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Successful rapid return to performance following non-operative treatment of proximal hamstring tendon avulsion in elite athletes

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ABSTRACT

Objectives Proximal hamstring tendon avulsion injuries are severe and potentially career-threatening for elite athletes. Until now, no data have been published on the non-operative treatment of this injury in elite athletes. Therefore, the objective of this case series was to describe return to performance in elite athletes after non-operative treatment of full-thickness proximal hamstring tendon avulsion injuries as well as provide detailed description of the rehabilitation process and provide a mechanobiological hypothesis on processes leading to successful outcomes.

Methods In this retrospective case series, we included three elite athletes with four MRI-confirmed acute proximal hamstring tendon avulsions of the conjoint tendon and/or the semimembranosus tendon who opted for non-operative treatment following shared decision-making, consisting of an individualised rehabilitation programme. The primary outcome was time to return to performance (in weeks). Secondary outcomes were time to and rate of return to competition, rate of return to performance and re-injury rate.

Results Four proximal hamstring tendon avulsions in three elite athletes were included. All elite athletes returned to performance within 8–33 weeks, which for three out of four cases was at Olympic (gold medal) level.

Conclusion This (pilot) case series indicates that non-operative treatment for full-thickness proximal hamstring avulsion injury can result in return to performance in elite athletes. Non-operative treatment may therefore be a viable treatment option in selected (elite) athletes.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Clinical and sports outcomes on non-operative treatment for acute full-thickness proximal hamstring tendon avulsions in elite athletes are scarcely reported.

WHAT THIS STUDY ADDS

⇒ With strict monitoring, repeated physical and radiological assessment and a well-experienced medical team, elite athletes could return to performance in a relatively short time.

⇒ The efficacy of the applied rehabilitation programme presented in this study suggests that it successfully promoted the temporal sequence of processes in tendon healing which are key in the regeneration process.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Non-operative treatment for acute full-thickness proximal hamstring tendon avulsions is a viable alternative treatment option for motivated elite athletes and could therefore be considered as a treatment option for future cases in elite athletes after thorough counselling.

⇒ The detailed working method and mechanobiological considerations could be used to generate hypotheses for future research on the tendon healing process.

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INTRODUCTION

Proximal hamstring tendon avulsions (PHA) are potentially career-threatening for elite athletes.[1–5] There is debate regarding operative indications in active, middle-aged patients. Systematic reviews reported superior (patient-reported and clinical) outcomes following operative treatment.[1 5–7] However, there is evident publication bias as these systematic reviews have included a low number of non-operative patients, hindering a proper comparison.[8–10] Adding to the debate, recent studies, including one randomised study, stated that non-operative treatment is non-inferior to operative treatment, suggesting that it is a viable treatment option.[4 11 12]

Until high-quality evidence is available on treating PHA in the general population, decision-making remains challenging.[4] This challenge is even greater in elite athletes. Little data exists on operative outcomes in elite athletes, and virtually no non-operative outcomes are published, rendering a comparison to optimise decision-making impossible.

Our objective was to describe return to performance (RTP) outcomes after non-operative treatment for elite athletes with an acute full-thickness PHA in a retrospective pilot case series, provide a detailed description of the rehabilitation process and discuss a mechanobiological hypothesis on processes leading to successful outcomes.

METHODS

Participants

Three elite athletes treated non-operatively for an MRI-confirmed full-thickness PHA of one or both of the proximal hamstring tendons were included. A patient was categorised as an elite athlete when he/she performed services in a professional athletic event for wages or other remuneration. The demographics of the included elite athletes can be found in the results section.

Patient and public involvement

No patients were involved in our study design.

Shared decision-making

Operative treatment is considered the standard treatment in elite athletes.[3] This assumption is supported by the lack of literature for non-operative outcomes in elite athletes, but the risk of publication bias should be considered. Non-operative treatment has traditionally been viewed as doing 'less than surgery'. In elite athletes, where athletic and economic stakes are high, proceeding to operative treatment for severe injuries seems straightforward. We underline that this is a reflection of current clinical practice patterns rather than evidence-based practice.

Operative reattachment of the avulsed proximal hamstring tendons is performed with suture anchors, according to the current standard.[13]

Operative treatment and postoperative rehabilitation lead to prolonged absence from sports participation. Extended absence may result from planning of surgery, initial protective phases of the rehabilitation programme and/or (e.g., brace) immobilisation. This prolonged absence due to surgery might be unacceptable in situations where an athlete aims to participate in a high-tier event (i.e., Olympic games) that is scheduled sooner than the expected return to play (and performance) after operative treatment. In such a situation, discussion and shared decision-making are essential, ideally including the athlete and their entourage. Shared decision-making means that treatment options (operative vs non-operative treatment) should be discussed with the patient, including expected short- and long-term advantages and disadvantages/risks, expected return to sport time frames and short- and long-term athlete goals should be weighed. In athletes in these specific situations (prolonged absence due to surgery), non-operative treatment was discussed as an alternative treatment option with a chance of being game-ready in time for said event(s). In our clinic, the treatment choice was made by the athlete and a team of treating clinicians including an orthopaedic surgeon (GK) with over 15 years of expertise in treating muscle/tendon injuries in elite athletes.[4]

Non-operative treatment and interim analyses

After decision-making, elite athletes were referred to a specialised physiotherapist with extensive experience in treating PHA. Rehabilitation was started immediately after confirmation of the diagnosis.

