EFORT open reviews

Indications for hydrodilatation for frozen shoulder

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- Frozen shoulder causes significant functional disability and pain in a population group constituted by patients who are often middle-aged and working.

- Frozen shoulder remains poorly understood. The available literature is limited and often prone to bias.

- A rapid, non-surgical and cost-effective treatment that reduces pain and restores function is an attractive option.

- Hydrodilatation is a potential first-line treatment of frozen shoulder in secondary care.

All aspects of frozen shoulder have attracted controversy and debate, reflecting our lack of a detailed understanding of this disease.

Hydrodilatation has emerged as a potential non-surgical option in the management of frozen shoulder. However, its role has yet to be fully clarified. There are unanswered questions relating to its use in diabetic patients. The role of hydrodilatation compared with more established treatments for frozen shoulder remains undefined.

The frozen shoulder

In 1934 Codman[1] described the clinical picture of frozen shoulder with an insidious onset of lateral shoulder pain with restriction in active and passive movement. Codman acknowledged the challenge in definition and treatment of the disease, which still remains today.[2,3]

Frozen shoulder classically occurs in female patients in their fourth to sixth decade of life and can be classified according to its presumed aetiology into primary idiopathic or secondary. Secondary frozen shoulder can be further categorised into that from intrinsic causes such as rotator cuff pathology, extrinsic causes such as humeral fractures, or systemic causes such as diabetes and thyroid dysfunction. The incidence of frozen shoulder in the background population is up to 5%, but in diabetics can be up to 20%.[4]

A recent study has supported a potential link between *Propionibacterium* (*P.*) *acnes* and frozen shoulder as a possible infective aetiology of the disease.[5] In this study ten patients undergoing arthroscopic arthrolysis underwent tissue biopsies, of which 60% were positive for *P. acnes.* This led the authors to question whether '*P. acnes* could be the Helicobacter of frozen shoulder'.[5]

The patho-aetiology of frozen shoulder is, however, complex and multifactorial with both genetic and environmental factors playing an important role.[6]

Frozen shoulder progresses in a cyclical manner, beginning with a painful 'freezing' phase which is characterised by hypervascularity seen during arthroscopy. The freezing (stiffening) stage lasts about three months. The disease then progresses to a painless 'frozen' phase which can last up to nine months, where marked restriction of movement is the predominant feature. Finally the disease progresses to a 'thawing' phase with remodelling, and patients begin to report an improvement in range of movement. This can last up to 18 months. The latter stages demonstrate significant capsular thickening with contraction which can be demonstrated clinically with loss of external rotation visualised during MRI or arthroscopy. The stage of the disease process that the patient is in may influence treatment, with steroid therapy preferred in the freezing phase whilst arthroscopic arthrolysis may be preferred in the frozen phase.

Historically patients were reassured that a complete recovery was inevitable; however, the evidence to support this is variable. In 1978, Grey[7] reported on 24 of 25 patients who improved with conservative treatment or observation over a two-year period. Another study with an average of nine-year follow-up data for patients with frozen shoulder reported 94% rate of spontaneous complete recovery without surgery.[8] However, no other papers have shown such successful outcomes. In other literature the rates of complete recovery have been reported to be as low as 39% and 50%.[9,10] There is a group

of patients who experience ongoing pain and disability and for whom complete spontaneous resolution cannot be guaranteed. This is especially the case in those with systemic secondary causes such as diabetes.[11] In addition, waiting for spontaneous recovery may not be deemed practical or acceptable as a management option to some patients, such as self-employed individuals.

Frozen shoulder typically affects the rotator interval and begins with thickening of the coracohumeral ligament.[2] This manifests itself as early loss of external rotation, which is a classical sign. As the disease progresses, there is contraction of the glenohumeral capsule and thickening of the glenohumeral ligaments, with decrease in soft-tissue compliance.[12] Such functional restriction is important for the differential diagnosis of shoulder pain. In the early stages of the disease, patients will report constant severe pain that is typically unguarded and has a strong night-time component, which may prevent them from lying on the affected side. Nocturnal variation and sleep disturbance is one of Codman's 12 original diagnostic criteria.[1] As the disease progresses, the pain typically resolves but disabling restriction of movement persists. The diagnosis is made clinically but radiographs must be obtained to differentiate frozen shoulder from alternative causes which lead to a restriction in external rotation, such as osteoarthritis or posterior dislocation. Case reports of misdiagnosed shoulder tumours mimicking the frozen shoulder are reported in the literature.[13] MRI scans of patients with frozen shoulder will show thickening of the coracohumeral ligament and capsule at the rotator interval. The evidence suggests the extent of these MRI findings may correlate to the stage of disease, with synovial thickening predominantly seen earlier in the disease and capsular thickening predominantly in the later stages.[14] Characteristic MRI features are illustrated in Figures 1 and 2.

