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Free nerve endings in the ligamentum capitis femoris

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ABSTRACT – We report the presence of free nerve endings (FNE) in the ligamentum capitis femoris (LCF). Qualitative and quantitative measurements on the incidence of FNE, as assessed by immuno-histochemistry for the S-100 protein, were obtained from 18 patients undergoing hip surgery. We found FNE in all LCF, with no association to age. The presence of FNE in the LCF suggests a role in noci-/proprioception of the hip.

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The ligamentum capitis femoris (LCF), also known as the ligamentum teres or round ligament, has been shown in the intrauterine hip to be involved in joint development (Gardner and Gray 1950) and to provide blood vessels to the femoral head (Trueta 1957), while its role in adulthood is largely unknown. Recently, arthroscopy performed in painful hips without diagnosis has revealed structural damage such as partial/complete tears or degeneration of the ligament (Gray and Villar 1997) suggesting a role of LCF in joint nociception.

So far, no information is available as to whether the LCF has the neuro-morphological structures required for noci- or proprioception. Therefore, we studied the neurogenic structures in LCF with immunohistochemistry.

Patients and methods

During 1999, we excised the LCF intraoperatively in 18 patients, median age 38 (9–94) years, undergoing hip surgery for various reasons. None of them had severe coxarthrosis (cases 17 and 18) (Table).

Demographic data for FNE assessment

Case	Age	Side	Gender	Diagnosis	FNE
1	94	L	F	Femoral neck fracture	11
2	92	R	F	Femoral neck fracture	26
3	86	L	M	Femoral neck fracture	5
4	82	R	F	Femoral neck fracture	23
5	76	L	F	Femoral neck fracture	13
6	74	L	M	Femoral neck fracture	11
7	49	L	M	Prearthrosis	48
8	45	L	M	Prearthrosis	17
9	41	L	F	Prearthrosis	23
10	35	R	M	Pipkin II fracture	10
11	30	L	M	Acetabular fracture	11
12	27	R	M	Prearthrosis	9
13	25	R	M	Acetabular fracture	20
14	23	R	F	AVN in SCFE	28
15	18	R	M	Prearthrosis	27
16	18	R	F	DDH and prearthrosis	13
17	9	L	M	Perthes' disease	54
18	9	R	M	Postcoxitis	48

FNE free nerve endings per 50 mm^2,
AVN avascular necrosis,
SCFE slipped capital femoral epiphysis,
DDH developmental dysplasia hip.

The LCF was sharply dissected from the femoral head and acetabular notch. To allow the pathologist to identify the orientation of the ligament correctly, it was marked by a suture. After removal, it was fixed in 4% formaldehyde solution and then embedded in paraffin. Each ligament was cut through its center into longitudinal slices with a thickness of 3 μm. They were stained with hematoxylin and eosin, and immunohistochemistry using the polyclonal antibody against the S-100 protein (Dako, Glostrup, Denmark) (Figure 1). This is an acidic, dimeric calcium-binding protein (molecular weight: 21,000 kD), composed of various combinations of alpha- and beta-subunits, which was first isolated in the central nervous system.

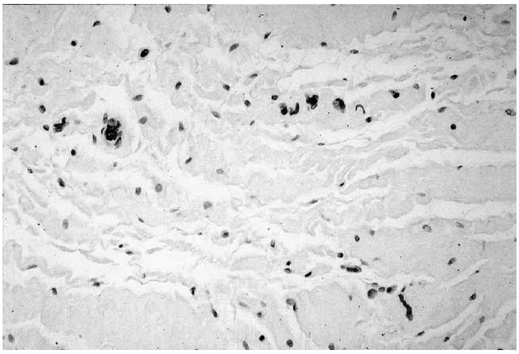

Figure 1. Central portion of a LCF stained by H&E and antibodies against the S-100 protein showing several FNE (red).

