Do Australian Football players have sensitive groins?

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Do Australian Football players have sensitive groins? Players with current groin pain exhibit mechanical hyperalgesia of the adductor tendon.

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ABSTRACT

Objectives This is the first study to evaluate the mechanical sensitivity, clinical classifications and prevalence of groin pain in Australian football players.

Design Case-control

Method Professional (n=66) and semi-professional (n=9) Australian football players with and without current or previous groin injuries were recruited. Diagnoses were mapped to the Doha Agreement taxonomy. Point and career prevalence of groin pain was calculated. Pressure pain thresholds (PPTs) were assessed at regional and distant sites using handheld pressure algometry across four sites bilaterally (adductor longus tendon, pubic bone, rectus femoris, tibialis anterior muscle). To assess the relationship between current groin pain and fixed effects of hyperalgesia of each site and a history of groin pain, a mixed-effect logistic regression model was utilised. Receiver Operator Characteristic (ROC) curve were determined for the model.

Results Point prevalence of groin pain in the preseason was 21.9% with a career prevalence of 44.8%. Adductor-related groin pain was the most prevalent classification in the pre-season period. Hyperalgesia was observed in the adductor longus tendon site in athletes with current groin pain (OR=16.27, 95%CI 1.86 to 142.02). The ROC area under the curve of the regression model was fair (AUC=0.76, 95%CI 0.54 to 0.83).

Conclusions Prevalence data indicates that groin pain is a larger issue than published incidence rates imply. Adductor-related groin pain is the most common diagnosis in pre-season in this population. This study has shown that hyperalgesia exists in Australian football players experiencing groin pain indicating the value of assessing mechanical pain sensitivity as a component of the clinical assessment.

Key Words: groin, athlete, pressure pain threshold, mechanical sensitivity, hyperalgesia, Australian football

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Introduction

The incidence of groin injuries in athletes has been reported between 2.6-3.6 new injuries per club per season in elite (AFL) Australian football players with recurrence rates ranging between 6% and 43%. However, the pre-season point prevalence and career prevalence of groin pain in these players has not been published nor has the described clinical classifications of groin pain aligned to the Doha Agreement Classifications of defined clinical entities: adductor-related, iliopsoas-related, inguinal-related and pubic-related groin pain; hip-related groin pain; and other causes.

Early identification of players at risk of injury would be valuable but it is currently unclear which objective parameters should be used for screening for susceptibility to groin injury. One factor could be an assessment of tissue sensitivity as this is both non-invasive and provides an objective estimate of the sensitivity of the pain system. Addressing alterations in sensory processing rather than the specific patho-anatomical hypotheses may be justified given the high prevalence rates of imaging findings in asymptomatic athletes and the frequent reporting of multiple diagnoses or entities.