Approximately 52% of non-operatively treated middle-aged patients will regain continuity of the proximal hamstring tendons.[4] To monitor the desired (spontaneous) restoration of proximal continuity, all included elite athletes were strictly monitored once every 2–3 weeks in our outpatient clinic. These follow-up appointments included MRI examination to assess radiological progression of tendon healing. Together with the athlete and treating physiotherapist, clinical progression through the rehabilitation process, physical examination including strength/flexibility testing and radiological variables were weighed.

With this strict monitoring, the option to convert to operative treatment within 6–8 weeks after injury remained available. A previous study demonstrated[4] no difference in clinical outcomes between patients who underwent surgery within 2 weeks versus within 6–8 weeks after injury. Therefore, we used a cut-off period of 6–8 weeks to evaluate progression of the rehabilitation. If the expected progression criteria were not met within this time frame, conversion to operative treatment could be discussed.

Individualised rehabilitation programme

To start the rehabilitation, a programme was set-up that mimicked athletes' normal week in terms of physiological and metabolic demands. In order to not lose time, emphasis was placed on halting decline in general and specific fitness. Personal physical goals were defined based on the athletes' pre-injury levels of strength and endurance based on Global Positioning System data. With this data, a benchmark for return to sports was set.

The rehabilitation programme started on the day of the injury or very shortly after on confirmation of the diagnosis. The athlete started with heavy loading, as early and as much as possible to maintain fitness levels, address central and local inhibition and build tissue capacity. On a daily basis, two sessions were executed with a 6-hour break in between. This is to maximise upregulation of collagen synthesis and to avoid central fatigue. Clinical testing was repeated pre-training and post-training to follow reactivity and progress accordingly. The aim of every session was to create a pain-free line of force through the injured area to upregulate and give direction to collagen synthesis.

After fulfilling isometric and concentric exercises pain-free, running and sprinting drills were incorporated after being non-reactive to multidirectional single-leg hops. Sprinting drills, bounds and skips approach angular

Table 1 A schematic overview for the individualised rehabilitation programme for elite athletes with proximal hamstring tendon avulsions. For a more detailed overview of the build-up scheme for rehabilitation in week 1, we refer to online supplemental appendix A

	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6
Strength	Gym 4×	Gym 4×	Gym 4×	Gym 4×	Gym 4×	Gym 3×
	Circuit 4×	Circuit 3×	Circuit 2×	Circuit 2×	Circuit 2×	Circuit 2×
Hamstring	Long holds, low jerk	Long and short holds, medium jerk	Long and short holds, medium jerk	Holds and pushes, high jerk	Holds and pushes, high jerk	Long and short holds, medium and high jerk
	Isometric, multiple angles	*Isometric, multiple angles*	Isometric, multiple angles	Isometric, multiple angles	Isometric, multiple angles	Isometric, multiple angles
Synergists	Hip extension	Hip extension	Hip extension	Hip extension	Hip extension	Hip extension
	Isometric, short lever only	Isometric and concentric, short lever only	Isometric and concentric, short lever only	Isometric and concentric, long lever	Isometric and concentric, long lever	Isometric and concentric, long lever
	Combined hip extension and knee flexion	Combined hip extension and knee flexion	Combined hip extension and knee flexion	Combined hip extension and knee flexion	Combined hip extension and knee flexion	Combined hip extension and knee flexion
	Isometric, short lever only	Isometric, long lever	Isometric, long lever	Isometric and concentric, long lever	Isometric and concentric, long lever	Isometric and concentric, long lever
Lateral force system	Restricted ROM squat variations	Restricted ROM squat variations	Full ROM squat variations	Full ROM squat variations	Full ROM squat variations	Full ROM squat variations
	Accessory strength work	Restricted ROM Olympic lifts	Restricted ROM Olympic lifts	Full ROM Olympic lifts	Full ROM Olympic lifts	Full ROM Olympic lifts
	LOAD: HIGH	Accessory strength work	Accessory strength work	Accessory strength work	Accessory strength work	Accessory strength work
		LOAD: HIGH	LOAD: HIGH	LOAD: HIGH	LOAD: HIGH	LOAD: HIGH
Running	Running 2×	Running 3×	Running 5×	*Running 5×*	*Running 5×*	Running 5×
	Ankle dominant plyo's	Jog, easy pace	Build volume<50% max velocity	Build volume <50% max velocity	Maintain baseline <50% max velocity	Maintain baseline <50% max velocity
	Running drills	Ankle and knee dominant plyo's	Increase comfortable running speed	Build volume 50–70% max velocity	Maintain baseline 50–70% max velocity	Maintain baseline 50–70% max velocity
		Running drills	Start slow accel/decel	Build volume slow accel/decel and COD	Build volume 70–85% max velocity	Maintain baseline 70–85% max velocity
		Stairs		Start increasing intensity accel/decel	Build volume slow accel/decel up to baseline	Build volume 85–95% max velocity
					Build volume fast accel/decel	Maintain baseline slow accel/decel
						Build volume fast accel/decel up to baseline
Accessory speed preparation			Ankle, knee and hip dominant plyo's	Ankle, knee and hip dominant plyo's	Ankle, knee and hip dominant plyo's	Ankle, knee and hip dominant plyo's
			Sprinting drills	Sprinting drills	Sprinting drills	Sprinting drills
			Resisted accel/decel	Resisted accel/decel	Resisted accel/decel	Resisted accel/decel
				Resisted sprints	Resisted sprints	Resisted sprints
Alternative conditioning	Alternative 2×	Alternative 2×	Alternative 1×			
	Pool	Pool	Assault bike			
	Assault bike	Assault bike				

ROM, Range of Motion.

velocities reached in sprinting in ankle, knee and hip without building horizontal velocity.

