⁷ assessed immediately before surgery, at each follow-up appointment and by postal questionnaire. Also, the ROM of the shoulder before and immediately after manipulation was recorded along with any complications.

Our experience with primary frozen shoulder has been that those who achieved a successful result following MUA and injection reported significant improvement in pain within three to four days and improvement in stiffness within the first three weeks. Therefore, those who had persistent symptoms at the follow-up appointment were considered unlikely to improve further and were offered a further MUA. A postal survey with follow-up telephone consultation was conducted to assess subsequent long-term progress.

3. Results

A total of 468 consecutive patients with a frozen shoulder were referred to the lead author (DAW) during this period and underwent MUA and injection. Out of these, ten patients (five male and five female) met the inclusion criteria to the study with a mean age of 48 (24–68) years old. There were no diabetics. All ten patients had simple anterior dislocations which were reduced in the emergency department with analgesia and sedation. The mean time from dislocation to MUA was 16 (9–28) weeks and the patients were subsequently followed up following MUA at a mean of 38 (15–72) days. There were no intra-operative or immediate complications following the MUA, including dislocations.

The mean improvement in OSS at follow-up following MUA and injection was 18 (range 2–30). All patients improved their score apart from one patient who subsequently re-dislocated before their follow-up appointment. The intra-operative ROM similarly improved by a mean of; 99° (forward flexion), 107° (abduction), 52° (external rotation) and 40° (internal rotation). (Table 1).

Two patients developed recurrent instability symptoms following their MUA and required stabilisation surgery in the form of one open and one arthroscopic anterior repair.

Table 1 – Range of motion (ROM) data.

Case	Pre-MUA ROM (Degrees)				Post-MUA ROM (Degrees)				Improvement in ROM (Degrees)			
	Forward flexion	Abduction	External rotation	Internal rotation	Forward flexion	Abduction	External rotation	Internal rotation	Forward flexion	Abduction	External rotation	Internal rotation
1	120	80	10	10	180	180	70	70	60	100	60	60
2	90	90	30	30	180	180	60	60	90	90	30	30
3	70	70	20	20	140	140	50	50	70	70	30	30
4	70	70	20	20	180	180	70	70	110	110	50	50
5	110	90	45	20	180	140	90	60	70	50	45	40
6	50	45	10	20	180	180	70	70	130	135	60	50
7	10	30	-10	40	170	160	70	70	130	130	80	30
8	40	30	0	20	180	180	70	60	140	150	70	50
9	80	45	15	40	180	180	70	60	100	135	55	30
10	90	80	30	30	180	180	70	70	90	100	40	60
Mean	76	63	17	25	175	170	69	65	99	107	52	40

Long-term scores for these patients were thus invalid as the scores were not related to the initial MUA. Out of the seven remaining patients, three patients required a revision MUA and injection at a mean of 10 (8–12) weeks but long-term scores were collated for all seven patients at a mean of 93 (6–413) weeks with all patients scoring a higher OSS than their pre-op OSS. (Table 2).

4. Discussion

We present a small observational study on the management of frozen shoulder secondary to previous anterior dislocation with MUA and injection. In practice, stiffness following anterior dislocation is common and the initial treatment of choice is almost always conservative with physiotherapy, with the majority resolving on its own accord. Despite this, there remains a small percentage of patients who have persistent pain and restricted movement that go on to develop secondary frozen shoulder. Over a thirteen-year period, we only encountered ten patients, which perhaps represents the very small cohort of patients that develop this difficult but nevertheless clinically important condition. Unfortunately, it remains not fully understood and this interesting subgroup of patients has had little attention in the literature to date to provide guidance on the natural history of their condition or their management. Having previously shown that treatment with MUA and injection in primary frozen shoulder results in good outcomes and is safe,⁵ we cautiously treated this cohort of patients similarly.

Our results demonstrate that the majority of our patients eventually went on to have comparable improvements in both OSS and ROM. However, three patients (30%) required revision MUAs within 12 weeks of their primary procedure. Our experience of recurrence in primary frozen shoulder is up to 19%⁵ whereas recurrence following arthroscopic release has been reported to be between 6 and 11%.^{8,9} Our results may be considered a failure of the initial treatment or it may represent the natural history of the condition, suggesting a higher recurrence rate in this group.