In musculoskeletal pain of non-athletic populations, sensory processing changes have been observed leading to central nervous system sensitisation. The sensation of pain may be triggered by a noxious stimulus (such as an injury or overuse of the structures in the region) with either inhibitory or facilitatory effects observed through the modulation within the peripheral nerves, spinal cord, brain stem and cerebral cortex. Sensory hypersensitivity is capable of predicting future pain yet it is currently unknown whether sensory deficits exist in athletes experiencing groin pain. Palpatory tenderness of structures is commonly reported and precedes training capacity restrictions. Widespread increase in pain sensitivity has been demonstrated in patients suffering from chronic pain affecting the upper and lower limbs which has been associated with decreased function and extended periods of symptoms. Currently, pain sensitivity has not been reported in populations of patients suffering from groin pain which should be considered relevant as palpation is a commonly used and recommended clinical test which may be influenced via regional or widespread pain mechanisms. Longstanding groin pain has been associated with an enlarged pain area, affecting both the adductor...
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101 area and the lower abdominal wall.\textsuperscript{14} This may potentially be related multiple pathologies or due to
102 the large overlap of anatomical structures in the area\textsuperscript{15} but it may also be caused by an activation of
103 central pain mechanisms which has been shown to occur soon after the initial nociceptive stimulus\textsuperscript{16}.
104 It is plausible that sensitivity of pain mechanisms is an important factor in groin pain and that
105 widespread sensory changes may be predictive of treatment response which is worthy of further
106 investigation.
107
108 Mechanical sensitivity assessed by manual palpation is a common\textsuperscript{5,9} \textsuperscript{ENREF_5, ENREF_2} and
109 reliable\textsuperscript{17} diagnostic tool to detect the affected structures in clinical groin pain and there is evidence
110 suggesting that tenderness to manual palpation of groin structures precedes training capacity
111 reductions in AFL players\textsuperscript{10} \textsuperscript{ENREF_9} However, there are currently no reports of a standardized
112 assessment of mechanical sensitivity of the groin region in athletic populations. It is currently
113 unknown whether a previous injury in the musculoskeletal system leads to persistent sensory changes
114 in the affected area but such a relationship might potentially explain the high recurrence rate of
115 injuries observed particularly in the Australian football population.\textsuperscript{18} Emerging evidence highlights
116 that mechanical sensitivity improves with self-reported recovery over a three month period in other
117 musculoskeletal conditions such as patellofemoral pain syndrome.\textsuperscript{19} If this is the case, then the use of
118 standardized screening tools in athletic populations might be warranted for early identification of
119 players at risk and to track progression of the condition.
120
121 The aims of this study were to report the point prevalence and career prevalence as well as the
122 clinical classifications of groin pain aligned to the Doha Agreement taxonomy.\textsuperscript{2} Further aims of this
123 study were to examine two hypotheses concerning the pain sensory profile of an athletic population.
124 These were: (1) mechanical hyperalgesia exists in Australian Rules football players experiencing
125 groin pain (2) mechanical hyperalgesia of the adductor tendon or enthesis persists following
126 resolution of symptoms in Australian Rules football players experiencing groin pain.
127
128 Methods
129
130 \textsuperscript{Page 4 of 19}
This study conforms to the minimum reporting standards for clinical research on groin pain in athletes. Professional (n=66) and semi-professional (n=9) male Australian football players (age, 23.1±3.1years; height, 187.9±7.3cm; weight, 85.2±7.9kg) with and without current or previous groin injuries were recruited during the pre-season (2015) through two professional clubs in the Australian Football League (AFL). All players (n=90) were offered to participate with recruitment ceasing once the required sample size were reached. Exclusion criteria consisted of analgesic medication use either on enrolment or on a regular basis, current lower limb (other than groin pain), pelvic or lumbar injury that required medical treatment (medical attention injury) or resulted in time loss (competition loss injury) and surgery to these regions in the last 12 months. That is, any injury or medication which would alter the results of the sensory testing. Subjects were given a detailed written and verbal explanation of the experimental procedure prior to giving their written informed consent. The study was conducted in accordance with the Helsinki Declaration and was approved by the University of Newcastle Human Research Ethics Committee (H-2013-0052).

This study used a case-control design. A case was defined as the participant currently experiencing groin pain at the time of testing as reported by the participant, confirmed where appropriate with the medical staff of the club. Current groin pain was defined by self-reports of groin pain at the time of data collection, confirmed where appropriate with the medical staff of the club, and defined as currently seeking medical attention or a physical complaint resulting in restricted training or competition capacity due to pain experienced in the groin region. For the purposes of secondary analysis, participants who have a history of groin pain, as reported by the participant, confirmed where appropriate with the medical staff of the club, and defined as seeking medical attention or a physical complaint resulting in restricted training or competition capacity due to pain experienced in the groin region in their career were categorised. Control participants were defined as having an absence of “current groin pain” or a “history of groin pain”. All diagnoses were mapped to the Doha agreement\(^2\) classifications of groin pain; Clinical entities: adductor-, iliopsoas-, inguinal-, pubic-
related groin pain; Hip-related groin pain; and other causes of groin pain. A further category of “unknown” is reported where insufficient clinical information was available to accurately classify the pain. The Hip and Groin Outcome Score (HAGOS) was applied and is reported separately.\textsuperscript{21}

