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ABO Blood Groups and Achilles Tendon Injury

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1. Introduction

The incidence of Achilles tendon rupture has significantly increased over the last 20 years (Moller, 1996; Young and Maffulli, 2007), achieving, within the tendon diseases, an incidence between 6 and 18% (Rees et al., 2006; Mazzone and McCue, 2002; Shepsis et al., 2002). The risk of the Achilles tendon rupture is greater in the male population with a ratio included between 1.7 : 1 and 30 : 1. This data would be justified by the high prevalence of males subjects in sports considered at risk (Rees et al., 2006; Mazzone and McCue). Despite its high incidence in the context of sports traumatology, the Achilles tendon rupture etiology remains poorly known (Maffulli, 1999; Williams, 1986) and it is essentially based on two main theories: the “degenerative theory” and the “mechanical theory”.

The “degenerative theory

From a biomechanical point of view, the “degenerative theory” is based on the assumption that a structurally intact tendon, even if subjected to significant tensile forces - however remaining within the physiological request - should not be subject to rupture (Cetti and Christensen, 1983). In fact, since 1959 (Arnero and Lindhom, 1959), we can find in bibliography several studies showing that the majority of patients undergoing Achilles tendon surgical repair, already had rather advanced degenerative processes that were considered responsible for the tendon structural failure (Davidsson and Salo, 1969; Fox et al 1975; Kannus and Jozsa, 1991; Jozsa et al., 1991; Jarvinen, 1997; Jozsa and Kannus, 1997; Waterston, 1997; Waterston and Maffulli, 1997). Although most of these degenerative involutions were not linked to a precise etiologic cause, the majority of the Authors linked these to an alteration in tendon microcirculation subsequent to hypoxia and altered metabolism (Jarvinen, 1997; Jozsa and Kannus, 1997; Waterston, 1997; Waterston and Maffulli, 1997). Others Authors noted that the degenerated tendon tissue showed an increased production of collagen type III and V that disturbs the normal architecture of the tendon tissue making it less resistant against mechanical stress (Waterston, 1997; Waterston et al., 1997).

The mechanical theory

Some Authors showed that the Achilles tendon structural failure can occur even if it is subjected to mechanical stress that is within a normal physiological situation but in a situation where the tendon was, or may be, subject to a series of cumulative microtraumatic injuries without having allowed a reasonable time for the biological repair (Knörzer et al., 1986; Selvanetti et al., 1997). Therefore, the complete rupture would be the result of a several

number of previous multiple microtraumatic injuries which would lead the tendon just to the point of its structural failure (Knörzer et al., 1986). In such conditions, the situation of greater risk for the tendon integrity would occur when they are present within the same moment three very specific biomechanical factors (Barfred, 1971; Hess et al., 1989).).

- The tendon is obliquely loaded in relation to its anatomical axis.
- The gastrocnemius-soleus muscle complex is in maximal contraction.
- The initial tendon length is reduced.

We can find this combination of factors as a usual situation almost in all sports requiring a "push off" quick action (Selvanetti et al., 1997). The mechanical theory could, at least in part explain the occurrence of the Achilles tendon complete rupture as this event is sometimes observed even in the absence of degenerative processes (Knörzer et al., 1986; Clement et al., 1994). In according with this situation it is interesting to note that some Authors have suggested a relationship between the musculoskeletal disorders and in particular, tendon disorders and subjects' blood group (Mourant et al., 1978; Lourie, 1983; Joza et al., 1989; Kujala et al., 1992). The scientific rationale for this correlation is based on the fact that subjects belonging to 0 blood groups show an N-acetylgalactosamine transferase activity higher compared to subjects belonging to the A and B group (Kujala et al., 1992). A greater N-acetylgalactosamine transferase activity leads to a collagen type III increased production within the tendon (Waterston and Maffulli, 1997). The type III collagen is less resistant against mechanical stress compared to type I collagen (Waterston and Maffulli, 1997) and would predispose the tendon to spontaneous rupture (Joza et al., 1989; Maffulli, 1999). This may explain the high incidence of Achilles tendon spontaneous ruptures found by some Authors in subjects belonging to the 0 blood group compared to the A and B groups (Joza et al., 1989; Maffulli, 1999).

