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Extracorporeal shockwave therapy (ESWT) for refractory Achilles tendinopathy: A prospective audit with 2-year follow up

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HIGHLIGHTS

- ESWT appears to improve pain and function in patients with refractory Achilles tendinopathy.
- ESWT should be considered when other conservative measures have failed.
- ESWT should be used with ongoing access to other conservative treatments.

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ABSTRACT

Background: Achilles tendinopathy (AT) represents a triad of tendon pain, swelling and impaired performance. Extracorporeal shockwave therapy (ESWT) has been endorsed by the National Institute for Health and Care Excellence (NICE) for refractory AT. This audit investigates the long-term outcomes of patients treated with ESWT for refractory AT.
Methods: Forty-six patients treated with ESWT for AT between October 2010 and August 2011 completed visual analogue, satisfaction scores and functional assessment questionnaires over two years. Patients were subdivided into two groups depending on whether their AT was insertional (IAT) or non-insertional (NAT).
Results: Forty-six patients (mean age 58 years) completed all treatments and full 2 year follow up. There was significant improvement in pain at rest, on activity and of function within both NAT and IAT groups over the two-year period. Satisfaction scores were significant in the NAT group but not in the IAT group.
Conclusions: ESWT appears to be of benefit in the long term improvement of pain at rest, on activity and functional outcome in patients with refractory AT. However, subjective patient opinion may not match the perceived clinical outcome observed in this audit in all patients and individuals should be counselled regarding this prior to treatment.

1. Background

Achilles tendinopathy (AT) represents a triad of tendon pain, swelling and impaired performance [1] and can be divided into insertional (IAT) and non-insertional tendinopathy (NAT). IAT affects the point of insertion of the TA at the calcaneus. NAT typically affects the mid-substance of the TA 2–7 cm above the tendon's calcaneal insertion.

Management strategies and novel therapies for AT are myriad. The mainstay of treatment is conservative, involving physiotherapy with eccentric loading exercises, which have been shown to be effective for both IAT and NAT [2,3]. Other therapies such as injections of platelet-rich plasma and autologous blood products have been trialled with variable results [4–6]. The use of corticosteroids carries potential morbidity in terms of tendon rupture and again the efficacy of the procedure is variable [7,8]. Surgeries involving percutaneous tenotomy, TA debridement and/or calcaneal osteotomy tend to be reserved for cases where conservative measures have failed [9,10].

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Extracorporeal shockwave therapy (ESWT) is a conservative treatment which has been shown to be of benefit to patients with AT and other recalcitrant musculoskeletal conditions [11]. As a result the procedure has been recommended by the National Institute for Health and Care Excellence (NICE) [12].

Shockwaves in ESWT may be generated using electrohydraulic, electromagnetic or piezoelectric principles [13] and either be focussed or emitted as a radial pressure wave for deep and superficial applications respectively [14]. Therapeutic effects can be obtained with shockwave energies ranging from low (<0.08 mJ/mm^2), medium (<0.28 mJ/mm^2) and high (>0.60 mJ/mm^2) [15]. In the context of the management of musculoskeletal complaints, energies in the low and medium ranges are generally employed. This enables the procedure to be performed without local anaesthetic [6].

The aim of this audit was to assess the long term response of a cohort of patients undergoing ESWT for refractory AT as per the guidance issued by NICE [12].

2. Methods

All patients with history, clinical and ultrasonographic features suggestive of refractory AT (>3 months duration) identified between October 2010 and August 2011 and to be treated with ESWT were approached for inclusion in the audit. All patients had initially failed treatment by conservative means with eccentric exercises, heel inserts and/or non-steroidal anti-inflammatory drugs (NSAIDs) for the period prior to commencement of ESWT.

Patients were asked to complete a questionnaire prior to their first session of ESWT which assessed visual analogue pain scores (VAS) at rest and on activity and the Victoria Institute of Sport Assessment–Achilles (VISA-A) questionnaire to assess functional disability [16]. The VISA-A questionnaire assesses a number of lower limb activities via a number of questions with a maximum achievable score is 100.

