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CASE REPORT

# Isolated posterior cruciate ligament aplasia: a case report

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**Abstract**
Congenital absence of the cruciate ligaments is a very rare condition, with most reported cases being related to the anterior cruciate ligament (ACL). Posterior cruciate ligament (PCL) congenital abnormalities have been described as almost always being associated with other anomalies. We present a case of a patient with lateral knee pain and subjective instability sensation after a low-energy trauma secondary to a lateral meniscus tear, with absence of the PCL on MRI examination and thickened meniscofemoral ligaments, with anterior and superior insertion at the femur, in the footprint of PCL. The patient had a menisci repair, without PCL reconstruction. At the 6-month follow-up, the patient did not report any instability symptoms during his daily work and sports activities. There are therapeutic implications in distinguishing a chronic PCL tear from an aplasia, so it is important to detect some signs in imaging that may guide to differentiate them, as there could be differences in the course and thickness of meniscofemoral ligaments, besides the absence of the entire PCL. To our knowledge, this is the first case of PCL agenesis with arthroscopic correlation.

**Keywords** Posterior cruciate ligament · Agenesis · Knee stability · Embryology

## Introduction

Congenital absence of the cruciate ligaments is a very rare condition, usually associated with other malformations, with a prevalence of 0.017 per 1000 live births [1–3]. Most cases are related to ACL agenesis [4–7]. PCL congenital abnormalities have been described as almost always being associated with other anomalies of the lower limb, such as ACL agenesis, meniscus alterations, and patellar dysplasia [6, 8]. We report a case of isolated congenital absence of the PCL, not associated with other abnormalities of the knee.

## Case report

A 53-year-old Caucasian male presented with a 3-month history of low-energy knee torsion (he stumbled when walking), following lateral knee pain and subjective instability sensations. No previous injuries or knee surgeries were reported.

During clinical examination, the lateral meniscal signs were noted as well as considerable posterior instability (positive posterior drawer sign 3+). There was no joint effusion. Clinical examination of the left knee did not reveal any abnormality.

On plain radiographs in a standing position, no bone alterations or axis deviation were found. On MRI of the right knee, the PCL was not detected. Also, thicker meniscofemoral ligaments (Wrisberg and Humphrey) were observed, with an anterior femoral insertion, near the footprint of the PCL (Fig. 1). A complex lateral menisci tear with compromise of body and anterior horn was detected (Fig. 2). A normal image of the intercondylar tibial eminence and articular cartilage, the medial menisci, collateral ligaments, and the ACL was noted.

The left asymptomatic knee was also examined with MRI, with no abnormalities found at this side, but it was useful to compare it with the right knee findings (Fig. 3).

The patient underwent arthroscopic knee menisci surgery. The orthopedic surgeon looked carefully at the tibial and femoral footprints of PCL, without signs of inveterate lesion, such as fragmented ligaments, bands with abnormal orientation or insertional surface irregularity (Fig. 4a). Thicker meniscofemoral ligaments were confirmed, with

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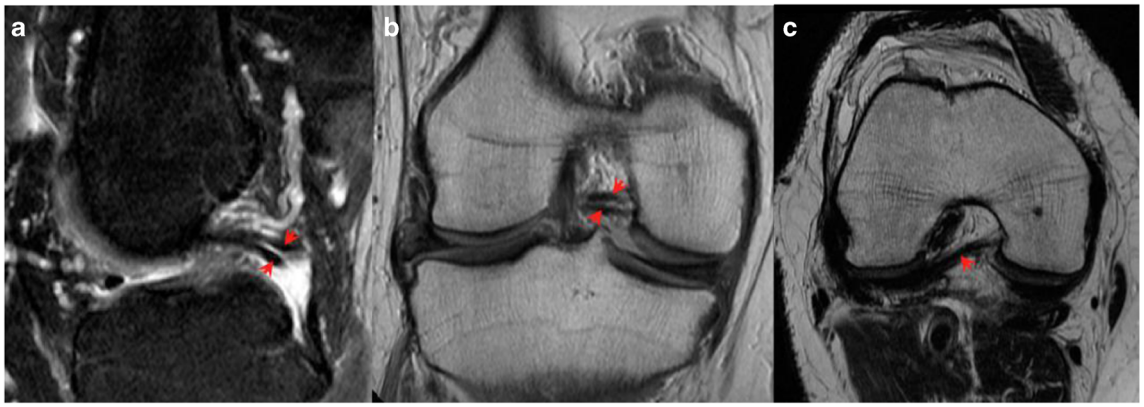


**Fig. 1** **a** Sagittal DP FS; **b** coronal DP; **c** axial oblique T2 WI. Thicker meniscofemoral ligaments (Humphrey and Wrisberg, *red arrows*) with absence of PCL, with meniscofemoral ligaments proximal insertion near normal PCL footprint

an anterior and superior insertion in the femur, at the footprint of PCL (Fig. 4b). No PCL reconstruction was performed.