In addition to these two main theories, the Achilles tendon rupture may result from other different factors that we can assume as follows:

2. The iatrogenic damage

The use of corticosteroids

The use of corticosteroids are widely used therapeutic practice in a various number of diseases, however their use represents a risk factor for tendon rupture (Fisher, 2004) In literature some Authors report clear associations between rupture of the Achilles tendon and oral assumption and tendinous or peritendinous injections of corticosteroids (Unverferth and Olix, 1973; Newnham et al., 1991). The recommendation to not use corticosteroids in tendinopathy is based over two main points:

- i. Their potential danger against the integrity of the tendon structure (Speed, 2001).
- ii. An insufficient evidence to justify their use in tendinopathy (Maffulli and Kader, 2002).

The use of fluoroquinolone

The use of some antibiotics such as fluoroquinolone is associated in literature with injury and / or to serious tendon ruptures, especially at Achilles tendon level (Huston, 1994; Royer et al., 1994; Filippucci et al., 2003 ; Vanek et al., 2003). The first Achilles tendon rupture due to the use of fluoroquinolone was described in 1992 (Ribard et al., 1992). The fluoroquinolone are drugs widely used in clinical practice but their association with

corticosteroid drugs, especially in the elderly subjects, is an important risk factor for tendinopathy and/or tendon rupture (Linden Van Der et al., 2003). The most important number of tendon damages caused by fluoroquinolone reported in the literature is caused by the use of ciprofloxacin followed by enoxacin, ofloxacin and enorfloxacin (Huston, 1994; Szarfman et al., 1995; Van der Linden et al., 2002; Filippucci et al., 2003). The fluoroquinolone-induced tendon degeneration would be due to the destruction of the extra cellular matrix, to the inhibition of tenocytes proliferation and to the reduction of collagen synthesis (Szarfman et al., 1995; Corps et al., 2003).

3. The intrinsic factors

The intrinsic factors, otherwise defined as endogenous factors, which may give rise to Achilles tendinopathy mainly concern paramorphisms or particular postural aspects that may lead to a functional overload at Achilles tendon level compromising its functionality and structure.

Among these we can mention:

The iper-pronation of the hindfoot associated or not to flat-foot (Ryan et al., 2009; Wyndow et al., 2010).

A varus forefoot associated or not to valgus hindfoot (Ajis et al, 2005)

A flat-supinated foot (Ryan et al., 2009; Wyndow et al., 2010).

A retrocalcaneal spur (Kearney and Costa, 2010)

An Haglund's deformity (Min et al., 2010)

A lower limb asymmetry (Kannus, 1997).

A limitation in ankle dorsiflexion (Kaufman et al., 1999).

A limitation of the subtalar joint mobility (Kvist, 1991).

A poor muscle strength of lower limbs in general and particularly of the calf muscles. In fact, a muscle with a stamina few level cannot effectively protects the tendon structure (Kannus, 1997).

The Dehydration (Hestin et al., 1993; Schwellnus et al., 1997; Gottschalk and Andrish, 2011).

The hyperlipidemia (Mathiak et al., 1999; Ozgurtas et al., 2003).

The hyperuricemia (Dodds e Burry, 1984).

The tendon temperature increase resulting from sporting activities (Wilson e Goodship, 1994).

Changes in the genes expression regulating the "cell-cell" and the "cell-matrix" interaction, associated to a matrix metal proteinase 3 (MMP-3) down-regulation and to a metal proteinase 2 (MMP-2) and Vascular Endothelien Growth Factor (VEGF). up-regulation (Ajis and Maffulli, 2007).

4. The extrinsic factor

In several sports, especially in those where its expected the run and/or the jumps, the injuries and / or the Achilles tendon tears are often associated with errors in the training planning (Hess et al., 1989; Clain and Baxter, 1992; Jozsa and Kannus, 1997; Mahieu et al., 2006). One of the most important causes seems represented by overtraining (Clement e coll, 1984), then follow the changes and / or the increasing in the training program carried out without an appropriate adaptation period (Kvist, 1991; Kwist, 1994; Järvinen et al., 2001) and a lack of specific athletic skill (James e coll., 1978). Others extrinsic factors that may give rise to Achilles tendinopathy that we must remember are: fatigue, poor technique, poor equipment and environment conditions (temperature, humidity, altitude, wind) (Young and Maffulli, 2007).