Treatments were performed using the Spectrum Technology Swiss Dolorclast Classic ESWT machine (EMS Electro Medical Systems, Nyon, Switzerland), which produces radial shockwaves by propelling a projectile at high speed against a second interface with compressed air. Three sessions of low energy radial ESWT (using 2500 pulses per treatment) were administered at weekly intervals. Frequency and pressure ranged from 10 Hz and 1.5 Bar respectively for the first 500 pulses increasing to a pressure of 2.5 Bar for the remaining 2000 pulses, largely dictated by patient comfort. VAS and VISA-A scores were subsequently obtained at six and 16 weeks and at 2 years post treatment. Likert satisfaction scores (ranging from 1 – completely recovered to 6 – worse) were also obtained at these follow up points. 56 patients completed the scores at baseline. 51 patients completed follow-up to 16 weeks. A further five patients were lost to follow-up at 2 years, resulting in 46 patients completing all treatments and scores at 6 weeks, 16 weeks and 2 years (17.8% loss to follow up at 2 years).

3. Results

Of the 56 patients taking part in the audit, 36 had a diagnosis of NAT and 20 of IAT. Forty-six patients (34 NAT, 12 IAT) completed two year follow up. There was a slightly higher male representation in both groups with 19 and 8 men in the NAT and IAT groups respectively. The mean length of symptoms was 20 (4–252) months in the NAT group and 42 months in the IAT group. Mean age of the cohorts was 54 (38–80) in both groups.

Table 1
Mean values of VISA-A scores.

	NAT	IAT
Baseline	40	43
6 weeks	50	51
16 weeks	55	58
2 years	66	70

Table 2
Likert satisfaction score.

Level of improvement	
Completely recovered	1
Much improved	2
Somewhat improved	3
Hardly improved	4
Not improved	5
Worse	6

Table 3
Mean Likert scores.

	NAT	IAT
6 weeks	3	3.2
16 weeks	2.7	3.1
2 years	2.2	2.7

3.1. Visual analogue scale scores

The Visual Analogue Score (VAS) was measured at rest (Chart 1) and on activity (Chart 2). A score from 0 to 10 was recorded using a whole number. Separate values were calculated for insertional and non-insertional AT to assess if there was any difference between the groups.

In the NAT group, there was improved pain at rest from a mean of 3.6 (0–8) to 2.4 (0–7) at six weeks (p = <0.01), 1.5 (0–6) at 16 weeks (p = <0.01) and 0.9 (0–9) (p = <0.01) at 2 years. On activity VAS scores improved from 7 (0–10) at baseline to 5 (0–8) at 6 weeks (p = <0.01), 4 (0–9) at 16 weeks (p = <0.01) and 2.18 (0–10) at 2 years (p = <0.01).

In the IAT group, there was improved pain at rest from a mean of 2.4 (0–9) pre-ESWT to 1.9 (0–8) at six weeks (p = 0.11), 1.5 (0–6) at 16 weeks (p = 0.04) and 0.8 (0–6) (p = 0.04) at 2 years. On activity VAS scores improved from 6.7 (0–10) at baseline to 5.5 (0–8) at 6 weeks (p = <0.01), 4.4 (1–8) at 16 weeks (p = <0.01) and 2.8 (0–10) at 2 years (p = <0.01).

3.2. VISA-A scores

The VISA-A score (maximum function 100) is shown at baseline, 6 weeks, 16 weeks and 2 years for the two groups (Table 1 and Chart 3). This shows a mean improvement from 40 (9–94) at baseline to 66 (18–94) at 2 years (p = <0.0001) in the NAT group. Mean VISA-A scores in the IAT group improved from 43 (7–72) at baseline to 70 (52–97) at 2 years (p = 0.0006). Improvement in VISA-A scores was statistically significant at all stages of follow-up in both groups.