Fig. 1 Coronal T2 fat suppressed. Thickening of the inferior joint capsule (7 mm) and some pericapsular oedema (arrow).

Histological analysis from an intra-operative biopsy study demonstrated that the underlying pathological process was one similar to that of Dupuytren's disease of the hand with fibroblastic proliferation and no evidence of inflammation.[15] Patients with Dupuytren's disease are reported to be eight times more likely to develop a frozen shoulder.[16] Subsequent work has demonstrated features of chronic inflammation and used this to explain the severe pain experienced by patients and increased vascularity seen intra-operatively in patients with early disease.[17]

The diabetic frozen shoulder

The diabetic frozen shoulder merits particular attention due to its disease profile, tending to be more severe in presentation and with inferior outcomes despite treatment.[11] The underlying mechanism of frozen shoulder in

Fig. 2 Sagittal Proton density (left) and Sagittal Proton density fat suppressed (Pd Fs) (right). There is effacement of fat in the rotator interval on the Sagittal Pd and thickening of the Superior glenohumeral ligament and Coracohumeral ligament and oedema on the Sagittal Pd FS (arrows).

the diabetic patient is presumed to be due to collagen cross-linking mediated by hyperglycaemia, which then results in loss of tissue compliance.[18] The clinical effect of this is limited joint mobility.[19]

Frozen shoulder is reported to be almost twice as common in insulin-dependent diabetics compared with non-insulin-dependent diabetics.[20] The same study showed that non-insulin-dependent diabetics on oral therapy are 1.5 times more likely to be affected by the disease than non-insulin-dependent diabetics on diet control. This study reported an increased association of frozen shoulder with prolonged duration of diabetic disease, i.e. more than ten years. They suggested that this may explain the increased incidence of frozen shoulder seen in insulin-dependent diabetics. Another study found a statistically significant difference in rates of frozen shoulder between 800 diabetic and 600 non-diabetic control patients, with 10.8% rate in the diabetic group compared with 2.3% in the control group.[21] A higher proportion of the diabetic patients with frozen shoulder were insulin-dependent (36% *versus* 23%).

The literature supports inferior outcomes in the management of an insulin-dependent population, whether that be conservative or surgical.[11] Thus, conservative treatment of the diabetic frozen shoulder is associated with inferior outcomes when compared with the non-diabetic population.

Current treatment options

Injections and physiotherapy

Initially frozen shoulder was predominantly thought to be an inflammatory condition, hence initial management has been with intra-articular injection of steroids and local anaesthetic into the glenohumeral joint and physiotherapy as first-line treatment. A randomised trial demonstrated superior outcomes of isolated treatment with injections over physiotherapy in primary frozen shoulder, with faster relief of symptoms in the injection group.[22] However, it has also been demonstrated that there is a statistically significant benefit with the addition of physiotherapy after a glenohumeral joint injection.[23] Despite the number of studies supporting glenohumeral injections in the management of frozen shoulder, significant heterogeneity in patient groups affects the conclusions that can be drawn. There is evidence of short-term benefits of oral steroids in the management of frozen shoulder but the benefits lasted less than six weeks.[24] Other authors have demonstrated good outcomes in the early stages of frozen shoulder with oral steroids combined with neuropathic agents and a home exercise programme.[25]

Manipulation under anaesthesia (MUA)

The intention of manipulation of the frozen shoulder under anaesthesia is to forcibly rupture the contracted capsule. MUA is often combined with intra-articular steroid injection to minimise the secondary inflammatory response in order to permit subsequent rehabilitation. However, the literature has called into question the benefit of intra-articular steroid injection after the procedure.[26] Unique complications of MUA include fracture (glenoid, proximal humerus, clavicle) and brachial plexus injury.[27] MUA may be performed in isolation or as an adjunct to arthroscopic arthrolysis. Short-term results within one year have been reported to be superior with combined MUA and arthroscopic arthrolysis compared with MUA alone, although after one year there was no difference.[28] The literature supports MUA as a treatment to accelerate recovery in frozen shoulder,[29] but historically the literature has failed to support MUA as a treatment in diabetic patients, with poor short-term outcomes reported.[30] However, more recent studies have demonstrated similar short- and long-term outcomes between non-diabetics and diabetic patients. These conclusions can only be made for non-insulin-dependent diabetics though, since insulin-dependent diabetics were excluded.[31] A repeated procedure was necessary in 36% of diabetic patients.[32]