The patient started physiotherapy after surgery, with almost immediate recovery of subjective knee stability. At the 6-month follow-up, the patient did not report any instability symptoms during his daily work and sports activities (recreational soccer 1 time/week).

## Discussion

Isolated absence of PCL is a very rare congenital abnormality. To our knowledge, only two cases have been reported in the literature [9, 10], none of them with surgical confirmation. Theodorou et al. [9] reported PCL aplasia in a 31-year-old woman with no history of trauma or instability, with a positive posterior drawer clinical sign. Da Gama Malchér [10] reported another case of isolated PCL aplasia in a 28-year-old male with anterior knee pain without trauma, with important posterior instability. Both cases described absence of PCL on MR images, with no images of inveterate lesion of the PCL. Neither of them underwent surgical confirmation.

An important feature of our case is the presence of thickened meniscofemoral ligaments, with an anterior insertion in the femoral condyle, near the PCL footprint. This fact, combined with the absence of signs of inveterate PCL lesion at arthroscopy, makes it probably a congenital defect. The previous reported cases of isolated PCL agenesis do not describe any abnormality about meniscofemoral ligaments. The only reference to meniscofemoral ligaments and PCL anomalies previously reported is from Benassi et al. [11], who described a hypertrophied meniscofemoral ligament of Humphrey in a patient with congenital absence of ACL and PCL, associated with type 1 A fibular hemimelia. To our knowledge, there are no reports about any relationship between the embryologic development of the cruciate and the meniscofemoral ligaments.