With the athlete able to perform endurance running sessions, normal week loads at lower running speeds were built. With increasing strength values, running speeds were gradually increased and acceleration/deceleration and change of direction were incorporated. In these cases, the athlete was able to run within 2 weeks, reaching 80% of pre-injury maximum velocity around day 30. In table 1 a schematic overview of the rehabilitation programme for elite athletes with PHA can be found.

Physical examination in outpatient clinic

During follow-up in the outpatient clinical strength tests with the handheld dynamometer, the Straight Leg Raise (figure 1A), Active Knee Extension Test (figure 1B),

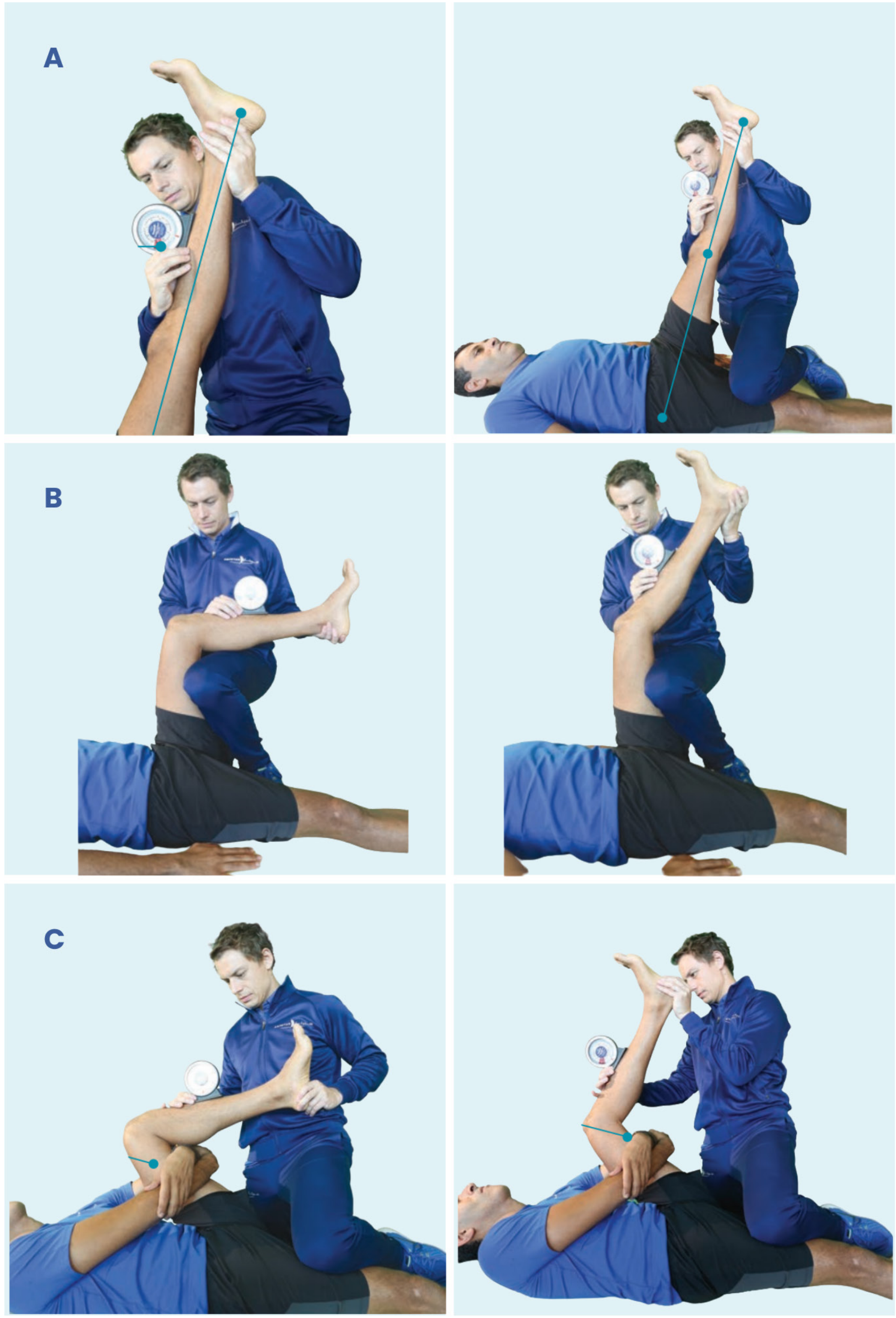

Figure 1 A schematic overview of the tests during physical examination in the outpatient clinic. (A) Is the Straight Leg Raise Test; (B) is the Active Knee Extension Test and (C) is the Maximal Hip Flexion Active Knee Extension test. Images from the Aspetar Hamstring Protocol were used after reprint permission from Aspetar (www.aspetar.com).

Maximum Hip Flexion Active Knee Extension Test (figure 1C), ability to develop tension on the hamstrings and presence/extent of pain on palpation were used for monitoring the progression of the elite athlete.

