

1   **Combined ACL Reconstruction and Segond Fracture Fixation Fails to**  
2   **Abolish Anterolateral Rotatory Instability – A Case Report**

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8

9   **Abstract**

10   The Segond Fracture (SF) is considered pathognomonic of an anterior cruciate ligament  
11   (ACL) tear. However, the precise anatomy of the soft tissue attachments responsible for  
12   avulsion of SF's have been a cause of controversy in the literature with some authors  
13   suggesting that they occur due to avulsion of the iliotibial band (ITB) and others reporting that  
14   it is the anterolateral ligament (ALL).

15  
16   A thirty-one-year-old male patient presented with a work-related injury to his right knee that  
17   resulted in ACL tear and a SF. Open SF fixation and arthroscopic ACL reconstruction were  
18   performed. The anatomical dissection performed in order to fix the SF demonstrated that the  
19   avulsion had occurred as a result of the tibial attachment of the ALL with a completely intact  
20   ITB.

21  
22   At one-year postoperative follow-up, the ACL graft had restored anterior tibial translation to  
23   within normal limits. However, residual rotational knee laxity was observed in the absence of  
24   any other secondary restraint lesions. This is an important finding because it highlights that  
25   patients with SF may be at increased risk of persistent instability after ACL reconstruction  
26   even in the presence of an anatomically correctly positioned and well-functioning ACL graft.  
27   It also demonstrates that anatomical reduction and fixation of SF at the time of ACLR does  
28   not necessarily restore normal knee kinematics and consideration should be given to recession  
29   of the fixation or augmentation of the ALL when dealing with this injury pattern.

30  
31   A thirty-one-year-old male patient presented with a work-related injury to his right knee that  
32   occurred when he was struck by a truck at low speed. The mechanism of injury involved

33 anterior tibial translation, varus stress and internal rotation. Physical examination revealed the  
34 following findings: large joint effusion, range of motion 0-100°, no neurological or vascular  
35 deficit, positive Lachman's test with a soft end-point, a side-to-side anteroposterior laxity  
36 difference of 7mm measured by the Rolimeter device (Aircast, Europe), and a grade II pivot-  
37 shift (clunk).

38 Plain radiographs demonstrated a fracture of the anterolateral border of the tibial plateau  
39 (figure 1A) and MRI showed a complete anterior cruciate ligament (ACL) rupture with a  
40 concomitant 3x16x18mm fracture of the anterolateral tibial border (figure 1B). MRI did not  
41 demonstrate any other intra- or extra-articular injuries. Specifically, there was no evidence of  
42 injury to any other ligamentous structure, chondral injury, lateral condyle notch sign, or any  
43 type of meniscal tear.

44

#### 45 *Examination Under Anesthesia*

46 The patient underwent an ACL reconstruction (ACLR) and open reduction and internal  
47 fixation of the Segond fracture five days following the injury. Examination under general  
48 anesthesia, prior to ACLR, confirmed the previous examination findings of a positive  
49 Lachman's test and a grade II pivot shift. Examination of other knee ligaments revealed no  
50 abnormality.

51

#### 52 *Lateral Surgical Exploration and Fixation of Segond Fracture*

53 The patient was positioned supine on the operating table in the standard arthroscopy position.  
54 First, the anterolateral compartment was approached, as described by Daggett et al.[1] A  
55 curvilinear incision starting proximal to the lateral epicondyle and extending distally between  
56 the fibular head and Gerdy's tubercle was made. The iliotibial band (ITB) was identified, and  
57 found to be completely normal, with no visible tear, bruise or hematoma. A longitudinal  
58 incision was made along the posterior aspect of the ITB. The biceps femoris bursa was  
59 opened and the tendon was found at its fibular insertion. The lateral collateral ligament (LCL)  
60 insertion was then identified. An anterolateral tibial bony avulsion was observed. Attached to  
61 the bony tibial avulsion, a strong ligamentous structure overlapping the LCL was dissected.  
62 This ligament had a femoral attachment proximal and posterior to the lateral epicondyle, and a  
63 broad tibial insertion to the bony avulsion, consistent with the known anatomy of the  
64 anterolateral ligament (ALL) (Figure 2).[1] Despite the large bony avulsion, the joint capsule  
65 was intact, as evidenced by the absence of any leakage of the hemarthrosis from the joint. The

66 fracture was anatomically reduced and fixed with a 3.5mm cancellous screw and a washer  
67 with the knee in full extension and neutral rotation.