Arthroscopic arthrolysis (capsular release)

Although various surgical techniques are available, a release of the anterior capsule and clearance of the rotator interval to include the superior and middle glenohumeral ligaments and the coracohumeral ligament is invariably performed in all reported studies. Variations on the technique include a spectrum of further releases, with some surgeons performing a full 360° release.[33] As discussed previously some authors have combined arthroscopic arthrolysis with MUA as a result of the evidence from Sivardeen and colleagues demonstrating superior short-term outcomes.[28] Literature comparing arthroscopic arthrolysis with MUA is lacking, suggesting the need for a high quality study to evaluate and compare these two main surgical options.[34] Despite this, arthroscopic arthrolysis remains the preferred surgical option when managing frozen shoulder.[35] It is anticipated that the currently recruiting multi-centre randomised United Kingdom Frozen Shoulder Trial (UK FROST), which is comparing structured physiotherapy *versus* manipulation under anaesthesia *versus* arthroscopic capsular release, will serve to further contribute to the literature base and answer some of the uncertainties regarding optimal management of the frozen shoulder.[36] Previous literature has shown the results of arthroscopic arthrolysis in diabetic patients to be inferior to those in non-diabetics.[37]

Hydrodilatation (distension arthrography)

Hydrodilatation is a non-surgical radiological intervention used in the management of frozen shoulder. Although therapeutic regimens will differ between units, common

Table 1. Summary of the literature relating to hydrodilatation

	Final number of patients	Regimen	Capsular rupture achieved	Follow-up	Outcome measure	Conclusion
Watson, 2007 (hydro)	41	40 mg triamcinolone, 10 ml 0.5% bupivacaine. Varying saline	Not documented	2 years	SPADI, SDI	Benefit maintained up to 2 years post-procedure
Clement, 2013 (hydro)	39	40 mg or 80 mg triamcinolone, 10 ml 1% lidocaine. 40 ml saline	Not documented	14 months	OSS	55%/63% of patients had near-normal shoulder function at 1 month/final follow-up
Bell, 2003 (hydro)	106	4 mg betamethasone, 2 ml 2% lidocaine. Varying saline	Yes	3 years	Visual analogue, clinical ROM	66% pain-free at 2 months. ROM improved 20–40°at 2 months. Worse in diabetics
Tveita, 2008 (hydro *versus* injection)	39 (hydro) 37 (injection)	20 mg triamcinolone, 4 ml 0.5% bupivacaine and 10 ml saline	Yes	6 weeks	SPADI, clinical ROM	No difference between two groups
Quraishi, 2007 (hydro *versus* MUA)	19 (hydro) 17 (MUA)	30 mg triamcinolone, 2% lidocaine. Varying saline	Yes	6 months	Visual analogue, constant scores	Patient satisfaction superior in hydro group (94% to 81%). No difference in constant scores
Trehan, 2010 (single hydro *versus* repeated hydro)	22	80 mg triamcinolone, 10 ml 0.5% bupivacaine. 25 ml saline	Not documented	15 months	OSS, SDQ-UK	No difference between single procedure *versus* repeated

Notes. SPADI - Shoulder Pain And Disability Index. SDI - Shoulder Disability Index. OSS – Oxford Shoulder Score. ROM – range of movement. Hydro – hydrodilatation. SDQ-UK – Shoulder Disability Questionnaire UK Score

to most is the instillation of a large volume of saline containing steroid, local anaesthetic and contrast material into the glenohumeral joint under imaging guidance, typically around 30 ml. The stated benefits of the procedure are achieved through hydraulic distension of the capsule and the main purpose of initial work was to achieve capsular rupture.[38] However, there is little in the way of evidence to determine whether capsule rupture must be achieved in order for the procedure to be successful, or whether it is the capsular distension which is most important. Most studies comment on their intention to achieve capsular rupture but have not investigated this.

The role of hydrodilatation

A Cochrane review in 2008 demonstrated only silver-level evidence to support hydrodilatation as a treatment modality to improve short-term pain and function.[39] This short-term benefit was only maintained up to three months. Data from five trials were included, although only one of these trials was low risk from bias. This highlights the deficiency in high-quality robust research available on hydrodilatation. The available literature is summarised in Table 1.