MRI examination

The MRI protocol can be found in online supplemental appendix B.[4] MRIs were scored by experienced musculoskeletal radiologists.

At baseline, MRI scoring of the proximal hamstring complex included bone-tendon/tendon continuity, direct tendon retraction, presence and size of haematoma/seroma, presence of organising scar tissue in the (lateral) haematoma/seroma wall. Proximal continuity was scored using the dropped ice cream sign.[14] Tendon retraction was measured via the direct measurement method.[14] As part of the standardised assessment, the

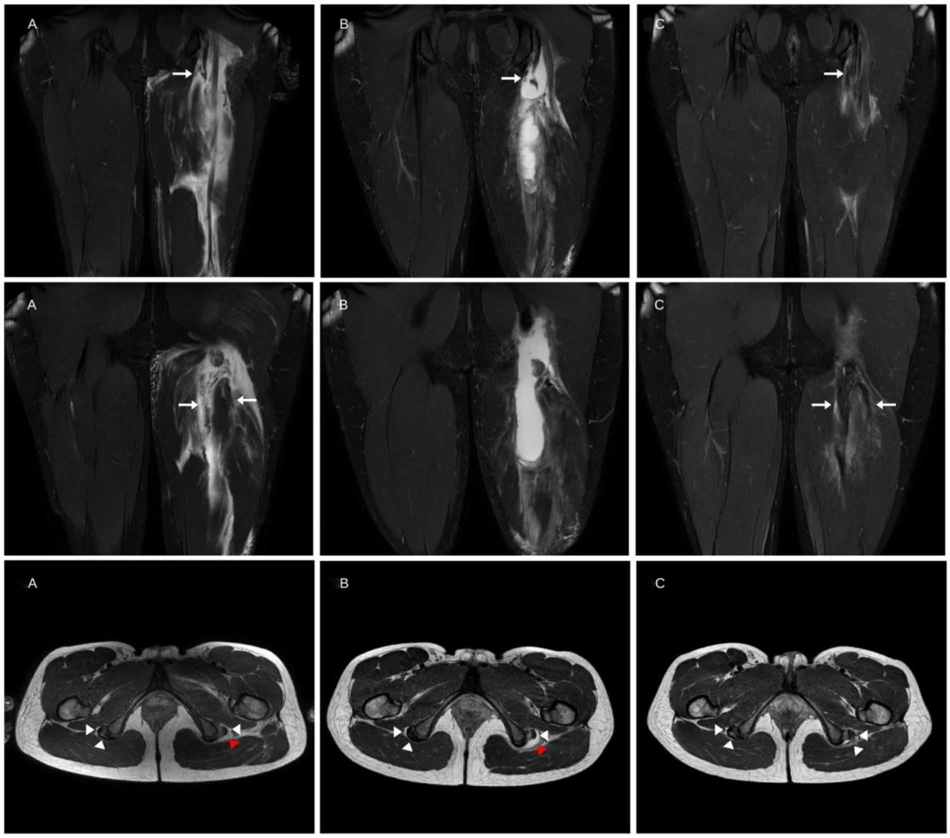

Figure 2 MRI series of an elite athlete. In the coronal series (1A/B/C) the healing process of a full-thickness injury of the free tendon of the semimembranosus muscle is displayed. Over time, a decrease in seroma/haematoma is seen in combination with forming proximal continuity indicated by the white arrow in 1C. In the second coronal series (2A/B/C) the healing process of the conjoined tendon of the long head of the biceps femoris and the semitendinosus is displayed. In 2A there is waviness of both the biceps femoris and the semitendinosus tendon, which is a sign of loss of tension suggesting a full-thickness disruption of the tendons. Over time there is formation of fibrotic tissue in the haematoma/seroma wall. In 2C tendon thickening and reduction of waviness are displayed as signs of tendon healing and recovery of tension. Furthermore, in the axial series (3A/B/C) the healing process of the full-thickness injury of the conjoint tendon is shown. In 3A and 3B, the white arrows show continuity of the tendons, the red arrows indicate the loss of proximal continuity of the conjoined tendon at the ischial tuberosity. In 3C the growth of a neotendon, adjacent to the semimembranosus tendon, is displayed.

intramuscular tendons and musculotendinous junctions were evaluated for oedema and focal disruption.

We used sequential MRIs (figure 2) to assess tendon healing tendency in elite athletes. It should be noted that this is eminence rather than evidence-based practice. On these interim MRIs, we essentially look for decrease of oedema/seroma, and formation of fibrotic tissue in the lateral haematoma/seroma wall that develops/remodels into a longitudinal structure connecting origin and tendon stump (ie, 'neotendon'). As part of this process, bridging of the 'gap' is seen, with recovery of tendon tension, signified by reduction of tendon waviness. Interim findings, along with clinical parameters, were shared/discussed with treating physiotherapists to further tailor the rehabilitation programme to the healing phase.

Return to play

With the athletes' pre-injury levels in mind, they were cleared for return to play if specific criteria were met. Before returning to play, the athlete was able to fulfil the training sessions without extensive reaction (i.e., no muscle soreness longer than 24 hours). The maximum deficit in strength should be no more than 15% in comparison to the contralateral leg. This is an arbitrary threshold that reflects the 'symmetry' of strength. The underlying rationale is that for certain activities or rehabilitation, sufficient hamstring strength (or recovery) is required as a condition for progress. This threshold was selected based on the study by Tol *et al*,[15] in which 67% of the clinically recovered hamstring injuries showed at least one hamstring isokinetic testing deficit of more than 10%. This implies that normalisation of isokinetic strength may not be essential for a successful return to play. Furthermore, the athlete should be able to run one and a half times the average distance covered at pre-injury level without reaction. Proximal continuity and minimal oedema on MRI were radiological criteria that should be met before the athletes were cleared for return to play.