3.3. Likert satisfaction scale scores

The Likert satisfaction score (Tables 2 and 3 and Chart 4) was completed at 6 weeks, 16 weeks and 2 years in both groups. There was a general trend towards improvement over time in both groups. However, the only statistically significant improvements were observed in the NAT group between both 6 and 16 week follow up (p = 0.054) and 16 weeks and 2 year follow up (p = 0.001). There was no significant improvement at any follow up point in the IAT group. The most common reason cited for Likert score of more than

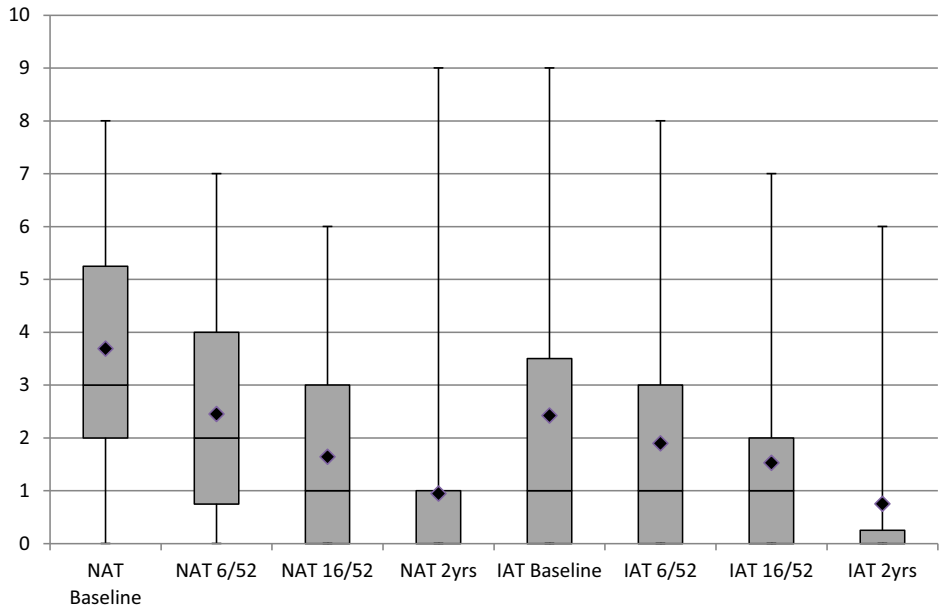

Chart 1. Mean VAS scores at rest.

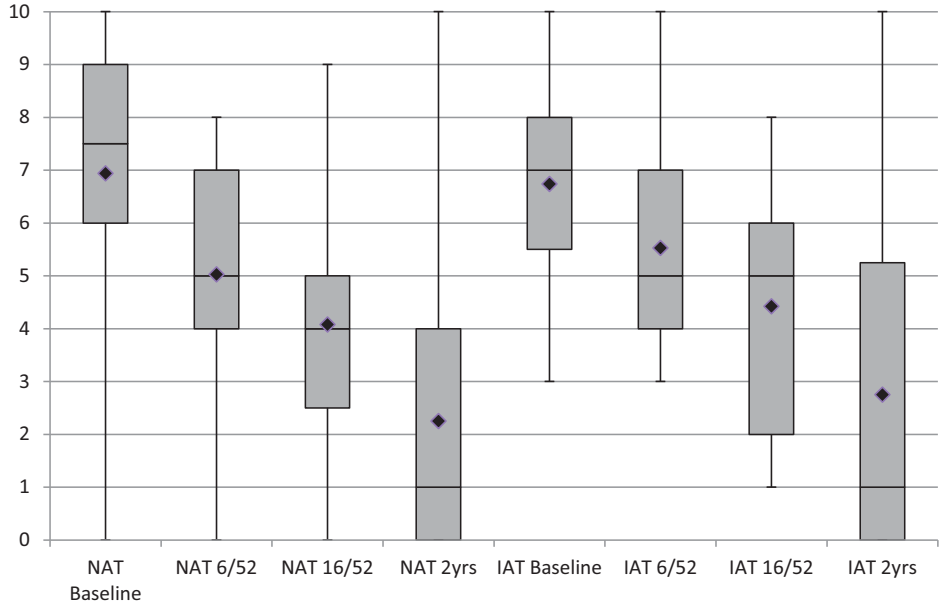

Chart 2. Mean VAS scores on activity.