The aim of this study is to verify, through a retrospective analysis, conducted in an active sports population, the incidence of both Achilles tendons spontaneous ruptures and tendinopathy in relationship to the blood group.

5. Subjects

We considered two different groups composed by 45 caucasian patients belonging to the Northern Italy population whose age, weight and height was respectively 32 ± 7 years, $78 \pm 6,7$ kg and $178 \pm 5,5$ cm. In the first group (G1), all subjects attended our medical center complaining of Achilles tendon pain or following a surgery of scarification or open tenorrhaphy at this level. G1 patients practiced sport activity at professional or amateur level. 24 patients (53,3% of the subjects) followed a conservative program for Achilles tendinopathy, while the two groups of patients surgically treated 15 subjects (equal to 33,3%), with open tenorrhaphy and 6 subjects, (13,3%) with tendon scarification) were following the specific post-surgery rehabilitation program. In the second group (G2) all subjects. practiced a sport activity at professional or amateur level but none of them had never suffered for tendinopathy during his career. All the subjects were informed of the aim of the study and gave their written consent for the data processing.

6. Protocols and methods

All subjects were required to produce an official document in which their blood group was annotated. The clinical diagnosis of Achilles tendinopathy, formulated for the 20 patients who had not undergone surgical treatment, was confirmed by ultrasonography performed in accordance with the criteria in the following form (Annex A). Both clinical and imaging criteria were adopted in accordance with the protocol already used by the Research Unit for Exercise Science and Sports Medicine, University of Cape Town (Schepesis et al., 2002; Alison et al., 2007; Collins et al., 2010). The clinical criteria allowing us to formulate diagnosis of Achilles tendinopathy were:

- Gradual progressive pain over the Achilles tendon area > 6 weeks
- Early morning pain
- Early morning stiffness
- History of swelling over the Achilles tendon area
- Tenderness to palpation over the Achilles tendon
- Palpable nodular thickening over the affected Achilles
- Positive "shift" test (movement of the nodular area with plantar dorsi-flexion).

7. Statistic

For all considered variables the usual statistical indices (average and standard deviation) were calculated. The normality of data distributions was checked with a Kolmogorov-Smirnov test. The difference between the recorded values and the expected values was calculated by a Chi Square Test, the statistic significance was posed to $p < 0.05$.

8. Results

G1 group

30 subjects (66.6%) belonged to group 0. 19 of these 30 subjects (equal to 63.3%) had undergone a surgical tenorrhaphy, 7 subjects (equal to 23.3%) had undergone a surgical scarification and the remaining 4 subjects (equal to 13.3%) had undergone a conservative treatment.

8 subjects, equal to 17.7%, belonged to group A. 6 of these 8 subjects (equal to 75%) had undergone a surgical tenorrhaphy and the remaining 2 subjects (equal to 25%) had undergone a surgical scarification.

4 subjects equal to 8.8% belonged to B group. 2 of these 4 subjects (equal to 50%) had undergone a surgical tenorrhaphy while the remaining 2 subjects (equal to the 50%) had undergone a conservative treatment.

3 subjects equal to the 6.6% belonged to the group AB. All these subjects had undergone to a surgical scarification.

All subjects were male.

G2 group

18 subjects (equal to 40%) belonged to group 0.

20 subjects (equal to 44.4%) belonged to the A group.

5 subjects (equal to 11.1%) belonged to the B group.

2 subjects (equal to 4.4%) belonged to the AB group

All subjects were male.

The difference between the subjects belonged to group 0 in G1 and G2 group was statistically significant ($p < 0.005$).

The difference between the subjects belonged to group A in G1 and G2 group was statistically significant ($p < 0.01$).

The difference between the subjects belonged to group B in G1 and G2 group was not statistically significant.

The difference between the subjects belonged to group AB in G1 and G2 group was not statistically significant ($p < 0.01$).