Table 2 Patient demographics and return to sport outcomes in elite athletes with proximal hamstring tendon avulsions

Case	Gender	Age (years)	Sport	Side	Affected tendons	Retraction (mm)	RTP	RTP level	OFR (weeks)	RTT (weeks)	RTC (weeks)	RTP (weeks)
1a*	Female	28	Field-hockey	Right	CT	21	Yes	Olympic gold medal level	2	7	13	13
1b*	Female	31	Field-hockey	Left	CT and SM	47	Yes	Olympic gold medal level	2	6	8	12
2	Female	26	Field-hockey	Left	CT and SM	41	Yes	National level	4	9	26	33
3	Male	28	Bobsleigh	Left	CT	37	Yes	Olympic level	4	6	7	8

*This athlete suffered two separate injuries.
CT, conjoined tendon; OFR, on-field rehabilitation; RTC, return to competition; RTP, return to performance; RTT, return to training; SM, semimembranosus tendon.

Data collection

The data collection was performed retrospectively by one researcher, who contacted the elite athletes and the treating physiotherapists to complete a standardised questionnaire with demographic data, return to sport outcomes and data on (re-)injuries (online supplemental appendix C).

Return to sport outcomes

The primary outcome was the RTP time in weeks. The RTP time in weeks is the number of weeks from injury until the time that the athlete is capable of performing at or above his/her pre-injury performance level after rehabilitation. Depending on the sport, variables like strength, speed and agility tests were recorded and personal match statistics were analysed and compared with pre-injury measurements. The return to sports continuum is divided into four stages: the on-field rehabilitation (OFR), return to training (RTT), return to competition (RTC) and finally RTP.[16] OFR is defined as the time from injury until the athlete is capable of resuming (individual) rehabilitation on the training pitch. RTT is the number of weeks before the athlete finishes the OFR and is capable of participating in team training (adapted or fully). The RTC is the number of weeks from injury until the time that the athlete is capable of participating in competitive matches without the necessity to be able to play at pre-injury performance level.[16 17] We recorded the RTP rate, which is the percentage of athletes that were able to perform at or above his/her pre-injury performance level after rehabilitation.

Re-injuries

A re-injury was defined as a new MRI-confirmed hamstring injury in the affected ipsilateral hamstring tendon(s) during or after completing the rehabilitation programme.

Statistical analysis

Due to the small sample size, no statistical analysis was performed. Data are presented using descriptive statistics.

RESULTS

Demographics

We included four full-thickness PHA in three elite athletes. One female athlete incurred two separate injuries; 136 weeks after her first injury, she obtained the second, so she was included twice. An overview of the patient demographics and return to sport outcomes is presented in table 2.

Re-injuries

We recorded one re-injury. One athlete suffered another full-thickness injury of both the conjoint and the semimembranosus tendon 4.5 years (234 weeks) after her index injury. She got injured after a contact in full sprint that forced her into an abduction side step during full extension. She had been performing at Olympic (gold medal) level the entire period after her first injury.

DISCUSSION

This pilot case series showed that non-operative treatment for acute PHA is a viable and safe alternative treatment option for elite athletes. With strict monitoring, repeated physical and radiological assessments and an experienced medical team, elite athletes can RTP in a relatively short time without the surgical complications risks, while maintaining the ability to convert to operative treatment within 6–8 weeks if indicated.

Return to performance rate

Although this pilot study consisted of a small group of elite athletes, all four cases returned to their pre-injury performance level. Due to the small sample size, findings cannot be extrapolated to all elite athletes. However, it highlights that non-operative treatment provides an opportunity for rapid RTP with low complication risk.

In a systematic review,[7] 221 recreational and competitive athletes were identified, of which 24 athletes were treated non-operatively with an RTP rate of 54%. In their operatively treated group of 197 athletes, a 99% RTP rate within 1 year of treatment was described. These results appeared to indicate that surgery is necessary for return to sporting activities. The difference in their results probably lies in the fact that no elite athletes were included. Elite athletes most probably have a different metabolism more suited for quick regeneration of tissue; elite athletes will also have a different mindset that allows an all-in rehabilitation period to facilitate more efficient healing and RTP and an additional factor is that most elite athletes will have better facilities for a 24–7 rehabilitation programme to achieve optimal outcomes.

Time to return to performance

Time to RTP level was a median 13 weeks. An explanation for the relatively short RTP duration is that we started rehabilitation directly after injury and tried to go through all phases of healing[18,19] (inflammation, proliferation, differentiation and remodelling phase). Parallel to our rehabilitation programme, without interruption, an operation will add a new trauma and therefore from operation a patient will have to go through all the phases of healing again, this will delay the RTP. Also, operatively treated patients will have limited mobilisation and weight bearing postoperatively. These factors could contribute to a prolonged recovery period postoperatively.

Re-injuries

We noted one re-injury in our study.

