INTRODUCTION

Inflammatory tendinopathy (tendonitis) is a common and often self-limiting disorder. However, sometimes the tendon will develop an area of chronic degeneration that does not heal rapidly (tendonosis). There are multiple factors that cause tendonosis (Astrom and Westlin, 1994; Leadbetter, 1992). Decreased blood supply is believed to play a role in the development of these disorders (Myerson and Biddinger, 1995). There are potent chemical factors in blood that can stimulate vascular ingrowth (Knighton et al., 1982; Pettet et al., 1996) and fibroblast activity (Arnoczky et al., 1988; Iyer et al., 1999). It is possible that injection of autologous blood can help stimulate a healing response in chronic tendon disorders. However, there are no known reliable animal models for chronic tendonosis (Backman et al., 1990; Leadbetter, 1989). This study is designed to assess the mechanical strength changes of normal rabbit tendon tissue to injected autologous blood and saline.

PROcedures

Adult post-breeding New Zealand white rabbits were used. In Series I, there were three groups: one control (N=7) and two test groups, six week (N=18) and 12 week (N=18). The test groups had 0.15 cc of autologous blood injected into the left patellar tendon and 0.15 cc of saline injected into the right patellar tendon. In the test groups, 13 specimens were randomly selected for mechanical testing and five for latex vascular injection and tissue clearing. In Series II, only the left tendon was injected. The right was used for control. Mechanical tests were performed on a Materials Testing System. The tendon strengths were then compared using two-tailed student t-tests.

RESULTS AND DISCUSSION

In Series I, we found at six weeks, the patellar tendon strength after injection with blood showed no statistical difference compared to the control group or the saline injection side (Table 1). At 12 weeks, both the saline and the blood injected tendons showed increased strength (p>0.001) compared to controls. However, there was no significant difference between the blood and saline injection groups. In Series II, there was no significant increase in strength at 6 weeks (Table 2). At 12 weeks, the blood injected left was increased significantly compared to the untreated right.

Surgical intervention for chronic tendinosis may have the benefit of direct debridement of damaged tissue (Organ et al., 1997). However, some surgical procedures, such as longitudinal tenotomy, are aimed at trying to stimulate a healing neovascular response without tissue debridement (Maffulli et al., 1997). While not exposing the patient to surgery, there are two mechanisms by which
Injecting blood may benefit a chronic, poorly healing tendon lesion. Blood clot stimulated new vascular ingrowth and fibrocyte activity (Knighton et al., 1982) and there is growing interest in the use of growth factors in promoting fibrous tissue healing. In this study, we found that saline injections alone caused significant increase in strength of the patellar tendon at three months. We speculate this is due to local tissue disruption and bleeding that stimulated a healing response. However, this increase was not as statistically significant as with the injection of blood. In Series I, some of the increased strength may be related to growth of the rabbits during the study. Series II controlled for this variable by using a matched untreated tendon to compare. No ill effects from the injection of tendons with autologous blood were noted. We found that the strength of patellar tendon complex at six weeks after treatments was not compromised by the procedure and at 12 weeks was significantly increased by 15% (Series II) to 27% (Series I).

**SUMMARY**

We found, in two different series, that 12 weeks after the injection of blood, the mechanical strength of rabbit patellar tendons significantly increased. In Series I, the increase was 27%. In Series II the increase was 15%. In this animal model, there were no complications to the injections.

**REFERENCES**


Table 1: Summary Series I Structural Properties of the Patella-Patellar Tendon-Tibia Complexes

*Statistically significant difference from control (p<0.05)*

<table>
<thead>
<tr>
<th>Property</th>
<th>Control</th>
<th>6 wk Rt (saline)</th>
<th>6 wk Lt (blood)</th>
<th>12 wk Rt (saline)</th>
<th>12 wk Lt (blood)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>14</td>
<td>13</td>
<td>13</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>Strength (N)</td>
<td>649.6(±109)</td>
<td>649.7(±107)</td>
<td>698.5 (±115)</td>
<td><em>801</em> (±61)</td>
<td><em>824</em> (±79)</td>
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<tr>
<td>Stiffness (N/mm)</td>
<td>173(±33)</td>
<td>168(±26)</td>
<td>164(±18)</td>
<td>160(±23)</td>
<td>154(±24)</td>
</tr>
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</table>

Table 2: Summary Series II Structural Properties of the Patella-Patellar Tendon-Tibia Complexes

*Statistically significant difference from control (p<0.05)*

<table>
<thead>
<tr>
<th>Property</th>
<th>6 WEEKS (N=10)</th>
<th>12 WEEKS (N=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rt (Control)</td>
<td>Lt (Blood)</td>
</tr>
<tr>
<td>Strength (N)</td>
<td>701(±146)</td>
<td>734(±115)</td>
</tr>
<tr>
<td>Stiffness (N/mm)</td>
<td>177(±23)</td>
<td>193(±21)</td>
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