9. Discussion

Already in the second half of the last century (Aird et al., 1953) several works showing an association between the blood groups and different types of pathology were published. In

this context, we can mention the relationship between blood groups and hip primary osteoarthritis (Lourie, 1983), acute hematogenous osteomyelitis (Eid, 1985), tendon ruptures (Jozsa et al., 1989; Kujala et al., 1992; Mafuli et al., 2000), nail-patella syndrome (Renwich and Lawler, 1955), spondylolisthesis (Wynne-Davies and Scott., 1979), delay in bone healing after fracture (Kujala et al., 1992), just mentioning some studies in a orthopedics context. The hypothesis in patients suffering from generalized tendinopathy (that may include rotator cuff pathology, epicondylopathy, carpal tunnel syndrome, triggering of the long finger flexor tendons and extensor tendon pathology such as de Quervain's disease) of a "mesenchymal syndrome", theorizing about a possible genetic component that could cause an abnormal formation of collagen was advanced by Nirshil (Nirshil, 1969). He was comforted in his theory by the fact that in this group, normal routine rheumatologic tests were normal. In general tendinopathy context, Achilles tendon has a particularly important aspect. Achilles tendinopathy and ruptures are increasing among the non-sporting population as well (Young and Maffulli., 2007). In this regard, several etiological hypotheses were formulated (Knörzer et al., 1986; Jozsa et al., 1989; Selvanetti et al., 1997; Maffulli, 1999; Vanek et al., 2003) but in any case, the etiology remains multifactorial (Bestwick and Maffulli, 2000; Wilson and Goodship, 1994).

From an anatomical point of view, as in any case, it is important to remember how the Achilles tendon received the blood supply. The tendon vascularity comes from two arteries which run longitudinally to the tendon belly. A part of its blood supply comes from vessels running in the paratenon that are mainly derived from the posterior tibial artery. An additional supply in the proximal part of the tendon is furnished by the muscle bellies that continues into the tendon via the endotenon, (in any case this contribution is not significant). Finally, the distal region receives vessels from an arterial periosteal plexus on the posterior aspect of the calcaneus. The Achilles tendon has, in its downward course, a kind of anatomical twist starting above the point at which is the fusion with the solearis tendon portion. This twist is much more evident as less is important the solearis fusion (Jozsa e Kannus, 1997). This twist causes the emergence of a stress area resulting in a lesser blood flow, displaced about 2-6 centimeters above the tendon heel insertion. For this reason, this area is the site most commonly encountered for pathological risk.

From a structural point of view, the Achilles tendon shows a tenocytes and tenoblasts percentage equal approximately to 90-95% of its cellular elements. Collagen and elastin are present at levels of about 70 and 2% respectively of the dry weight of the tendon structure and form the most part of the extracellular matrix. The collagen production in the tendon can be affected by many factors, such as the inheritance, the diet, the nerve supply, besides genetic and hormonal factors (Viidik., 1973). Corticosteroids inhibit the new collagen growth while insulin, estrogen and testosterone favor it (O'Brien, 2005). Even the mechanical stimulus related to physical exercise promotes the new collagen synthesis, the increase in the myofibrils number and size and the metabolic enzymes concentration. This will result in increasing the tendon tensile strength (O'Brien, 2005). The Achilles tendon has a high capacity to withstand the tensional forces created by the movements. In vivo peak force of the Achilles tendon has been measured at more than 2.200 Newtons (Fukashiro et al., 1995), namely in the order of 50-100 N/mm (Waggett et al., 1998). However, Achilles tendon transmits forces that are approximately seven times the subject body weight during activity such as running, this represents a huge increase on the forces that normally act during standing which are about half the body weight (Ker et al., 1987). When the Achilles

tendon is subjected to tensile stress its wavy configuration disappears and its collagen fibers respond in linear manner to increasing load and a strain level greater than 8% leads to macroscopic rupture due to tensile failure of the tendon collagen fibers (O'Brien, 1992)