Only one study reported re-injuries following PHA in elite athletes.[20] No re-injuries were reported in two operatively treated elite athletes. One re-injury was reported in a competitive athlete, for which surgical repair followed. It should be mentioned that this study had a mean follow-up of 20 months. In our study, the re-injury occurred after 4.5 years and was at a different anatomic level as was the index injury. The injury occurred after an adequate contact trauma as mentioned in the results. No other studies were published that reported complications in elite athletes with full-thickness PHA, neither for non-operative treatment, nor for operative treatment.

Clinical/radiological assessment

The cut-off period of 6–8 weeks that is used to evaluate progression of rehabilitation was based on the systematic review of van der Made *et al*[5] and national guidelines for PHA. Reaching definitive conclusions on tendon healing on MRI within this period is challenging. It should be mentioned that the frequency of imaging (every 2–3 weeks) was not evidence-based but eminence-based. It is reasonable to anticipate that within 2–3 weeks, both clinical and radiological parameters will demonstrate progression. The extent of this progression guided the decision-making process regarding the continuation of non-operative treatment or potential conversion to surgical intervention.

Tendon healing hypothesis

The fast recovery and return to play of the non-operatively treated elite athletes raises the question of how the rehabilitation programme promoted the restoration of proximal continuity of the tendons and functional recovery of the hamstrings. Although not proven for our cases, it is expected and highly conceivable that a key factor in the rehabilitation programme is that from the day of injury the programme aims to apply mechanical loads onto the affected muscles as early and as much as required to maintain fitness levels. The mechanical loading has likely been critically involved in promoting the regeneration during the different phases of tendon and muscle regeneration.

Muscle and tendon regeneration after severe injury consists of four phases, that is, the inflammation, proliferation, differentiation and remodelling phase.[18,19] Acutely after the rupture, the local arterial and venous circulation will be damaged leading to a haematoma which could develop into a seroma. With the damage to the vascular system, immune cells such as monocytes and neutrophils will enter the site of injury and start to proliferate and transform into a pro-inflammatory phenotype, which will enable them to remove the debris of the injured tendinous tissue. The second phase consists of the activation and proliferation of (myo)fibroblasts, tenocytes and muscle stem cells to increase their number. Since these cells are the collagen-producing cells, their activation will lead to the synthesis of collagen and non-collagen extracellular matrix proteins as building blocks for the new tendon to be formed.

Key in the optimisation of restoration is the anatomical and biomechanical situation acutely after the injury and the graded mechanical loading of the muscle bellies, fascia and tendonous structures during rehabilitation. With a PHA, these muscles lost their tendinous connections with their origins at the ischial tuberosity and as such resistance to their shortening. If the tendons were the only structures via which the muscle fibres could have transmitted forces to the muscle origins at the bone structures, the muscle fibres would have become maximally shortened and would not be able to exert forces anymore according to their parabolic length-force relation.[21] However, since muscle fibres are embedded in a honeycomb network of connective tissue sheets (ie, fascia), which is what connects the muscle fibres to the fascia between muscles (ie, epimysium) and to the intermuscular septa between compartments, muscles can transmit their force laterally to their neighbouring structures by exerting shear forces onto these fascia.[22] This force transmission is referred to as myofascial force transmission and will cause shear deformations in the fascia surrounding the muscle belly and will provide resistance to shortening of the muscle belly.[22] This prevention of shortening is likely crucial for the process of muscle and tendon regeneration as myofascial connections of the muscles will allow the application of mechanical loads onto the muscle belly as well as on the sleeve of fascia surrounding the ruptured proximal tendon and the seroma. There are two potential mechanisms via which myofascial forces transmission between muscle belly and fascia surrounding the muscle, which are likely crucial for the regeneration of muscle and tendon injury. First, mechanical loading of the muscle belly will prevent muscle atrophy and shortening of the muscle fibres (ie, loss break down of contractile filaments in parallel and in series, respectively). Unloading of skeletal muscle by disuse or immobilisation in a shortened position will reduce protein synthesis and stimulate protein breakdown.[23] These processes would make the muscle weaker and shorten the length range of force exertion.[21 24] The other reason is that the mechanical loading of the fascia will stimulate the regeneration of the tendon by promoting the phases of regeneration. Mechanical loading of macrophages can enhance their pro-inflammatory activity and phagocytosis,[25 26] which will enhance the clearance of cell and collagen debris. Activated macrophages will produce signalling molecules that promote the activation and proliferation of mesenchymal stem cells and collagen-producing cells such as (myo)fibroblasts and tenocytes.[18 19] Simultaneously, these embedded cells in the fascia will be subjected to mechanical loads and in response to this loading will produce and secrete growth factors enhancing the synthesis of collagen.[27 28] The MRI imaging showed that over time there was formation of fibrotic tissue in the seroma wall. The thickening of the connective tissue at this location is indicative of the start of collagen production within the fascia surrounding the tendons and muscles. Moreover, the reduction of waviness in the imaging is displayed as a sign of tendon healing and recovery of tension. These observations support the notion that the fascia are the sites where tendon regeneration is initiated.

Key in the regenerative process is that the pro-inflammatory state of macrophages will switch towards an anti-inflammatory state which will enhance the production of the highly potent transforming growth factor β (TGF-β) for collagen synthesis (myo)fibroblasts.[29 30] Here, the mechanical loading of the pro-inflammatory macrophages as well as that of the other cells may be a critical factor as well, as this will foster the expression of insulin-like growth factor 1 (IGF-1) and interleukin-4,[25 30] which will promote immunological switch in macrophage phenotype[30 31] resulting in elevated local TGF-β levels.[32] Note that overload and associated renewed damage should be prevented as this will prolong the proliferation of cells, and impede the collagen production by anti-inflammatory macrophages.