Pressure pain thresholds (PPTs) were assessed at regional and distant sites using a handheld pressure algometer (Somedic, Sweden) with a 1 cm\textsuperscript{2} probe using a 30 kPa/s ramp. The algometer was applied perpendicularly to the measurement site with the participant asked to press a button when the sensation turned from pressure to pain. This threshold is recorded in kilopascals (kPa). Pressure algometry has demonstrated good inter-rater and intra-rater reliability\textsuperscript{22,23} ($IC^2_{2,1}$ 0.91, 95\%CI 0.82-0.97), low standard error of measure\textsuperscript{22} (6.27N/cm\textsuperscript{2}, 95\%CI 5.35-7.59) and correlation with other measures of pain across all age groups.\textsuperscript{24} The algometer was calibrated prior to each testing period using the manufacturer’s instructions and equipment. Pressure pain thresholds were assessed across four sites bilaterally and based on an experimental pain model\textsuperscript{25} the adductor longus tendon site (AL) defined as 5 cm distal from the crease where the leg joins the pelvis, the rectus femoris site (RF), defined as the portion of the muscle that bisects the distance between the superior pole of the patella and the anterior superior iliac spine (ASIS), the anterior surface of the superior pubic body lateral to the pubic symphysis joint line where the adductor longus tendon coalesces with the pubic bone (PB) and the tibialis anterior (TA), the probe was placed in the mid-belly at the point measured as the proximal site 1/3 the distance from the lateral joint line of the knee to the inferior aspect of the lateral malleolus. Each measurement was recorded twice with the average of the measurements used for statistical analysis. The AL and PB sites were chosen given its relevance to clinical diagnostic tests of palpation. The RF site was chosen as it represents a body region close to the adductor site which is functionally and neuro-anatomically distinct from the adductors and the TA site was chosen as it is a common site for similar studies and represents a remote, unrelated site to the pelvic region with respects to function, anatomy and neutral systems. These sites have also been utilised in experimental
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pain studies of the groin region allowing future comparisons between clinical and experimental pain.25,26

Point prevalence was defined as currently having groin pain in the late preseason period (2015). Career prevalence was defined as ever having groin pain which has affected training or competition throughout their professional career including adolescence. Each prevalence type was calculated as the proportion of participants reporting groin pain during the pre-season period or throughout their entire senior career (Prevalence=number athletes reporting groin pain/total number of participants).

All data was assessed for normality. All statistical analyses were undertaken in Stata 13 (Stata 13 IC, StataCorp, USA). Significance was set at p<0.05 for all statistical tests. A priori power calculations utilising pilot data indicated that a minimum of 14 cases and 56 controls were required27 (α=0.05, β=0.20; PS Power and Sample Size Calculations, Version 3.0, 2009). To compare the PPTs between sites and sides, a single multivariate analysis of variance (MANOVA) with fixed factors of site and side was utilised. Odds ratios with associated attributable fractions in the exposed (AFE) and attributable fractions in the population (AFP) with 95% confidence intervals (using Fisher’s exact) of current groin pain and a history of groin pain were calculated using separate two-by-two tables to highlight the percentage of disease in the exposed group that can be attributed to mechanical hyperalgesia. The exposure variables pertained to hyperalgesia (increased pain from a stimulus that normally provokes pain).28 Hyperalgesia was defined as an asymmetry in PPT values between sides of greater than 10% for each site. This was confirmed post-hoc as the best cut-off using Receiver Operating Characteristic (ROC) curves which indicated a significant difference between asymmetry of 5% with 10% or greater (data not presented). ROC curves allow selection of the optimal diagnostic conditions through evaluation of the performance of a diagnostic test.29 A stepwise, backward, mixed-effect logistic regression model removing the least significant fixed effect was utilised to assess the relationship between current groin pain and fixed effects of hyperalgesia of each site (AL, PB, RF and TA) and a history of groin pain with random effects of the participant to account for within subject
correlation. Additionally, a ROC curve was used to run a sensitivity analysis on the significant model to assess the ability of the predictive model to discriminate between athletes experiencing groin pain and asymptomatic athletes at the time of testing. A non-parametric bootstrapped ROC curve was produced for the model, and the area under the curve (AUC) was calculated, with an AUC of 1 representing perfect discriminative power.