68

69 *Arthroscopic Evaluation and ACLR*

70 A thorough arthroscopic evaluation of the knee was performed. This confirmed the absence of  
71 medial and lateral meniscal injury (including the absence of ramp lesion or meniscal root  
72 avulsion), abnormal medial or lateral compartment opening, or chondral injury. The only  
73 abnormality present was an isolated complete rupture of the ACL at its femoral insertion. The  
74 ACL was reconstructed using the single anteromedial bundle biological augmentation  
75 (SAMBBA) technique with a tripled semitendinosus 9mm graft.[2] A 9mm tibial tunnel was  
76 drilled at the center of the native footprint with a guide set at 60°. A 9x25mm femoral socket  
77 was drilled with an outside-in technique (flip cutter, Arthrex, Naples, USA). The center was  
78 located at the anatomic insertion of the ACL, midway between “resident’s ridge” and the  
79 posterior wall of the femoral condyle. The graft was passed through the joint via a suture loop  
80 retrieved through the tibial tunnel. Fixation was achieved using an adjustable loop cortical  
81 button (TightRope RT, Arthrex, Naples, USA) on the femoral side, and a 9x23mm absorbable  
82 biocomposite interference screw (Arthrex, Naples, USA) on the tibial side, fixed in 30  
83 degrees of flexion, with a posterior drawer applied. The iliotibial band and skin were sutured,  
84 and no drains were used.

85

86 The rehabilitation program used was the same as the standard protocol used for ACLR at our  
87 institution. The patient was discharged on the day of surgery and immediate, brace free, full  
88 weight bearing with crutches was allowed. Initial emphasis was placed on quadriceps  
89 activation with voluntary muscle contraction, and on achieving immediate full extension. Full  
90 range of motion through passive flexion and patellar mobilization were also allowed.

91 At one-year postoperative follow-up, the patient had a full, pain-free range of motion. The  
92 subjective IKDC score was 68.97. The Lysholm score was 79. Lachman’s test showed a  
93 restored normal laxity with a firm end-point. The side-to-side difference was +2 mm, as  
94 measured by the Rolimeter (Aircast, Europe). These results confirm that the ACL graft had  
95 restored anterior tibial translation to within normal limits. However, residual rotational laxity  
96 was observed. The pivot shift test was positive, Grade II (Clunk). The KiRA test (Orthokey,  
97 Carrara, Italy) showed a differential range of 3.0. Plain radiographs showed union of the  
98 Segond fracture in a perfectly anatomic position and no ACL tunnel malposition.

99

100 **Discussion**

101 The lateral capsular sign was first described in 1879 by Dr. Paul Ferdinand Segond and is  
102 frequently referred to as the Segond fracture.[3] The SF is considered pathognomonic of an  
103 ACL tear. In patients with the radiographic diagnosis of SF, up to 95% are reported to have an  
104 ACL rupture.[4] However, in acute injuries of the ACL, SF is inconstant, ranging in incidence  
105 from 1.1% to 30%. [4–9] SF most commonly results from an internal rotation and varus stress  
106 on a flexed knee. These forces tension the anterolateral structures resulting in a bony avulsion  
107 of the anterolateral tibial plateau.[1,10] However, the precise anatomy of the soft tissue  
108 attachment responsible for the avulsed SF have been a cause of controversy in the literature  
109 with some authors suggesting that it is due to avulsion of either the ITB or anterolateral  
110 ligament (ALL).[4,11–13]

111

112 The anatomical dissection of the anterolateral structures in this clinical case help to delineate  
113 the pathoanatomy of SF. Previous reports have been a cause of confusion but in this case it  
114 was clearly demonstrated that the Segond fracture is the result of an avulsion of the tibial  
115 attachment of the ALL. The dissection revealed that the ITB was entirely normal and that the  
116 structure responsible for avulsing the SF passed superficial to the LCL to attach proximal and  
117 posterior to the lateral epicondyle. Furthermore, this structure was independent of both the  
118 ITB and LCL and had a broad attachment to the proximal tibia, posterior to Gerdy's tubercle.