The efficacy of the applied rehabilitation programme presented in this study suggests that it successfully promoted the temporal sequence of processes which are key in the regeneration process. The graded loading of the hamstring muscles guided discomfort has likely provided the basis for reinforcement of the myofascial linkages between the muscle belly and the extramuscular fascia and subsequent activation of the immune cells and collagen-producing cells. Noteworthy is that the success could also be related to the fact that elite athletes are physically well-trained and therefore their fascia were anatomically adapted to withstand the myofascial loads and transmit forces onto the fascia and seroma/haematoma. Another factor that could have contributed to the success is that the elite athletes are endocrinologically primed (ie, high serum levels of growth factors like IGF-1[33–35]), hence able to respond by early initiation and effective progression of the phases of regeneration. Future research should focus on individual differences in the morphology of anatomical structures including the interaction between muscle and fascia as well as in the spatial-temporal molecular processes. Such insights will help to optimise the personalised rehabilitation of patients with PHA even further.

Clinical implications

We consider these findings clinically relevant as they provide data on non-operative outcomes in elite athletes, a detailed description of the rehabilitation process and underlying mechanobiological theory. With a 100% rapid RTP rate, even at Olympic gold level in pivoting sports, this study shows promising results in treating this severe injury non-operatively. Nevertheless, it remains a pilot study as the sample size was small. Additionally, it should be mentioned that non-operative treatment is still a highly active process with strict monitoring and follow-up. If treatment for PHA in elite athletes continues to shift towards non-operative approaches, the healthcare system must be adequately equipped to ensure comprehensive monitoring and long-term support for these high-demand elite athletes.

Strengths and limitations

Our study is the first to report RTP outcomes in non-operatively treated elite athletes. Such data is scarce as most elite athletes are treated operatively due to expected superior outcomes and considering the athletic/economic stakes. Our data indicate that further research on (optimising) non-operative outcomes is possible and warranted. Hence, the medical staff was able to manage athletes' expectations and demands in the rehabilitation process before returning to sports.

Obvious limitations of this study were the retrospective study design and the small sample size. As a result, no formal statistical analysis was done, firm conclusions can therefore not be drawn.

Future prospective studies with large sample sizes in elite athletes with PHA are therefore encouraged.

CONCLUSION

This pilot case series indicates that non-operative treatment can result in successful RTP in selected elite athletes with a PHA injury. Using an individualised rehabilitation protocol with clinical progression criteria in combination with strict clinical and radiological monitoring, the elite athletes returned to performance in a relatively short time frame without complications.

For evidence-based indications, large studies investigating long-term outcomes are required to determine whether non-operative treatment provides acceptable outcomes in elite athletes. Furthermore, the detailed working method and mechanobiological considerations could be used to generate hypotheses for future research on the healing process of (hamstring) tendon injuries.

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Contributors KL was involved in study design, data collection, data analysis, data interpretation and drafting. ADvdM was involved in study design, data collection, data interpretation and drafting. RJ and RT were involved in drafting. MM was involved in drafting and for his radiological expertise and assessment of MRIs. GK was involved in the treatment process, study design, data collection, data interpretation and drafting and is the guarantor.

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Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request.

ORCID iDs

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REFERENCES

1 Bodendorfer BM, Curley AJ, Kotler JA, *et al*. Outcomes After Operative and Nonoperative Treatment of Proximal Hamstring Avulsions: A Systematic Review and Meta-analysis. *Am J Sports Med* 2018;46:2798–808.
2 Harris JD, Griesser MJ, Best TM, *et al*. Treatment of proximal hamstring ruptures - a systematic review. *Int J Sports Med* 2011;32:490–5.
3 Plastow R, Kerkhoffs GMMJ, Wood D, *et al*. London International Consensus and Delphi study on hamstring injuries part 2: operative management. *Br J Sports Med* 2023;57:266–77.
4 van der Made AD, Peters RW, Verheul C, *et al*. Proximal hamstring tendon avulsions: comparable clinical outcomes of operative and non-operative treatment at 1-year follow-up using a shared decision-making model. *Br J Sports Med* 2022;56:340–8.
5 van der Made AD, Reurink G, Gouttebarge V, *et al*. Outcome After Surgical Repair of Proximal Hamstring Avulsions: A Systematic Review. *Am J Sports Med* 2015;43:2841–51.
6 Best R, Eberle J, Beck F, *et al*. Functional impairment after successful surgical reconstruction for proximal hamstring avulsion. *Int Orthop* 2019;43:2341–7.
7 Buckwalter J, Westermann R, Amendola A. Complete proximal hamstring avulsions: is there a role for conservative management? A systematic review of acute repairs and non-operative management. *J ISAKOS* 2017;2:31–5.
8 van der Made AD, Hölmich P, Kerkhoffs G.M.M.J., *et al*. Proximal hamstring tendon avulsion treatment choice depends on a combination of clinical and imaging-related factors: a worldwide survey on current clinical practice and decision-making. *J ISAKOS* 2019;175–80.
9 Pasic N, Giffin JR, Degen RM. Practice patterns for the treatment of acute proximal hamstring ruptures. *Phys Sportsmed* 2020;48:116–22.
10 Laszlo S, Nilsson M, Pihl E, *et al*. Proximal Hamstring Tendon Avulsions: A Survey of Orthopaedic Surgeons' Current Practices in the Nordic Countries. *Sports Med Open* 2022;8:49.
11 Pihl E, Skoldenberg O, Nasell H, *et al*. Patient-reported outcomes after surgical and non-surgical treatment of proximal hamstring avulsions in middle-aged patients. *BMJ Open Sport Exerc Med* 2019;5:e000511.
12 Pihl E, Laszlo S, Rosenlund A-M, *et al*. Operative versus Nonoperative Treatment of Proximal Hamstring Avulsions. *NEJM Evid* 2024;3.
13 Moatshe G, Chahla J, Vap AR, *et al*. Repair of Proximal Hamstring Tears: A Surgical Technique. *Arthrosc Tech* 2017;6:e311–7.
14 van der Made AD, Smithuis FF, Buckens CF, *et al*. Good Interrater Reliability for Standardized MRI Assessment of Tendon Discontinuity and Tendon Retraction in Acute Proximal Full-Thickness Hamstring Tendon Injury. *Am J Sports Med* 2021;49:2475–81.
15 Tol JL, Hamilton B, Eirale C, *et al*. At return to play following hamstring injury the majority of professional football players have residual isokinetic deficits. *Br J Sports Med* 2014;48:1364–9.