**Results**

Seventy-five professional and semi-professional Australian football players volunteered for this study with 16 cases (with current pain at the time of testing) and 57 without current pain. Seven of the current pain cases (43.75%) had a previous history of groin pain. Two subjects were excluded due to one sustaining a concussion the previous day and one having a car accident on the day of testing.

The point prevalence of groin pain in the preseason phase was 21.9% with career prevalence 44.8%.

Participants reporting current pain were mapped to the classifications of adductor-related (n=8, 50.0%) and psoas and adductor-related (n=1, 6.3%), hip-related (n=2, 12.6%) and undiagnosed (n=1, 6.3%) and unknown (n=2, 12.6%). Participants reporting a history of groin pain were mapped to the classifications hip-related (n=8, 24.4%; femoroacetabular surgery, n=6), adductor-related (n=7, 21.2%), pubic-related (n=3, 9.0%), inguinal-related (n=2, 6.1%; surgery, n=1), multiple classifications (n=2, 6.1%) and unknown (n=11, 33.3%).

The distributions (median, interquartile range) of the PPT values in the history free controls were: AL (755.0, 455.5-964.8), PB (732.3, 454.0-1005.8), RF (701.3, 310.8-1066) and TA (672.5, 325.5-1084.5). The random effect of participant was not significant (p>0.05) and therefore removed from all models. There was no significant difference between PPT values on each site (df=3, F=2.15, p=0.14) or side (df=1, F=1.03, p=0.38). A significant model of current groin pain was observed (p=0.03) with a significant fixed effect of hyperalgesia of the AL (OR=16.27, 95% CI 1.86 to 142.02) and non-significant fixed effects of hyperalgesia of the RF (OR=0.36, 0.09 to 1.48) and the TA (OR=3.89,
The area under the ROC curve for this model (Figure 1) was fair (AUC=0.76, 95% CI 0.54 to 0.83). The results of the two-by-two tables are presented in Table 1 for participants reporting current pain in the groin and indicate adductor longus tendon hyperalgesia is significantly related to current groin pain (OR=12.58, 95% CI 1.66 to 549.48). Participants who reported a history of groin pain but no current symptoms did not have increased odds of having hyperalgesia of the adductor tendon (OR=2.73, 95% CI 0.81 to 9.49, p=0.07). Post-hoc power calculations indicate that this study was not adequately powered (β=0.33) to detect a true odds ratio of 2.73 due to the sample size recruited for the analysis of the historical groin pain calculations. To test this hypothesis using the data presented, 75 cases and 75 controls would be required.

Discussion

This study is the first study to demonstrate that primary mechanical hyperalgesia of the proximal adductor longus tendon exists in Australian Football players currently experiencing groin pain. Furthermore, the attributable fraction in the participants with current groin pain was 92% indicating that mechanical hyperalgesia is a significant component of their presentation. This is novel as this is the first report of mechanical hypersensitivity in this population and differs from clinical diagnostic tests with respect to discrete sensory changes that occur in the presence of a noxious stimulus. This is also the first study to describe the clinical entities of groin pain in Australian football using the Doha Agreement taxonomy.² It indicates that during pre-season adductor-related groin pain represents the largest classifications (50%) with hip-related pathologies the highest reported classification in the historical pain group.
The point prevalence of groin pain (21.2%) in Australian football players during the preseason of which, 50% are classified as adductor-related, has not previously been reported. Career prevalence was 45% indicating that almost half of professional and semi-professional players report seeking treatment or reduce their capacity to train and compete due to groin pain throughout their career. This indicates that the burden of groin pain in Australian football may be underestimated by reports of incidence rates. Hip-related groin pain was the most prevalent cause of pain in the historical groin pain group. This may reflect the recent popularity of femoroacetabular impingement as a diagnosis in this population over the last decade.