1 at final follow up was persistent residual symptoms on activity, which was variable in intensity between patients.

3.4. Impact of patient age on outcome (Table 4)

There was a general trend for improvement in VAS and VISA-A scores across all age groups and for those with both NAT and IAT. However, Likert satisfaction scores did not improve significantly during follow up.

3.5. NAT group

Pain at rest improved significantly by 6 weeks post treatment in those aged under 60 ($p = <0.01$). In the over-60s significant relief of pain took slightly longer to achieve, however these patients achieved this by 16 weeks post treatment ($p = 0.014–0.017$). Pain on activity reduced significantly for all age groups by six weeks post treatment. VISA-A scores improved significantly by six weeks in all age groups other than 60–69 year olds, in whom this was achieved by 16 week follow up. Likert satisfaction scores only improved significantly in patients over 70 years of age and this was only observed at 2 year follow up.

3.6. IAT group

Significant improvement in pain at rest was only observed in 50–59 year olds and only at 2 year follow up ($p = 0.03$). Pain on activity was significantly reduced in all groups except the over 70s by 16 week follow up ($p = 0.01–0.03$). The over-70s groups could not be statistically assessed due to the low sample size in this subgroup. VISA-A scores improved for the under-60 s between 6 and 16 weeks post treatment, but did not improve significantly for those

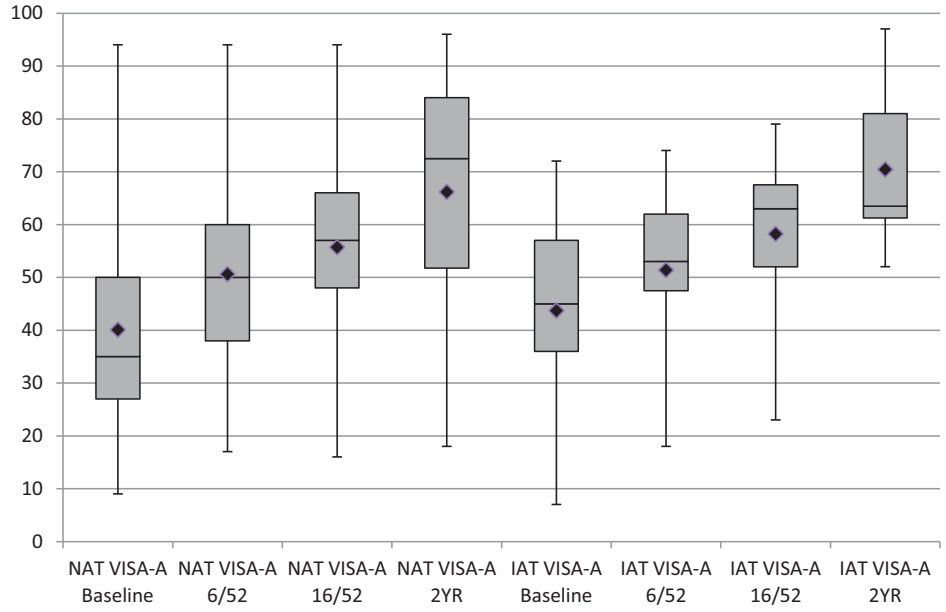

Chart 3. Mean VISA-A scores.