16 Buckthorpe M, Frizziero A, Roi GS. Update on functional recovery process for the injured athlete: return to sport continuum redefined. *Br J Sports Med* 2019;53:265–7.
17 Ardern CL, Glasgow P, Schneiders A, *et al*. 2016 Consensus statement on return to sport from the First World Congress in Sports Physical Therapy, Bern. *Br J Sports Med* 2016;50:853–64.
18 Nourissat G, Berenbaum F, Duprez D. Tendon injury: from biology to tendon repair. *Nat Rev Rheumatol* 2015;11:223–33.
19 Voleti PB, Buckley MR, Soslowsky LJ. Tendon healing: repair and regeneration. *Annu Rev Biomed Eng* 2012;14:47–71.
20 Folsom GJ, Larson CM. Surgical treatment of acute versus chronic complete proximal hamstring ruptures: results of a new allograft technique for chronic reconstructions. *Am J Sports Med* 2008;36:104–9.
21 Kruse A, Rivares C, Weide G, *et al*. Stimuli for Adaptations in Muscle Length and the Length Range of Active Force Exertion-A Narrative Review. *Front Physiol* 2021;12:742034.
22 Huijing PA. Epimuscular myofascial force transmission: a historical review and implications for new research. *J Biomech* 2007;42:9–21.
23 WesselT*et al*. The muscle fiber type-fiber size paradox: hypertrophy or oxidative metabolism. *Eur J Appl Physiol* 2010;110:665–94.
24 Huijing PA, Jaspers RT. Adaptation of muscle size and myofascial force transmission: a review and some new experimental results. *Scand J Med Sci Sports* 2005;15:349–80.
25 Jain N, Moeller J, Vogel V. Mechanobiology of macrophages: How physical factors coregulate macrophage plasticity and phagocytosis. *Annu Rev Biomed Eng* 2019;21:267–97.
26 Younesi FS, Miller AE, Barker TH, *et al*. Fibroblast and myofibroblast activation in normal tissue repair and fibrosis. *Nat Rev Mol Cell Biol* 2024;25:617–38.
27 Kjaer M. Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. *Physiol Rev* 2004;84:649–98.
28 Wang JH. Mechanobiology of tendon. *J Biomech* 2006;39:1563–82.
29 Hillege M, Galli Caro R, Offringa C, *et al*. TGF-β Regulates Collagen Type I Expression in Myoblasts and Myotubes via Transient Ctgf and Fgf-2 Expression. *Cells* 2020;9:375.
30 D'Urso M, Kurniawan NA. Mechanical and Physical Regulation of Fibroblast-Myofibroblast Transition: From Cellular Mechanoresponse to Tissue Pathology. *Front Bioeng Biotechnol* 2020;8:609653.
31 Tonkin J, Temmerman L, Sampson RD, *et al*. Monocyte/Macrophage-derived IGF-1 Orchestrates Murine Skeletal Muscle Regeneration and Modulates Autocrine Polarization. *Mol Ther* 2015;23:1189–200.
32 Nuñez SY, Ziblat A, Secchiari F, *et al*. Human M2 Macrophages Limit NK Cell Effector Functions through Secretion of TGF-β and Engagement of CD85j. *J Immunol* 2018;200:1008–15.
33 Ardawi M-SM, Rouzi AA, Qari MH. Physical activity in relation to serum sclerostin, insulin-like growth factor-1, and bone turnover markers in healthy premenopausal women: a cross-sectional and a longitudinal study. *J Clin Endocrinol Metab* 2012;97:3691–9.
34 Curiel-Cervantes V, Solis-Sainz JC, Camacho-Barrón M, *et al*. Systematic training in master swimmer athletes increases serum insulin growth factor-1 and decreases myostatin and irisin levels. *Growth Factors* 2022;40:1–12.
35 Yargic MP, Torgutalp S, Akin S, *et al*. Acute long-distance trail running increases serum IL-6, IL-15, and Hsp72 levels. *Appl Physiol Nutr Metab* 2019;44:627–31.