Collagen type most represented is the type I, with a rate of approximately 95% (Kastelic et al., 1978; Maffulli and Benazzo, 2000). Type I collagen fibrils are grouped into fibers, fiber bundles, and fascicles, so that the Achilles tendon is a very similar to a multistranded cable. However, Achilles tendons which have undergone a complete rupture, show an increase of type III and type V collagen which are known to be less resistant against tensile stress (Coombs et al., 1980; Wren et al., 2000; Koob and Vogel, 1987; Benjamin and Ralphs, 1998). In fact, the type III collagen is mainly present in the tendon during the healing process (Józsa e Kannus, 1997; Myerson and McGarvey, 1999) while type V collagen increases with the tendon aging process and is associated with a decrease in diameter of tendon fibers as well with a decrease in its mechanical properties (Dressler et al., 2002; Goncalves-Neto et al., 2002). In fact, some studies have shown that the tenocytes, following a damaging and / or degenerative event, show an increase in type III and V collagen production that perturb the tendon tissue architecture decreasing its stress resistance (Waterston e coll., 1997). Also other Authors underline that an important proportion of type I collagen increases the strength of the tendon structure (Butler e coll., 1978) and that the minor structural strength of type III and V collagen compared with type I collagen is attributable to the fact that the first two types (III and V) have fewer crosslink than the second one (I) (Józsa e coll., 1990). In addition, some studies indicate an inverse correlation between the type III collagen reactivity and diameter, and thus the structural strength, of tendon fibers (Birk e Maine, 1997). So, a repeated micro-injuries history to the Achilles tendon level may lead to the formation of a hypertrophic tendon tissue which is biologically weaker and which also tends to replace the normal tissue.

In fact, during the normal physiological activity, some microscopic breaks occur in the tendon substance. They are continually remodeled by the formation of new collagen. Obviously, this destruction-remodeling process is particularly emphasized in some sports activities, as for example the run, that strongly stresses the tendon structure (Kirkendall, 1997; Selvanetti et al., 1997). Subjects belonging to blood group 0 shows a N-acetylgalactosamine transferase activity much higher than in subjects in A and B group (Kujala e coll., 1992). This increased N-acetylgalactosamine transferase activity would be result in an increase in type III collagen production within the tendon.

Since the type III collagen shows less resistance against mechanical stress compared to type I collagen (Waterston e Maffulli, 1997), its abnormal proliferation may expose to tendinopathy, which could end in tendon spontaneous rupture (Joza et al., 1989; Maffulli, 1999; Maffulli et al., 2000). Is also interesting to note that recently a type III collagen abnormal proliferation and a decreasing in type I collagen are always present in the Achilles tendon calcific insertional tendinopathy (Maffulli et al., 2010). Interesting to note that, since the ABO gene encodes for transferases, some studies have suggested that the different enzymes produced by the ABO gene, could determine not only the glycoprotein antigens structure determination on the red blood cells, but also some types of glycoproteins found within the ground tendons substance (Jozsa et al., 1989). Other studies have proposed that other genes, closely linked to the ABO gene on the tip of the long arm of chromosome 9q32-q34, which encode for components of the extracellular matrix, are more likely associated with Achilles tendon pathology (.Kannus and Natri, 1997; Kujala et al., 1992).

Two of these genes, tenascin-C gene and COL5A1, encode for structural components of tendons (Mokone et al., 2005). The COL5A1 gene encodes for the pro alpha1 (V) collagen chain, found in most of the isoforms of type V collagen (Birk, 2001; Silver et al., 2003).

In literature, we can find other examples concerning other pathologies, such as the nail-patella syndrome, where the LMX1B gene, is closely linked to the ABO gene, encoded for a protein responsible for the pathology (Bongers et al. 2002).

However, as already suggested by other Authors (Adam and Maffulli, 2007), it is highly unlikely that a single gene, or a group of genes, are exclusively associated with the development of the symptoms of Achilles tendon injury. In fact, it's more probable that this condition is polygenic, and other genes that encode for important structural components of tendons are also associated with Achilles tendon injury.

In any case, the above could explain the high incidence of the Achilles tendon spontaneous ruptures found by some Authors in individuals belonging to blood group 0 than in A and B group (Joza e al., 1989; Maffulli, 1999). Furthermore, it is interesting to underline that the type III and V collagen high percentage would may explain the occurrence of a spontaneous rupture in tendons that does not show previous degenerative processes.