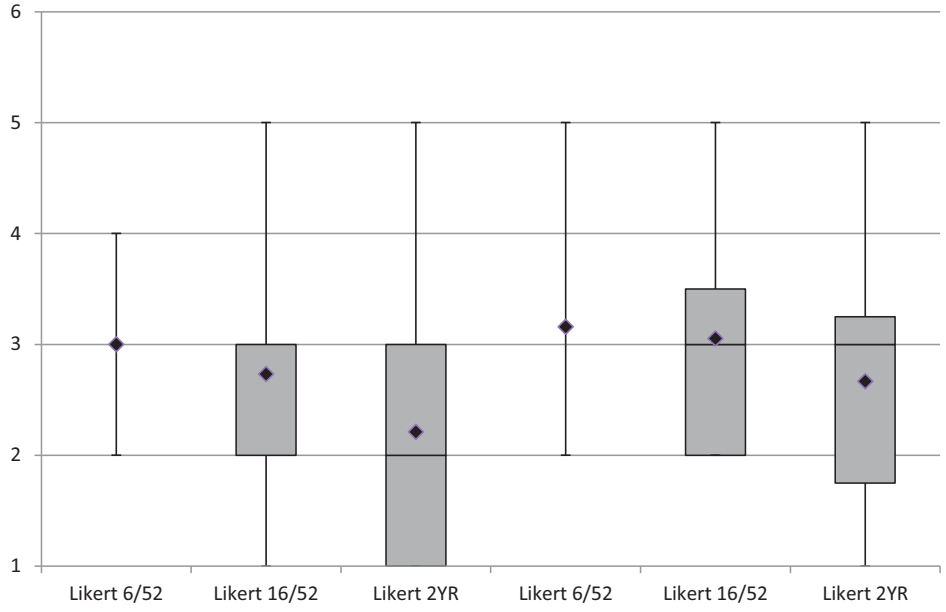

Chart 4. Mean Likert scores.

over 60 years of age. Likert satisfaction scores were not significantly improved in any group.

3.7. Impact of length of problem on outcome (Table 5)

Data was also subdivided depending on the length of symptoms at the start of treatment (Table 5).

3.8. NAT group

Unfortunately, due to small sample size in this subgroup, not all subgroups could be analysed. Significant reduction rest pain was observed at 16 week follow up in those with NAT for less than 6 ($p = 0.03$) months and 24–48 months ($p = 0.01$). Pain on activity also reduced significantly in these groups at 6 ($p = 0.003$) and 16 weeks ($p = 0.03$) respectively. Significant improvement in VISA-A scores was also seen at 16 week follow up in those with NAT for less than six months. Other data did either not reach significance or sample size was too small to permit analysis.

3.9. IAT group

Pain on activity reduced significantly in those patients who had had IAT for between 7 and 24 months ($p = 0.02$). VISA-A scores also improved in the 12–24 month group at 16 week follow up ($p = 0.04$). No significant reduction in rest pain, improvement in Likert scores or VISA-A scores was observed in the remainder of this subgroup, perhaps again as a result of small sample size and low rest pain scores on initial evaluation.

There were minimal side effects of ESWT recorded for our patient population. Only a few patients mentioned some erythema and tenderness over the area for the following days after the

Table 4
Mean scores categorised by age for main outcome measures.

	NAT				IAT			
	<50	50–69	60–69	>70	<50	50–69	60–69	>70
VAS rest								
Baseline	3.1	4.7	3.1	4.3	2.8	1.4	4.7	0
6 weeks	2.3	2.8	2.3	2.6	2.5	1.1	3.2	0
16 weeks	1.3	1.6	1.6	2	2.3	0.8	2.2	0
2 years	0.7	1	0.75	1.5	2	0	0.7	0
VAS activity								
Baseline	6.8	8	6.7	6.8	5.8	7.4	7.2	6
6 weeks	5.2	5.5	5	4.8	5.3	5.8	6	4
16 weeks	4.1	4.1	4.1	3.5	4.8	4.4	4.5	3
2 years	2.5	2.1	1.8	2	3.6	2.3	3.7	0
Likert								
6 weeks	3.1	3.1	2.7	3	3.3	3.1	3	3
16 weeks	2.8	2.6	2.7	2.6	3.5	2.8	3	2.5
2 years	2.5	2.2	2.1	1.6	3	2	3.2	2
VISA-A scores								
Baseline	39.4	32.1	42.7	48.8	44.5	39.1	58.7	27
6 weeks	48.2	44.1	52	61.6	47.6	49.8	62.2	46
16 weeks	52.8	50.6	54.5	69.3	52.5	57.5	66.7	60.5
2 years	62.6	71.2	65.8	68.8	69.6	71.6	72.2	66