Although similar, the capsular pathology in both primary and secondary frozen shoulder is not believed to be the same. In the more common idiopathic primary frozen shoulder, it is characterised by an inflammatory contracture of the glenohumeral synovial capsule as well as other extra-articular soft-tissue structures such as the coracohumeral ligament, soft tissues of the rotator interval, the subscapularis muscle, and the subacromial bursae as a result of hyperplastic fibroplasia and excessive type III collagen secretion.¹⁰ This reduces the available intra-articular volume thus limiting glenohumeral movement. Although generally considered self-limiting, it can lead to long-term disability and pain.¹¹ The pathology of secondary frozen shoulder remains poorly understood but there may be additional rotator cuff and soft tissue contracture leading to a poorer prognosis.¹

All three patients had good initial improvement in OSS and ROM at their initial follow-up. In our experience with primary frozen shoulder, those patients with improvement in OSS and ROM at initial follow-up usually represents a successful primary procedure. Although we acknowledge that the

Table 2 – Summary of patients.

Case	Gender	Age at MUA (years)	Dislocation to MUA (weeks)	Pre-MUA OSS	Post-MUA OSS	Long-term OSS	Long-term F/U (weeks)	Outcomes
1	M	43	15	14	44	42	76	Required further MUA within 12 weeks but had initial good outcome and long-term outcome
2	F	68	10	23	43	27	63	
3	F	31	13	27	46	48	413	
4	M	24	16	20	37	n/a	n/a	Recurrent instability and required stabilization (16/7/09 MUA, Dislocation 12/3/11)
5	F	50	28		23	n/a	n/a	Recurrent instability and required stabilization (11/5/10 MUA, Dislocation 8/6/10)
6	M	59	9	25	44	48	99	Required further MUA within 10 weeks but had initial good outcome and long-term outcome
7	F	51	17	24	39	42	54	
8	M	52	14	13	35	39	12	Required further MUA within 8 weeks but had initial good outcome and long-term outcome
9	F	45	14	38	40	44	6	
10	M	52	21	30	48	48	22	
Mean		48	16	24	42	42	93	

pathology may be slightly different in secondary frozen shoulder, this may suggest that this was an early recurrence of their frozen shoulder rather than insufficient release in the primary procedure. Interestingly these three patients were also the oldest patients in our study with a mean age of 60 years. Poorer soft-tissue and bone quality in these older patients or perhaps compliance with physiotherapy may make them more prone to recurrence. Alternatively, post-traumatic or secondary frozen shoulder may generally be more recalcitrant due to a greater amount of adhesions and contracture and perhaps therefore may be less successful to MUA when compared to primary frozen shoulder and more aggressive management should be instituted in the first place as the chance of spontaneous resolution is less for these types of frozen shoulder. Our recurrence rate may simply reflect the natural history of this more resistant frozen shoulder and without a greater number of patients, this cannot be confirmed. However, even with small numbers it suggests that primary and secondary frozen shoulders behave differently to MUA & injection.

Two patients unfortunately developed instability symptoms following MUA and injection, requiring surgery. 20% of patients developing instability is clearly a significant proportion with an unsatisfactory result. The risk of re-dislocation of the shoulder following first-time dislocation peaks in adolescents with an incidence of approximately 75%.¹² This risk reduces with age and in those younger than 30 years old it has been reported as up to 32%¹³ whilst in the older group it can be up to 20%.¹⁴ Recurrent instability following a dislocation can be a consequence of a structural abnormality due to the significant damage that can occur to the joint when the shoulder is dislocated. Along with bony lesions, soft tissue structures that lend stability to the shoulder joint including the labrum, joint capsule and rotator cuff may tear. Bankart lesions¹⁵ are commonly seen at arthroscopy in patients with recurrent instability as well as capsular laxity. Hovelius¹² reported that 54% of anterior dislocations were associated with a Hill Sachs lesion. Between 14 and 63% are also associated with rotator cuff tears with the incidence particularly higher in older patients.¹⁶

Manipulation of the shoulder releases the synovial capsule. Reservations exist with regards to MUA due to the potential risk of uncontrolled articular and periarticular damage. Reported complications following MUA include fractures, brachial plexus injury, rotator cuff tears and glenohumeral dislocation. The arthroscopic findings of soft tissue trauma immediately following an MUA in primary frozen shoulders has been described by Loew.¹⁷ There is uncertainty in the role of the MUA in secondary frozen shoulder. In the younger patient, the manipulation may tear healing structures such as labral tears and capsule thus increasing the risk of recurrence. Equally, in the more elderly population with a known higher risk of associated rotator cuff tears, is manipulation a safe option without knowing the status of the cuff prior to the procedure?

