Common peroneal nerve palsy complicating knee dislocation and bicruciate ligaments tears

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KEYWORDS
Common peroneal nerve palsy; Traumatic knee dislocation; Bicruciate ligament injuries

Introduction: The occurrence rate of common peroneal nerve (CPN) palsy associated with knee dislocation or bicruciate ligament injury ranges from 10 to 40%. The present study sought first to describe the anatomic lesions encountered and their associated prognoses and second to recommend adequate treatment strategy based on a prospective multicenter observational series of knee ligament trauma cases.

Material and methods: Twelve out of 67 knees treated for dislocation or bicruciate lesion presented associated CPN palsy: two females, 10 males; mean age, 32 years. Four sports injuries, three traffic accidents and five other etiologies led to seven complete dislocations and five bicruciate ruptures. Four cases involved associated popliteal artery laceration ischemia; one of the dislocations was open. Paralysis was total in eight cases and partial in four. There were two complete ruptures, three contusions with CPN in continuity stretch lesions and three macroscopically normal aspects.

Results: At a minimum 1 year’s follow-up, regardless of the initial surgical technique performed, recovery was complete in six cases, partial (in terms of motor function) in one and absent in five. Without specific CPN surgery, spontaneous recovery was partial in one case, complete in
In traumatic dislocation of the knee and bicruciate rupture involving severe initial displacement, the peroneal branch of the sciatic nerve is subjected to very severe traction, being immobilized by its passage around the head of the fibula [1–3]. The incidence of associated common peroneal nerve (CPN) palsy ranges from 10 to 40% (Table 1). Prognosis is especially poor as there is often associated rupture of the popliteal artery axis [4–8]. Few studies have specifically focused on the neurological prognosis, whether spontaneous or secondary to surgery, in case of CPN lesion [9–12].

The present study described the anatomic lesions and prognosis for remission of CPN palsy associated with dislocation of the knee and bicruciate rupture. The secondary objective was to assess the medium-term impact of CPN palsy on the overall functional result and to recommend a specific treatment strategy to be integrated in the management of ligament trauma.

Material and methods

This prospective study recruited 66 patients (67 knees) treated for dislocation of the knee or bicruciate rupture between January 2007 and January 2008 in the 12 member-centers of the 2008 French Society of Orthopedic and Traumatologic Surgery (SOFCOT) symposium. The series comprised 51 knee dislocations and 16 bicruciate ruptures; 12 lesions showed associated CPN involvement. The neurologic lesions concerned two female and 10 male patients, with a mean age of 32 years (range, 21 to 53 years). Trauma velocity was judged low in five cases and high in seven. Trauma circumstances were varied: four sport accidents (two soccer, one rugby, one judo), three road accidents (two two-wheel, one four-wheel) and five other (one fall, two farm accidents, one site accident).

Seven of the 12 cases of CPN palsy were associated with complete knee dislocation (one anterior, one anteromedial, one medial, two posteromedial) and five to bicruciate rupture, all associated with on-the-spot reduction of dislocation by the emergency team. One dislocation was open; four CPN palsies were associated with ischemia secondary to popliteal axis rupture. Five patients presented with associated contralateral (two coxofemoral lesions, one bicruciate rupture, one diaphyseal fracture) or ipsilateral peripheral trauma (one bi-malleolar fracture and one lateral femoral condyle fracture).

CPN palsy was always isolated, with no posterior sciatic involvement. There was total (motor plus sensory) paralysis in eight cases and partial (motor only, with conserved sensitivity) in four. In eight cases, immediate vascular repair or an early secondary ligament approach disclosed the anatomic status of the nerve, with two complete ruptures, three confusions with elongation and three macroscopically normal aspects. No emergency neurologic repair was performed.

The multicenter design did not include any prospective neurologic treatment strategy. Revision criteria were based on the usual clinical and X-ray semiology for knee ligament trauma. Neurologic remission was assessed in terms of the four muscle groups innervated by the CPN: tibialis anterior, extensor digitorum longus, extensor hallucis longus and peroneus longus and brevis. Sensory recovery was assessed at the first dorsal commissure. Patients were also interviewed on general motor function, with mandatory reference to neurologic status (walking and stair climbing, possibility of intense physical activity).