119

120 This demonstration that the SF is due to an avulsion of the tibial attachment of the ALL is  
121 supported by the findings of several previous cadaveric studies, including biomechanical  
122 analyses, that have proposed that the ALL tibial attachment is linked to the SF fracture.[4,14–  
123 16] Claes et al. described that “anatomic data on the tibial ALL insertion site would match the  
124 constant anatomic location on the proximal tibia from where Segond fractures do avulse”. [4]  
125 Dodds et al. stated that the ALL is the only structure inserting in the location where SFs  
126 occur.[15] Kennedy et al. showed that the location and strength of the tibial ALL attachment  
127 is sufficient to produce a SF, and in addition, several authors have reported iatrogenic SFs  
128 occurring during biomechanical tests aimed at evaluating the strength and stiffness of the  
129 ALL.[7,16]

130

131 Part of the reason for the previous controversy regarding which structure attaches to SF's is  
132 likely due to the fact that dissection of the anterolateral aspect of the knee can be difficult and  
133 this has led to conflicting evidence in the literature with regards to the anatomy, function and

134 even the existence of the ALL.[17] Based on many hours of cadaveric dissection, Daggett et  
135 al. provided a simple and reproducible dissection protocol by which the ALL can be easily  
136 found in all knees.[1] An important pitfall to avoid during ALL dissection is anterior to  
137 posterior ITB reflection, as this can make the ALL difficult to identify. ITB reflection must be  
138 performed from proximal to distal, until its insertion to Gerdy's tubercle. Surgeons have also  
139 tried to find the ALL at its femoral origin during many dissection studies, which can be  
140 considered almost impossible to do without damaging some of its fibers because of its  
141 surrounding tissue and fine insertion. The ALL must be first identified at its larger tibial  
142 insertion, between Gerdy's tubercle and the fibula head, and this is aided by posterior  
143 reflection of the biceps femoris.

144 Another important reason for the previous controversy is the over-reliance on laboratory  
145 studies and therefore the current report is important in confirming the findings of previous  
146 cadaveric studies in a clinical case. Additional clinical evidence is provided by Ferretti et al.  
147 who performed anterolateral knee exploration in patients undergoing ACLR.[13] In keeping  
148 with the surgical findings of the current case, they found that the ITB was completely normal  
149 in 33% (n=20) of cases but that in the remainder it was either ecchymotic or swollen.  
150 However, the rate of injury to the ALL was considerably higher (90%, n=54) than the rate of  
151 ITB injury. In 6 of the cases, Ferretti et al. reported the presence of SFs and also reported that  
152 this was due to avulsion of the ALL.[13]

153

154 The second major learning point from this case report is that anatomic reduction and fixation  
155 of SFs may not be enough to control anterolateral rotatory instability in the context of an  
156 anatomically correctly positioned and well-functioning ACL graft. A recent large  
157 retrospective cross sectional study concluded that SF is not a risk factor for ACL graft failure  
158 but did not specifically assess the rate of persistent instability.[18] Unfortunately, the study  
159 had numerous limitations. The authors used only plain radiographs and MRI to evaluate for  
160 the presence of SF. However, it is recognized that these imaging modalities are not as  
161 sensitive as ultrasound, which detects SF at a rate of approximately 30% in ACL injured  
162 knees.[8] Gaunder et al. reported that only 5.3% (29/552) of patients had an avulsion of the  
163 anterolateral tibia which suggests that some diagnoses of SF were likely missed.[18] Another  
164 major limitation was that they did not report the rate of recognized risk factors for ACL graft  
165 failure, for example pre-operative side-to side laxity difference, participation in contact sports,  
166 age, lateral femoral condyle notching, meniscal injury or an evaluation of tunnel position all  
167 of which confound the study. Finally, the authors did not have a robust follow up arrangement

168 and assumed that patients had not had a graft failure unless they had presented to their  
169 institution for revision. This raises concerns about the validity of the authors conclusions that  
170 recommended against repair of SF or ALL reconstruction at the time of primary ACLR. In  
171 contrast, evidence from a recent biomechanical cadaveric study has demonstrated that the  
172 mean anterior tibial translation and axial tibial rotation were both significantly higher in knees  
173 with combined ACL rupture and SF when compared to isolated ACL injured knees.[19]  
174 Furthermore, the findings in the current case suggest that SF may be an important reason for  
175 persistent instability.