Surprisingly, a history of groin pain was not a significant factor involved in players currently experiencing groin pain in any of the statistical models although seven players (44%) with current groin pain had historical groin pain. It is well established that previous groin injury is a predictor of future injury. The results of this study suggest that a history of injury may not be a variable associated with current pain if mechanical sensitivity is accounted for, instead suggesting that mechanical sensitivity after injury exhibits a stronger association than the occurrence of injury alone. Evidence from other bodily areas supports this claim although future research is warranted to investigate these hypotheses.

Investigations of the pathoanatomical explanations of groin pain in athletes have increased in the last decade. This study indicates the presence of altered sensation to mechanical stimuli on the adductor longus tendon which is similar to previous findings in athletes with patella tendinopathy and patellofemoral pain. In our study, we found that pressure pain thresholds at the adductor tendon were significantly affected by the presence of pain in the region. This is novel as we defined hyperalgesia as a deficit of 10% or greater compared with the non-affected side; a precision which is unable to be measured in manual palpation. The attributable fractions of AL hyperalgesia (92%) were surprising given that 50% of the diagnoses were mapped to the adductor-related classification. This
disproportionate result indicates that hyperalgesia of the AL contributes to the presentation across the
cases independent of clinical entity. No hyperalgesia was observed of the PB site. This is simply
hypothesised to be explained by the lack of cases mapped to pubic-related groin pain classification
and might also indicate differences in sensory processing between tendons and entheses. A further
hypothesis is that the discrimination between the sides is reduced due to the confluence of structures\(^\text{15}\)
and possible common somatosensory distribution.

The intent of this study was to investigate mechanical hyperalgesia as a defined sensory test rather
than clinical palpatory tenderness. The PPT data was modelled to determine whether hyperalgesia
could dichotomise Australian football players with and without groin pain. A significant model for the
identification of groin pain patients was observed with fair discriminatory power. This model has
approximately 75% discriminatory power interpretable as identifying three out of four players with
pain. Therefore assessment of mechanical pain sensitivity can be used to identify Australian football
players with groin pain or to identify those who share a profile similar to those in pain. The ability of
this model to predict players who are likely to develop groin pain was not examined in this study and
is an area of future research as tenderness on manual palpation has been shown to be predictive in this
population.\(^\text{10}\) It opens possibilities for trialling alternative therapeutic options more closely aligned to
non-sporting pain patients such as pain education, psychological therapy, desensitisation modalities,
and analgesic medications to alter the sensation.

Assessment of mechanical hyperalgesia of the adductor tendons should be integral to a comprehensive
clinical assessment along with distal anatomical sites; regional as well as a non-regional control site
such as tibialis anterior. Asymmetry of AL pressure pain thresholds greater than 10% should be
considered relevant to the patient and represents primary hyperalgesia of the structure. The results of
this study indicate no increased benefit from assessing the pubic bone (adductor enthesis) however,
this should be interpreted with caution as the majority of participants did not report entheseal/public-
related diagnoses. Interestingly, participants who reported a history of pain but did not have symptoms during testing did not display a statistically significant hyperalgesia of their adductor longus tendon (p=0.07) however, this could potentially represent Type II error (underpowered to detect small changes) and caution is recommended when interpreting this result.

Given pain is the major complaint of this population we defined _ENREF_7 current groin pain by the presence of pain in the region where the thigh joins the abdomen rather than on pathoanatomical grounds. Due to limitations in sample size subgrouping was not achievable without increasing the Type II error. Future studies should evaluate the effect of subgrouping into the Doha Agreement taxonomy^2 has on mechanical sensitivity of the region and furthermore whether these subgroups affect pain modulation. Further diagnostic studies are required to accurately be able to define the anatomical source of nociception given the high rate of `positive` imaging findings in asymptomatic populations. A reduced sample size in the analysis of hyperalgesia with respect to historical pain was utilised as we excluded participants with current pain in the groin as this alters the results of PPT measurement. This reduced the power of the study. Examining hyperalgesia in those with a history of groin pain but currently asymptomatic was the secondary aim of the study with sample size calculated for the primary aim of examining those with current pain. Future research should examine hyperalgesia and/or other deficits within the somatosensory system in asymptomatic athletes with a history of groin pain in a larger cohort (with 75 cases and 75 controls) as it could potentially be a component of pain recurrence.