Using light microscopy, we determined in 2–4 sections of each LCF sample the the presence of specific receptor organs (types I–III) or FNE (type IVa), classified according to their shape, function (Freeman and Wyke 1967, Schimek 1985) and labeling by the S-100 protein (Biedert et al. 1992). Type I (Ruffini) mechanoreceptors are globular in shape (slow adapting-static and dynamic stress loads), type II (Pacini) are cylindrical (rapidly reacting-acceleration receptors), type III (Golgi) are fusiform (very slow reacting-inhibitory reaction), and type IVa are unmyelinated FNE transmitting information about pain and inflammation (Kennedy et al. 1982).

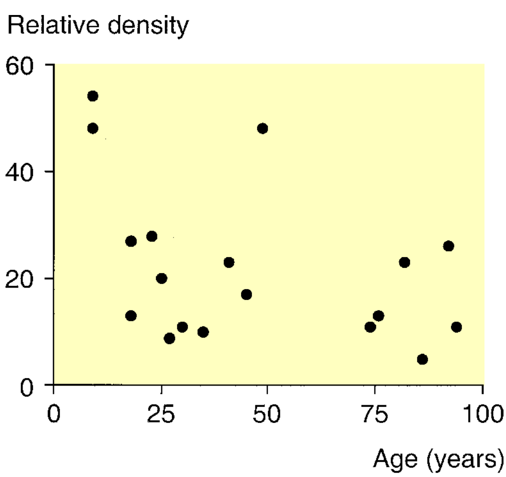

Figure 2. Relative density of FNE (per 50 mm^2) as a function of the patient's age. No significant correlation was found between both parameters.

We counted the neurogenic structures in a given section (in mm^2) and calculated the relative density in a standardized area unit of 50 mm^2. Presented values are the average of 2–4 quantified sections of each LCF sample. We used the non-parametric Spearman rank correlation test for the statistical analysis.

The study was approved by our Ethics Committee.

Results

We found FNE (type IVa) in all LCF (Figure 2) with a frequency ranging between 5 and 54 per 50 mm^2 (Table). Although the highest FNE count was found in a 9-year-old boy and the lowest FNE count in an 86-year-old man, there was no significant age-correlation of FNE counts (Spearman rank correlation $p = 0.08$). In none of the specimens could other specific receptor organs (types I–III) be identified.

Discussion

We found free nerve endings (type IVa) in all 18 ligamentum capitis femoris and the counts were higher than those reported for the anterior cruciate ligament or iliotibial tract (Biedert et al. 1992). FNE are afferent sensory organs, found in most connective tissues (Zimny 1988), which transmit mechanically- and/or chemically-triggered information about pain and inflammation (Kennedy et al. 1982). Since FNE (type IVa) are intraarticular nociceptive receptors, they represent, in addition to the arthrostatic (type I activity and type III inhibition) and arthrokinetic (type II activity and type III inhibition), the third group of reflexogenic function (Schimek 1985). Therefore, in addition to its mechanical and structural functions, the LCF may be involved in transmitting specific somatosensory afferent signals to the spinal and cerebral regulatory systems.

Since the adult hip resembles a well-constrained socket joint, the LCF may be part of an integral reflex system involved in joint protection, acting as a rein avoiding excessive motion potentially harmful to the joint. The distended LCF may give afferent signals to inhibit further joint excursion by reflectory muscular action. The presence of nerve endings in the acetabular labrum (Kim and Azuma 1995) provides further evidence that intraarticular free nerve endings (type IVa) may help to prevent excessive joint motion causing damage of the acetabular rim and/or adjacent cartilage. According to this hypothesis, FNE that are damaged by traumatic or degenerative lesions of the LCF lose their ability to transmit a mechanical stimulus as an efferent impulse. This absence of the muscular reflex might impair, through loss of fine coordination, the protective function of the joint with ensuing micro- and macrotraumata. In inflammatory or degenerative joint disease, mediators such as bradykinin, histamine, and prostaglandin E2 might activate nociceptive nerve endings (Biedert et al. 1992) responsible for the hip pain reported by these patients (Gray and Villar 1997).

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