Interestingly, the intra-operative findings of the two patients who developed instability showed no evidence of a rotator cuff tear, bony Bankart lesion or capsular laxity that could have been attributed to the MUA. Both had moderate Hill-Sachs lesions which we have attributed to the initial

dislocation. In these two patients, if the frozen shoulder was treated without manipulation and left to resolve as the natural history of frozen shoulder has been described,⁴ would the shoulder have become unstable irrespective of the MUA? Given that the risk of re-dislocation following a first-time dislocation in the older patient has been described as between 20%¹⁴ and 32%,¹³ it is not unreasonable to assume that some of the patients in this study would have gone on to develop instability irrespective of their manipulation. A re-dislocation rate of 20% would then be comparable to the published rates.

Looking at the two instability patients in further detail, the male patient was the youngest in the study at 20 years old, perhaps reflecting the higher likelihood of progression to re-dislocation irrespective of any intervention. The other patient was older but had the longest time interval between dislocation to MUA at 28 weeks. In this case, the shoulder may have been resolving on its own accord and the ROM data reflects this, having the best pre-op ROM in the series. The MUA may therefore have accelerated the natural history of the frozen shoulder and induced instability earlier than without intervention.

With the cohort of patients examined in this study, it is therefore very difficult to differentiate pre-existing structural damage as a consequence of the original injury from any damage that may have occurred secondarily to the MUA or extended by the MUA, without arthroscoping or imaging the shoulder before and post MUA. Although all patients had simple pre-operative radiographs that excluded any gross structural defects that would predispose the shoulder to instability, an axial view was not taken to look specifically for a Hill-Sachs lesion. In retrospect, all patients prior to MUA should have had a CT or MRI to look at the shoulder in greater detail to help identify pathologies that may correlate as being a risk factor for re-dislocation. Further imaging would have also helped to ascertain whether the study group had other pathologies that may have had significant bearing on recurrence of frozen shoulder. This would help definitively quantify and either lend further support or challenge the role of MUA in secondary frozen shoulder following anterior dislocation.

There are no comparable studies of other treatments including hydrodilatation, arthroscopic and open release following frozen shoulder secondary to dislocation to compare our outcomes. Dislocation following arthroscopic release and manipulation procedure for frozen shoulders has been described but none of these cases were frozen shoulders secondary to dislocation.^{18,19} We can only hypothesise with the small amount of data that we have and it is therefore very difficult to determine whether the instability of the shoulder is as a consequence of excessive release of the soft tissue structures following MUA or the normal progression of the dislocated shoulder. Developing a frozen shoulder secondary to dislocation may provide the shoulder with some initial stability following the injury. Therefore, by treating this group of patients with an MUA, we may actually induce instability in a proportion of patients who may have developed instability anyway, by merely shortening the natural history of the condition.

We accept that there are limitations to our study and that it is too small for meaningful statistical analysis. However, we believe that ten out of an original 468 patients over a 13-year

period who fulfilled our inclusion criteria represents a fair study population for this condition. We do not know the natural history and why presumably the majority of patients do not go on to develop frozen shoulder following anterior dislocation. The findings in our study may therefore be clinically important and a larger study number with a comparative cohort of patients treated non-operatively would clearly further enhance our knowledge on this topic and inform our treatment of this clinical problem.

5. Conclusion

Secondary frozen shoulder may be more recalcitrant to conventional conservative treatment. Some risk of recurrent instability is part of the natural history of anterior shoulder dislocation. However, it is feasible that by performing an MUA to maximise mobility, stability may be sacrificed in some patients. Given this, we believe that MUA and injection is an optimal procedure for primary frozen shoulder but should be performed with caution in those with post-traumatic dislocation of the shoulder. Although this is a small single surgeon series, we believe it is still a useful study for this important topic and hope that it provides some insight and discussion for practicing surgeons faced with the same problem.

Conflicts of interest

All authors have none to declare.

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