treatment. All the patients continued through all the ESWT treatments despite these minor side effects.

4. Discussion

AT is believed to relate to a failed healing response of the Achilles tendon (TA), which may be linked to ongoing mechanical stresses being placed on the tendon secondary to excessive tendon loading [17]. The exact pathophysiological changes in refractory AT remain contested. However the changes in refractory Achilles tendinopathy are not thought to be primarily inflammatory in nature. Rather AT has been described as arising as a result of non-inflammatory intra-tendinous degeneration with resulting disruption of the normal fibrillar pattern of the tendon [18,19], although a 'peritendonitis' may be present in a number of subjects in the early phases of the condition [20]. Indeed, changes on ultrasound including a hypoechoic, thickened portion of the TA with evidence of increased vascularity on colour or power Doppler supports these histological findings.

Tendons in AT exhibit a disordered pattern of collagen fibres (with type III predominance) and increased vascularity [21–23]. It has long been thought that due to their poor vascular supply and regenerative ability, tendons subsequently heal primarily by scarring. Whilst this is true in part due to influx of fibroblasts and myofibroblasts in the chronic tendon damage, emerging evidence suggests a much more dynamic process following tendon injury, involving increased vascularity, cell matrix turnover and matrix metalloproteinases (MMPs), which have an important role in tissue degradation and repair [24]. Pain in AT is thought to be due to ingrowth of nerves from the peritendinous tissues into the substance of the damaged tendon. Increased levels of nociceptive peptides have also been observed in association with this phenomenon and are thought to further exacerbate pain in AT. Simultaneously, growth factors including insulin-like growth factor-1 (ILGF-1), platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF) and transforming growth factor beta (TGF-beta) are released which may stimulate healing via the promotion

Table 5
Mean scores categorised by length of symptoms.

	NAT					IAT				
	<6 mths	7–12 mths	13–24 mths	25–48 mths	>48 mths	<6 mths	7–12 mths	13–24 mths	25–48 mths	>48 mths
VAS rest										
Baseline	3.6		8	1.5	4.5	9	1	2.2	4	0.3
6 weeks	3.1			0.6	4	8	1	1.2	3	0.3
16 weeks	1.6			0.3	4	7	1	0.5	3	0.3
24 months	0.6			0.2	2.5	2	0	0.25	6	0
VAS (activity)										
Baseline	8.7		0	5.6	7.5	10	7.7	6	6.5	5.6
6 weeks	6.4			3.8	6.5	10	6	4.7	6	4.3
16 weeks	4.5			3.3	6	8	3.5	4.1	5.5	3
24 months	3.2			2.2	2.5	6	0.5	4	10	0
Likert										
6 weeks	3.3			2.8	4	4	3	2.8	3.5	3.3
16 weeks	2.8			2.6	4	5	2.5	2.8	3.5	3
24 months	3			2.4	2	5	1.5	3.2	4	2
VISA-A scores										
Baseline	28.1			56.1	26	65	36.2	46.4	53	42.6
6 weeks	37.6			64	30	63	46.5	53.4	54	53.6
16 weeks	47			64	30	66	56.2	59.1	57	65
24 months	54.8			75.6	55.5	64	78	62.2	63	74.5

of angiogenic effects, cellular proliferation and differentiation [25–27].