Conclusion

The point prevalence and career prevalence of groin pain were 21.9% and 44.8% respectively. The most prevalent clinical classification of groin pain aligned to the Doha Agreement taxonomy was adductor-related groin pain. This study has shown that Australian Football players currently
experiencing groin pain have primary hyperalgesia of the adductor tendon. A significant model
utilising pressure pain thresholds was observed to have fair discriminatory power. Future studies
should examine other quantitative sensory testing parameters in this population as well as the value of
such measurements in terms of predicting the likelihood of injury and clinical progression toward
recovery.

Perspectives

- Adductor-related groin pain is the most prevalent diagnosis classification in pre-season
- Primary mechanical hyperalgesia exists in Australian Rules football players with current
groin pain
- Assessment of pressure pain thresholds has fair discriminatory power between Australian
Rules football players with and without current groin pain

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### Tables

Table 1 – Odds ratios, attributable fraction in the exposed and population for hyperalgesia at each muscle site and history of groin pain

<table>
<thead>
<tr>
<th>Explanatory Variable</th>
<th>Point Estimate</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adductor longus tendon hyperalgesia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>12.58</td>
<td>1.66 to 549.48&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Attributable Fraction in the Exposed</td>
<td>0.92</td>
<td>0.40 to 1.00&lt;sup&gt;†&lt;/sup&gt;</td>
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<tr>
<td>Attributable Fraction in the Population</td>
<td>0.86</td>
<td>-</td>
</tr>
<tr>
<td><strong>Pubic bone hyperalgesia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>1.19</td>
<td>0.32 to 4.99</td>
</tr>
<tr>
<td>Attributable Fraction in the Exposed</td>
<td>0.16</td>
<td>-2.11 to 0.80</td>
</tr>
<tr>
<td>Attributable Fraction in the Population</td>
<td>0.11</td>
<td>-</td>
</tr>
<tr>
<td><strong>Rectus femoris hyperalgesia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>0.95</td>
<td>0.24 to 3.41</td>
</tr>
<tr>
<td>Attributable Fraction in the Exposed</td>
<td>0.05</td>
<td>-2.41 to 0.75</td>
</tr>
<tr>
<td>Attributable Fraction in the Population</td>
<td>0.02</td>
<td>-</td>
</tr>
<tr>
<td><strong>Tibialis Anterior hyperalgesia</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>2.80</td>
<td>0.75 to 10.21</td>
</tr>
<tr>
<td>Attributable Fraction in the Exposed</td>
<td>0.64</td>
<td>-0.32 to 0.90</td>
</tr>
<tr>
<td>Attributable Fraction in the Population</td>
<td>0.32</td>
<td>-</td>
</tr>
<tr>
<td><strong>History of groin pain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Odds Ratio</td>
<td>1.00</td>
<td>0.27 to 3.49</td>
</tr>
<tr>
<td>Attributable Fraction in the Exposed</td>
<td>&lt;0.00</td>
<td>-0.49 to 0.73</td>
</tr>
<tr>
<td>Attributable Fraction in the Population</td>
<td>&lt;0.00</td>
<td>-</td>
</tr>
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95%CI, 95% confidence interval; <sup>a</sup>Statistically significant at the level of 0.05

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Figure 1

Receiver Operator Characteristic Curve for the significant model of current groin pain including fixed effects of hyperalgesia of the AL, RF and TA and random effects for participant.

Figure Legend

SD(area)=standard deviation of the area under the curve