Results

All 12 CPN palsy patients were followed up for at least 1 year, with examination, except for one patient who had moved out of the area and was contacted by telephone (Table 2). Overall, and independently of surgical attitude, six patients showed total remission, with force equal to the contralateral side and normal sensitivity at the first dorsal commissure. Five patients failed to recover, with total sensorimotor impairment in four cases and partial impairment in one; the remaining patient showed partial remission, with persisting 3/5 paralysis of the anterolateral compartment muscles. Two patient groups could be distinguished in terms of CPN surgery. Four patients underwent no neurological surgery; spontaneous evolution gave partial remission in one case, total remission in two and no remission in one. Five of the other eight patients underwent simple emergency or secondary exploration, according to the spontaneous evolution: four showed total remission and one no remission. The four cases of total remission (from two total and two partial palsies) comprised three normal aspects and one CPN contusion. The failure of recovery...
Table 1  Frequency of common peroneal nerve (CPN) palsy following dislocation of the knee or bicruciate lesion and relation to popliteal artery rupture and joint opening, in recent reports.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Number of dislocations</th>
<th>CPN palsy (%)</th>
<th>Ischemia</th>
<th>Opening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Honton et al. [18]</td>
<td>11</td>
<td>1 (9)</td>
<td>5</td>
<td>?</td>
</tr>
<tr>
<td>Wright et al. [7]</td>
<td>19</td>
<td>9 (47)</td>
<td>5</td>
<td>19</td>
</tr>
<tr>
<td>Wascher et al. [19]</td>
<td>47</td>
<td>16 (22)</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>Yeh et al. [8]</td>
<td>23</td>
<td>2 (8)</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Richter [21]</td>
<td>89</td>
<td>15 (17)</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>Twaddle et al. [20]</td>
<td>63</td>
<td>9 (14)</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Rios et al. [34]</td>
<td>28</td>
<td>6 (23)</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Liow et al. [30]</td>
<td>22</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Dubrana [10]</td>
<td>91</td>
<td>36 (39)</td>
<td>25</td>
<td>13</td>
</tr>
<tr>
<td>Harner et al. [29]</td>
<td>33</td>
<td>4 (12)</td>
<td>Excluded</td>
<td></td>
</tr>
<tr>
<td>Wong et al. [31]</td>
<td>29</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Niall et al. [12]</td>
<td>55</td>
<td>14 (25)</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Bonnevialle et al. [4]</td>
<td>37</td>
<td>23 (62)</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td>Plancher [33]</td>
<td>50</td>
<td>18 (36)</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>SOFCOT symposium</td>
<td>67</td>
<td>12 (18)</td>
<td>8</td>
<td>6</td>
</tr>
</tbody>
</table>

Table 2  Characteristics of the 12 total (T) or partial (P) CPN palsies following dislocation of the knee (Dis) or bicruciate rupture (Bi).

<table>
<thead>
<tr>
<th>Patient</th>
<th>Knee lesion</th>
<th>Ischemia</th>
<th>Palsy</th>
<th>Lesion aspect</th>
<th>Secondary surgery</th>
<th>Spontaneous remission</th>
<th>Post-op remission</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>F/37</td>
<td>M/53</td>
<td>F/29</td>
<td>M/41</td>
<td>M/42</td>
<td>M/22</td>
<td>M/21</td>
</tr>
<tr>
<td></td>
<td>M/29</td>
<td>M/22</td>
<td>M/21</td>
<td>M/21</td>
<td>M/36</td>
<td>Total</td>
<td>Partial</td>
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</tbody>
</table>

Discussion

The multicenter series comprised 12 patients presenting with CPN palsy associated with bicruciate lesion or dislocation of the knee sustained during a 1-year period; the numbers are small compared to studies over a longer term and/or focused on this specific post-traumatic neurologic lesion, which include dozens of cases [13–17]. The present study incurs several limitations: the macroscopic aspect of the paralyzed nerve was known in only eight cases, precluding any prognostic analysis of spontaneous remission according to the CPN trauma found peroperatively. The prospective data for the associated lesion were observational, with no recommendations as to treatment. And in assessing the overall clinical result for the knee ligament trauma, the CPN lesion was analyzed separately.

CPN palsy depends on the direction and degree of the initial displacement causing the various ligament ruptures, which in turn depends on trauma velocity: the neurologic lesion, like the popliteal vascular lesion, is part of the regional trauma sustained by the knee. The frequency of CPN palsy varies in the literature according to trauma circumstances and to the surgery team’s recruitment (Table 1). There is consensus as to the parallel nature of the neurologic and ligamentary lesions: the more extensive and severe the latter, the more frequent and severe the CPN palsy; the same correlation obtains between cutaneous wound, vascular rupture and palsy [7,8,18–20]. Trauma sustained in an agricultural context or on a two-wheel vehicle is most frequently associated with palsy in case of multiple trauma or ipsi- or contralateral limb lesion. The present 18% incidence of 12 CPN palsies out of 67 dislocations or bicruciate ruptures matches the epidemiological data and corresponds to the varied recruitment of regional hospitals. In the series as a whole, four of the nine popliteal artery axis ruptures were associated with CPN palsy: i.e., 44%, compared to 14% when the vascular axis was conserved. The trauma context should thus guide clinical exploration for possible peroneal sciatic palsy, which might
go undetected in an unconscious patient or in case of multiple fracture where bone lesions obscure the neurological signs.