External factors such as poor footwear and training on hard surfaces may predispose to AT. In addition to external stresses, factors intrinsic to the patient such as excessive hindfoot motion and forefoot varus may also predispose to tendinopathy [24].

Although many patients are known to recover from AT through simple physiotherapy with eccentric exercises, a significant number fail to respond to these measures and the management of more recalcitrant cases has been the subject of some debate. Uncertainty regarding the aetiological and pathophysiological phenomena behind AT have led to numerous conservative treatments which have been trialled with varying success, including corticosteroid injection, platelet-rich plasma or aprotinin injection, laser therapy, electrocoagulation, ESWT and various surgical interventions [28]. ESWT has gained the support of NICE in the treatment of refractory Achilles tendinopathy and appears safe and effective in the management of refractory AT [12].

The exact mechanism of action of ESWT is still debated, although there is growing knowledge regarding the effects of ESWT on various tissues. Shockwaves have been shown to increase levels of type I and III collagen, TGF-beta and ILGF-1 molecules [24], which in turn increase extracellular matrix formation [25] and promote healing by providing a scaffold for tendon repair. Shockwaves also inhibit the release of cytokines and MMPs seen in diseased tendons [29]. Regarding relief of pain, the application of shockwaves to tendinopathic tendons has been suggested to be attributable to a direct analgesic effect perhaps mediated via the pain gate theory of Melzack and Wall [15]. Nitric oxide production is also stimulated at lower energy levels, which has analgesic and anti-inflammatory properties [24]. ESWT is also believed to promote healing through neovascularisation. This neovascularisation in association with the pain relief afforded by ESWT provides the opportunity for an individual to perform more activity which in turn promotes further vascularisation and positive reinforcement of the tendon healing process [25,26].

4.1. ESWT treatment regime

Patients in this audit were treated with three sessions of low energy radial ESWT (using 2500 pulses per treatment) administered at weekly intervals. Frequency and pressure ranged from 10 Hz and 1.5 Bar respectively for the first 500 pulses increasing to a pressure of 2.5 Bar for the remaining 2000 pulses as advised by the manufacturer. This allowed the patient to become accustomed to the sensation of ESWT. Energy levels used in ESWT have also been shown to have various effects on tissue with low energies yielding positive effects through stimulation of production of type I and III collagen, TGF-beta and nitric oxide [24]. Lower energy ESWT also reduces or negates the need for local anaesthesia. Indeed no patients treated during the audit period required local anaesthetic. High dose ESWT on the contrary has been shown to have at best significant inhibitory effects on the promotion of tissue regeneration and at worst increased tissue reaction and potential tendon damage [13] by invoking histological changes of inflammation, oedema and fibrosis [15]. For these reasons high dose ESWT does not constitute part of our treatment regimen for patients with refractory AT.

The macroscopic, microscopic and biologic features of damaged tendons described above would seem to represent an ideal theoretical candidate for treatment by ESWT considering the effects of low dose ESWT on tendon tissues described above. These assertions are also supported by the overall outcomes achieved by patients involved in this audit. Overall mean pain scores at rest and on activity reduced significantly in patients with both NAT and IAT. This occurred by six weeks post treatment in all groups except one (pain at rest in the IAT group). The improvements in VAS scores outlined above and the possible underlying mechanism of tendon healing/regeneration were also borne out by functional improvement in the groups. Both NAT and IAT groups improved significantly in this regard at all follow up points. These data support a role for ESWT in the management of both refractory NAT and IAT.

Patients with NAT reported significant improvement in Likert satisfaction scores over follow-up. This was, however not replicated in the IAT group despite mean Likert scores appearing similar. Patients in the IAT group did not achieve as low mean Likert scores as those in the NAT group, suggesting that these patient's expectations were not entirely met by treatment. This may be due to a smaller sample size in the IAT group, or possibly the fact that these patients had, on average, suffered AT for 22 months longer than their NAT counterparts. In addition we did observe several individuals in the IAT group who were diagnosed with a Haglund deformity and subsequently required surgery, which was beneficial in all of these patients. This supports the consensus that patients with bony exostoses or Haglund deformity may benefit from surgical intervention. However, ESWT may still be of benefit for some patients in this situation, as although heel spurs are not eradicated by ESWT [30], its effects may continue to be beneficial.