Currently the longest follow-up study investigating the outcomes of hydrodilatation is a two-year follow-up study on 41 patients undergoing hydrodilatation with an almost even distribution of patients with primary and secondary frozen shoulder.[40] The technique involved a mixture of steroid (40 mg triamcinolone) and local anaesthetic (10 ml 0.5% bupivacaine), and varying volumes of saline to achieve full distension or rupture. Capsular rupture was recorded but the results were not published

in the study. Diabetic patients were excluded. Primary outcomes included Shoulder Pain And Disability Index (SPADI) and Shoulder Disability Index (SPI) scoring.[41] No control group was used in the study but the authors considered (for non-specified reasons) that due to the natural history of the disease, if a control group had been used then it would likely have shown similar outcomes to the hydrodilatation group at two years due to tendency for spontaneous improvement of the disease. The greatest magnitude of improvement was between three days and one week in terms of SPI scoring and between pre-treatment and three months in terms of SPADI scoring. The authors commented that most subjects demonstrated some functional deficit at final follow-up but referenced further literature to support a significant variation in the definition of 'normal shoulder function' in patients aged about 40 years.[42]

A study by Clement et al[43] provided post-hydrodilatation outcomes with a mean follow-up of 14 months. The authors included 53 procedures in 51 patients, 12 of whom were diabetic. The technique involved a mixture of steroid (40 mg or 80 mg triamcinolone depending on whether they were diabetic or not) and local anaesthetic (10 ml 1% lidocaine) with 40 ml saline. Capsular rupture was not documented. At one month post-procedure 55% of patients gained normal or near-normal shoulder function, and this result was maintained at final follow-up, with 63% of patients gaining normal or near-normal shoulder function as assessed by the Oxford Shoulder Score.[44] One patient developed septic arthritis after hydrodilatation. Although they demonstrated similar outcomes in diabetic patients, the authors acknowledged the small sample as a potential bias in the results.

The study involving the largest number of patients reported on 109 frozen shoulders which were managed with hydrodilatation in 106 patients over a three-year period.[45] In all, 15 patients included were diabetic (insulin status not stated). Their technique involved a mixture of steroid (4 mg betamethasone) and local anaesthetic (2 ml of 2% lidocaine), and varying volumes of saline required to achieve capsular rupture (between 10 ml and 55 ml). Patients were reviewed at two and four months. The primary outcome measures were pain scores on a visual analogue scale and clinical assessment of range of movement. No scoring systems were used. At two months, patients had a mean improvement of 30° of external rotation, 25° of abduction and 40° of elevation. Outcomes in diabetic patients showed a mean improvement of 30° of external rotation, 20° of abduction and 30° of elevation. Patients with severe loss of external rotation (< 15°) achieved the greatest improvement in range of movement, although still tended to have inferior ranges when compared with the patients without such profound external rotation loss. Patients with prolonged disease (> 12 months) achieved similar improvements when compared with those with a disease history of less than one year. On assessment of pain scoring, most patients reported their pain as moderate (47%) prior to the procedure and nil (66%) at two months. In the diabetic group, most patients reported their pain as moderate (67%) prior to the procedure and mild or nil (33% each) at two months. No further statistical analysis was performed. There were 29 repeat hydrodilatation procedures performed (22 non-diabetics, seven diabetics). Of these, five non-diabetics (23%) and two diabetics (29%) did not improve with the repeat hydrodilatation and went on to have arthroscopic arthrolysis, representing a failure rate of 5.5% in the original non-diabetic group and 13% in the original diabetic group (23% *versus* 29% failure rate after repeat procedure). The study stated that inferior outcomes of frozen shoulder in the diabetic patient generally appears to be seen also when the diabetic patient undergoes hydrodilatation.

There is currently no evidence in the literature to support superiority of surgical treatment for frozen shoulder over conservative treatment, but this may be due to small numbers of patients in small numbers of trials.[4] Comparative studies between hydrodilatation and other treatment modalities are also lacking. Tveitå et al[46] compared the outcomes of patients randomised to undergo hydrodilatation (20 mg triamcinolone with 4 ml 0.5% bupivacaine and 10 ml saline) with those randomised to fortnightly image-guided steroid injections (20 mg triamcinolone with 4 ml 0.5% bupivacaine) for six weeks. Diabetic patients were excluded. Capsular rupture was achieved in all but one patient in the hydrodilatation group. There were 39 patients in the hydrodilatation group and 37 in the injection group, and follow-up assessment was at six weeks from the last intervention. Patients were not allocated to physiotherapy but the study acknowledged that some patients were already enrolled in a physiotherapy regimen, a potential source of bias. Our standard practice is to begin physiotherapy within one week of hydrodilatation.