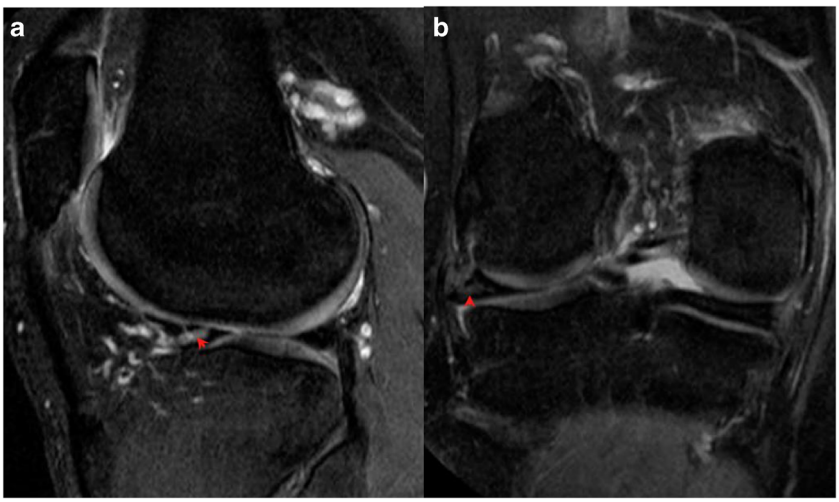


**Fig. 2** **a** Sagittal DP FS; **b** coronal DP FS. Complex lateral menisci tear (*red arrows*)

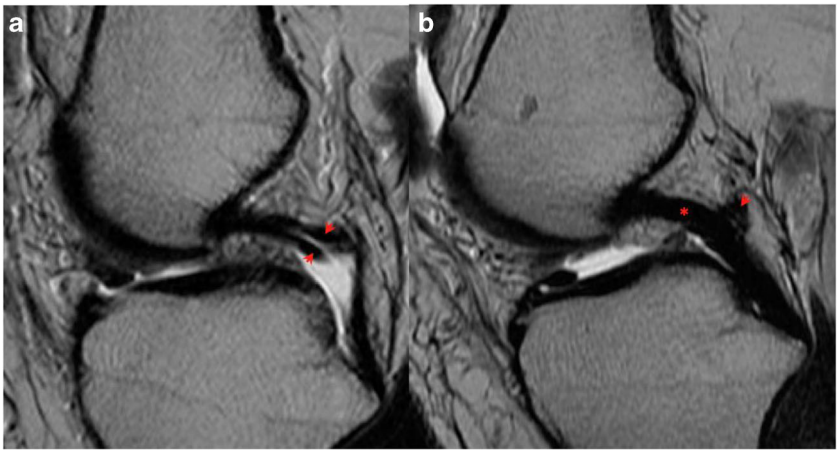


**Fig. 3** **a** Sagittal T2 WI of right knee; **b** sagittal T2 WI of left knee. **a** Absence of PCL with thicker meniscofemoral ligaments (*red arrows*) and anterior proximal insertion in the right knee, versus **b** normal PCL (*asterisk*) and Wrisberg meniscofemoral ligament (*red arrow*) in the left knee

The thickened meniscofemoral ligaments in our case may explain the absence of subjective clinical instability before the meniscal tear in our patient, as they have been described to play a role as a secondary posterior knee stabilizer [12]. The lateral meniscal tear could have produced secondary knee instability, breaking the compensatory status in a knee with congenital absence of PCL, considering the secondary stabilizer role of the lateral meniscus [13], which probably was bigger in the absence of the PCL.

The embryological development of cruciate ligaments seems to be sequential, and it occurs simultaneously with the tibial and femoral condyles, which explains tibial and femoral abnormalities described in ACL agenesis [3]. During the 7th to the 10th week of intra-uterine life, the intra-articular structures of the knee begin to form by direct condensation and differentiation of the blastemal tissue of the interchondral disc [14]. The organization of the cruciate ligaments begins in O'Rahilly stage 21, and the posterior ligaments are the first to become distinguishable [15].

In isolated ACL agenesis or PCL associated with ACL agenesis, some associated bone anomalies are described, such as hypoplastic intercondylar tibial spines [11]. In our case of isolated PCL congenital absence, there are no bone abnormalities, neither in this case nor in the previously reported cases.

In ACL and PCL agenesis, it seems that the anomaly that causes the anatomical defect expresses around the 7th–8th week. Three patterns of ACL/PCL agenesis have been described [3]: Type I is defined by hypoplasia or aplasia of the ACL with a normal PCL; type II is defined by aplasia of the ACL in combination with hypoplasia of the PCL; and type III, by aplasia of both cruciate ligaments. This classification does not mention the isolated agenesis of PCL, so we propose the addition of a type IV.

The therapeutic approach in the case of cruciate ligament agenesis is controversial, and some authors think that there is no surgical indication for several reasons: congenital absence of the cruciate ligaments is usually well tolerated, and reconstructing them can involve difficult technical problems, with a high percentage of failure or poor results. In addition, the articular surface is usually abnormal and it does not seem reasonable to break the compensation that has existed in a congenitally pathologic knee [14, 16]. Because there are therapeutic implications in distinguishing a chronic PCL tear from an aplasia, it is important to detect some signs in imaging that could guide to differentiate them, as there could be differences in the course and thickness of meniscofemoral ligaments, besides the absence of the entire PCL.

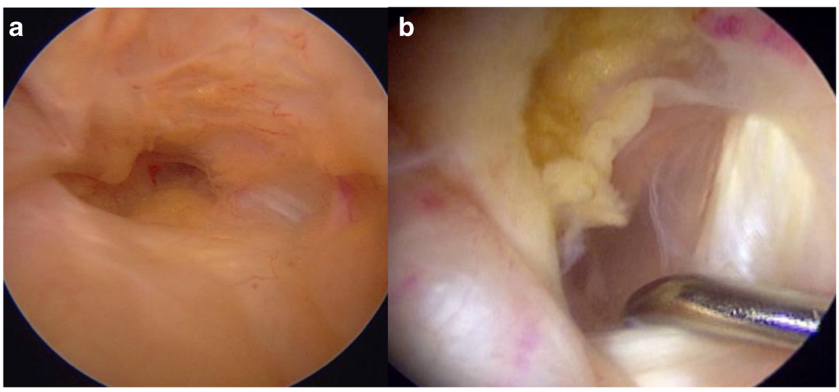


**Fig. 4** **a** Arthroscopic view of posterior tibial plateau, at the distal footprint of PCL, without signs of inveterate PCL lesion. **b** Thicker meniscofemoral ligaments tractioned with an arthroscopic hook

## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflicts of interest.

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