Subdivision of NAT and IAT data by patient age and chronicity of AT potentially yielded yet more information regarding the conditions. Regarding patient age, pain scores on activity improved amongst all age groups with both NAT and IAT. However symptom relief at rest and improvement of VISA-A scores tended to occur more quickly in those aged less than 60 years in both the NAT and IAT groups. Regarding chronicity there was a general trend for earlier and more reliable improvement in patients with pain symptoms less than 48 months (NAT) or 12 months (IAT). Improvement in VISA-A scores was only observed in patients with AT for less than 12 months. This has important implications for both prognostication in different age groups and suggests that earlier treatment in the course of refractory AT may be beneficial. Unfortunately, small sample sizes led to the inability to perform statistical analysis in some subgroups and therefore these findings should be viewed in context.

4.2. Contraindications to ESWT

ESWT was generally very well tolerated by the patients undergoing treatment in this audit. A number of contraindications are cited by manufacturers, including coagulation disorders, pregnancy, pacemaker devices and pulmonary tissue, tumours, epiphyseal growth plates and inflammatory changes in the vicinity of shockwave therapy field. Many of these did not apply to our group of patients and the main side effect of treatment appeared to be transient skin reddening and mild to moderate discomfort at the time of the ESWT procedure. We recorded no instances of tendon rupture during our 2 year follow up, although the authors feel that due to the similar pathophysiologic and histopathological changes seen in tendinopathic and ruptured TAs the two conditions may share a pathological basis [20].

4.3. Audit limitations

There were unfortunately several limitations to this audit. Due to the fact that this project was not designed as a research study, no control group was included. It is well known that the natural history of many musculoskeletal complaints is to tend towards resolution over time. However, in auditing patients with refractory tendinopathy we hope to have identified patients who failed management with initial conservative measures. In addition, the acceptance of ESWT by NICE as an efficacious treatment for refractory AT supports

its use, and despite this limitation it is felt that important findings have been highlighted in this project.

Losses to follow up also limit the validity of the audit by reducing sample size by almost 18%. At 16 week follow up, losses amounted to 9%. Reliance on follow up via telephone consultation did make contacting working individuals difficult and some patients lost to follow up had moved house by 2 year follow up.

It was also difficult to accurately assess other treatments that patients had received during the follow up period. No patients had any interventions other than eccentric loading exercises and/or heel pads and simple analgesia prior to the course of shockwave therapy. However, following treatment a minority of patients disclosed having had either steroid injection ($n = 2$) or surgery for a Haglund deformity ($n = 2$). Exclusion of these patients from the result cohort made no difference to the overall outcomes observed in this audit but would clearly further reduce sample size.

5. Conclusion

Overall, there was an improvement in patient symptoms of both NAT and IAT following treatment with ESWT as defined by VAS scores at rest and on activity and functional assessment via the VISA-A questionnaire. There were few side effects recorded and every patient was able to complete their full course of treatment. Although patients generally showed improvement from their treatment a number were not wholly satisfied with their perceived outcome of treatment and complained of residual symptoms. There is a possibility that associated bony abnormalities in patients with IAT have an adverse effect on the efficacy of ESWT, although conflicting data exists on this topic. Patients under the age of 60 and those with AT of less than 12 months duration may benefit earlier and to a greater extent than older patients and those with increasing chronicity.

The authors experience and the results of this audit have led them to support ESWT in patients with refractory AT but to council them that the treatment is likely to lessen, not cure, their symptoms. We feel that ESWT could play a part in the future management of patients with this challenging condition.

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