176

177 Although persistent instability occurs in up to 30% of patients after ACLR, this can typically  
178 be attributed to secondary restraint lesions or technical error such as tunnel malposition.[20–  
179 22] In this case, the tunnels were well positioned, and apart from SF there were no other  
180 concomitant injuries. However, it is recognized that in the presence of injury to the  
181 anterolateral structures of the knee, isolated ACLR fails to restore normal knee kinematics  
182 unless ALL reconstruction or another type of lateral extra-articular tenodesis type procedure  
183 is performed.[23] In this case, it was assumed that reduction and fixation of the SF, in  
184 addition to ACLR, would therefore abolish the abnormal kinematics known to occur as a  
185 result of injury to the anterolateral knee structures. This strategy was supported by a recent  
186 case report which shows that repair of an acute ALL tear can abolish the pivot shift.[19]  
187 However, direct repair of an ALL injury allows restoration of normal ligament tension  
188 whereas fixation of the SF does not address any potential injury to the structure of the ALL  
189 itself. It is therefore postulated that a possible elongation, multi-level injury or partial failure  
190 of the ALL may have occurred and offers an explanation as to why the pivot shift persisted  
191 despite restoration of normal AP stability. These biomechanical concepts have already been  
192 described for another similar scenario: ACL injury in the setting of a tibial spine avulsion.[24]  
193 Interstitial damage of a ligament can occur, and secondary laxity may be present even after  
194 fracture fixation.[25] As a true ligament, the ALL might have similar intrinsic behavior to the  
195 ACL. Some clinical evidence to support this again comes from the surgical exploration study  
196 performed by Ferretti et al. because they found that 58% of patients had multi-level injury to  
197 the ALL in apparently isolated ACL-injured knees.[13] Similarly, other authors also report  
198 the identification of both proximal and distal ALL abnormalities occurring in the same knee,  
199 at the time of ACL rupture.[7]

200

201 These findings suggest that if the bony fragment is large enough to warrant surgical fixation,

202 recession of the fixation or further augmentation of the ALL should be considered to  
203 compensate for possible stretching of the ALL fibers.

204

205 In conclusion, this clinical case report confirms the findings of previous cadaveric and clinical  
206 studies that have suggested that avulsion of the SF is due to the attachment of the ALL to this  
207 region of the proximal tibia. The findings of this case report also suggest that interstitial  
208 injury, possible elongation and multi-level injury may occur to the ALL during SF and  
209 therefore simply fixing the fracture may not be enough to restore normal knee kinematics.

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277 Figure 1. A) Antero-posterior radiograph of the right knee demonstrating avulsed Segond  
278 fracture fragment (white arrow). B). Axial MRI of the same knee showing avulsed Segond  
279 fracture fragment (white arrow).

280 Figure 2. Intraoperative images of the lateral aspect of the right knee. (A) An incision has  
281 been made along the posterior aspect of the intact ITB. The biceps femoris tendon has been  
282 reflected. (B) Reflection of the biceps femoris tendon allows the LCL and the tibial  
283 attachment of the anterolateral ligament (ALL) to be easily identified. (C) The Segond  
284 fracture has clearly been avulsed by the ALL which passes deep to the ITB and is separate  
285 from it. The ALL is located superficial to the LCL and attaches proximal and posterior to the  
286 lateral epicondyle.

287 Figure 3. Postoperative radiographs of the right knee at 1 year follow up demonstrating  
288 healing of the Segond fracture in a perfectly anatomical position and no ACLR tunnel  
289 malposition.

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