Quraishi et al[47] randomised 17 patients to MUA and 19 patients to hydrodilatation; there were three insulin-dependent diabetics in each group. The technique used was the injection of steroid (30 mg triamcinolone) and local anaesthetic (2% lidocaine) mixture with varying volumes of saline required to rupture the capsule. The study demonstrated hydrodilatation as a successful treatment in the management of frozen shoulder with the success rates based upon patient satisfaction superior to manipulation under anaesthetic: 94% *versus* 81% at six-month final follow-up. Constant scores demonstrated a statistically significant improvement in both groups, but without a statistical significance between the two. They concluded that hydrodilatation attracted an added cost but a health benefit of avoiding a general anaesthetic, as well as reduced risk of surgical morbidity such as fracture or cuff injury.

There is no current literature comparing hydrodilatation with arthroscopic arthrolysis. However, there is evidence to suggest how many patients go on to require arthroscopic arthrolysis after suboptimal outcomes post-hydrodilatation.[42]

Target hydrodilatation to the rotator interval has also been reported in the literature.[48] This variation on the traditional technique has been investigated by one study using the novel approach in 22 patients, in whom they injected 21 ml of steroid (40 mg triamcinolone) and local anaesthetic mixture (10 ml 0.5% bupivacaine and 10 ml 1% lidocaine). Capsular rupture was not documented. Three patients were diabetic. Patients were followed up for four months, and at this time a statistically significant improvement in Oxford Shoulder Score compared with pre-operatively was reported from 13.6 to 36.5.

In our institution, patients who have achieved an improved response after hydrodilatation in terms of pain or function but still report ongoing deficit are offered a second hydrodilatation procedure. However, this is not standard practice and tends to be reserved for those who have initially responded well but have not fully benefited from the procedure. Trehan et al[49] reported on 22 patients who underwent repeat hydrodilatation at six weeks. Their technique used the injection of steroid (80 mg triamcinolone) and local anaesthetic (10 ml 0.5% bupivacaine) mixture with 25 ml of saline. Capsular rupture during the procedure was not documented, neither was the patients' diabetic status. They found that there was no statistically significant difference in the final Oxford Shoulder Score for patients undergoing single or repeat treatments.

Proposed mechanism of action of hydrodilatation

The mechanism of action of hydrodilatation is hotly debated. Most clinicians intuitively suggest that capsular rupture contributes to a mechanical resolution of the shoulder stiffness. However, it is well recognised that lack of capsular rupture during a hydrodilatation is not associated directly with failure of resolution of symptoms. Therefore, it is unclear whether the dilatation or slow capsular deformation aspect of the treatment is the key element rather than the capsular rupture. There is certainly evidence from studies examining the outcome of MUA to guide us.[50] There seems to be no relationship between the clinician not feeling the tearing sensation during a MUA and the patient's final outcome. In other words, whether or not the clinician feels the rupture of the capsule, the chance of recovery is the same. Therefore it is likely that there are intrinsic and extrinsic factors contributing to the resolution of frozen shoulder after treatment with hydrodilatation. One possible intrinsic mechanism is that the increased glycosaminoglycan concentration seen in the joint capsule in frozen shoulder promotes myofibroblast activity and this is reversed by the joint distension.[51] We do not yet fully understand the contribution of the dilatation, steroid or the local anaesthetic to the successful outcome of hydrodilatation, therefore more research needs to be conducted to answer these remaining important questions.

Conclusion

Many cases of frozen shoulder are mild and will resolve with analgesics and physiotherapy. However, for patients who are not improving or in whom watchful waiting is not practical, hydrodilatation can be supported for short-term management. Diabetic patients must be counselled about the anticipated inferior outcomes when compared with the non-diabetic population. There is a role for hydrodilatation as a repeat procedure in patients with incomplete recovery but not as a standardised treatment plan.

This review highlights the need for high-quality controlled studies ensuring rigorous study design incorporating: (1) aetiology of the disease to differentiate between primary and secondary (extrinsic, intrinsic and systemic); (2) diabetic treatment regimen; (3) validated scoring systems and; (4) robust statistical analysis.

Currently unanswered questions regarding the technical aspect of hydrodilatation include: (1) the role of capsular rupture and whether this is essential for a successful outcome; (2) the optimal hydrodilatation regimen in terms of volume and individual components and; (3) whether steroid is a key aspect in this regimen.

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