In our subjects, the group 0 most found frequency (66.6% $p < 0.005$) confirms the hypothesis of an increased susceptibility of individuals belonging to this group in developing tendinopathy at Achilles tendon level and would agree with previous results findings by others Authors (Joza e al., 1989; Maffulli, 1999). Obviously, the limited sample that we considered in our study does not allow us to affirm with certainty this hypothesis which should be confirmed by further studies to be carried out on a larger population. For this reason the results of our study must be interpreted with the greatest attention taking into account this limitation.

In any case, this finding seems particularly interesting in a sporting context, especially concerning its possible use as a preventive measure. In fact, our study may suggest the importance for athletes belonging to 0 group, that practice a sporting activity, such as football, athletics, jogging, running, and/or at least all the other sporting activities which strongly encourage stress to the Achilles tendon, to carry out a regular preventive program based primarily on increasing the Achilles tendon eccentric strength (Allison and Purdam, 2009; Gärdin et al., 2009).

In conclusion, during a sporting activity there is a high incidence of tendon injuries, however, the exact etiology of this condition is not yet fully understood. Some studies, to which our study is a part of, suggest that the genetic component can play an important role; so given the interest and the importance of these problems, we hope that in the future, others studies will further clarify and deepen this exciting topic.

10. Appendix A

Soft tissue diagnostic ultrasound examination of the Achilles tendon

Right Achilles tendon:

Tendon:

- Shape:

- Angular: —
- Fusiform: —
- Margin:
 - Sharply defined —
 - Poorly defined —
- Contour:
 - Smooth —
 - Nodular —
 - Tapered —
- Max. diameter:
 - AP: — mm
 - TRV: — mm
- Internal architecture
 - Organised —
 - Central hypoechoic foci (med/lat/ant/post) —
 - Disrupted fibres (mild/mod/severe) —
 - Haematoma —
 - Calcification —
 - Acoustic shadowing —
- Power Doppler vascularity
 - Absent —
 - Present —
 - Prominent —

Paratenon

- Fluid:
 - Absent —
 - Present
 - Site —
 - Amount —
- Soft tissue swelling
 - Absent —
 - Present —
- Power Doppler Vascularity
 - Absent —
 - Present —
 - Increased —
- Retrocalcaneal Bursa
 - Normal —
 - Fluid filled —
- Kager’s fat
 - Normal —
 - Hyperechogenic —

Myotendinous junction

- Normal —

- Tear —

Calcaneus

- Normal —
- Abnormal —

Left Achilles tendon:

Tendon:

- Shape:
 - Angular: —
 - Fusiform: —
- Margin:
 - Sharply defined —
 - Poorly defined —
- Contour:
 - Smooth —
 - Nodular —
 - Tapered —
- Max. diameter:
 - AP: — mm
 - TRV: — mm
- Internal architecture
 - Organised —
 - Central hypoechoic foci (med/lat/ant/post) —
 - Disrupted fibres (mild/mod/severe) —
 - Haematoma —
 - Calcification —
 - Acoustic shadowing —
- Power Doppler vascularity
 - Absent —
 - Present —
 - Prominent —

Paratenon

- Fluid:
 - Absent —
 - Present
 - Site —
 - Amount —
- Soft tissue swelling
 - Absent —
 - Present —
- Power Doppler Vascularity
 - Absent —

- Present —
- Increased —
- Retrocalcaneal Bursa
 - Normal —
 - Fluid filled —
- Kager’s fat
 - Normal —
 - Hyperechogenic —

Myotendinous junction

- Normal —
- Tear —

Calcaneus

- Normal —
- Abnormal —

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Achilles tendon has always attracted a great attention. Its disorders include various problems from pain and swelling with bumps to functional impairment or even ruptures. Debates concerning aetiology and optimal treatment are still going on. A lot of efforts and research have already been put on to find the answers to unsolved problems and this book is an attempt to share (some of) these findings to the readers. If only one of the papers helps the therapists or patients in understanding and solving their problems, we will consider that the mission of the book was accomplished.

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