Isolated CPN palsy shows a characteristic motor and sensory semiology enabling straightforward diagnosis based on objective pathognomic signs, which the patient, in great pain even if conscious, may not be fully aware of. The only factors liable to mislead diagnosis of the anatomic location of the neurological lesion are femoral diaphysis fracture and/or ipsilateral coxofemoral lesion or pelvic ring rupture. Even in direct sciatic trunk trauma, CPN involvement may appear clinically isolated. Neurologic symptoms in the territory of the tibial nerve suggest an underlying knee lesion. In ischemia due to popliteal artery rupture, after a few hours’ evolution the clinical symptoms include a neurological component leading to total sensorimotor motor and sensory paralysis, precluding detection of any CPN palsy “behind” the vascular symptomatology [4,8,11]. Likewise, compartment syndrome requiring aponeurotomy or muscular sequelae of ischemia also hinder neurologic assessment. Only early clinical examination within the first hours can solve this diagnostic issue. In the present series, only one patient presented with dislocation of the knee associated with a displaced lateral condyle fracture: during open fixation, the CPN was found to be continuous and no anatomic factor underlying the paralysis could be determined. In the four cases of ischemia due to popliteal artery rupture, CPN palsy was isolated, without impact for the posterior sciatic territory, illustrating the independence between the neurologic and vascular lesions.

The anatomic status of the CPN, subjected to violent traction during the dislocation, is the prime prognostic factor for spontaneous neurological remission. This is not always specified in the literature, often because peroperative CPN exploration and imaging were lacking. Some authors perform systematic early exploration [11,12], others only during lateral ligament repair [21]. The anatomic aspect of the traumatized CPN is generally known when palsy occurs secondary to open dislocation and/or vascular rupture, which require surgical exploration. Nerve rupture is then found in about a quarter of cases: it is never regular but always associated with several centimeters of laceration (Table 3). In complete paralysis, the other anatomic aspect found is contusion and/or elongation of the nerve along several centimeters. With such an aspect of non-ruptured nerve and associated palsy, spontaneous remission is hard to predict. Niall et al. [12] gave a precise macroscopic description of the paralyzed CPN in 14 dislocations of the knee and drew up a prognosis chart in terms of the extent of contusion. Three out of 10 non-ruptured CPNs showed less than 7 cm contusion and seven between 7 and 12 cm. In two cases in the former group, total spontaneous remission was obtained in only 3 months; in two cases in the latter group, the CPN remained paralyzed, two showed partial remission and four recovered virtually normal function in 12–18 months. Thus, the length of the trauma lesion in a non-ruptured CPN was predictive of recovery; and extensive CPN contusion on top of total anatomic rupture was predictive of failure of spontaneous remission, as also highlighted by Piton et al. [16] in a retrospective study of more than 150 CPN lesions: 22 CPN palsies were found in a series of knee dislocations in which the CPN was explored only in case of failure of spontaneous remission, with nine non-ruptured nerves that were simply released and 13 ruptures, managed by graft. Six of the nine neurolyses gave excellent results, although the exact anatomic status was not specified. Lesion length is also a prognostic factor for regrowth following interfascicular graft [13–17]. With only eight anatomic lesions known out of 12 paralyzed nerves, the present series cannot provide quantitative data, but is in line with literature findings. The neurologic lesion is situated at the passage around the fibular head; but the proximity of the bifurcation may also account for the tearing of the terminal branches where they penetrate the muscle [12]. In emergency, the likelihood of irreversible CPN lesion is not clearly known. NMR spectroscopy sheds light on capsule-ligamentary anatomic status, but not on the neurologic lesion [22–24]. Systematic exploration to assess CPN anatomy in case of failure of EMG and/or clinical recovery at 3 months may be rendered superfluous by the simplicity and reliability of sonography, which can easily be repeatedly performed during surveillance and, in expert hands, provides reliable assessment of the neurologic lesion [25,26].

Functional prognosis for the limb following dislocation or bicruciate rupture depends not only on ligament healing but also on neurologic remission when there is total CPN palsy, as seen in a steppage gait and tibio-talar joint instability. Neurologic sequelae inevitably impact overall limb function and rapid walking and running in particular. In recent series of surgery for dislocation of the knee, this functional impact has seldom been highlighted. Certain authors excluded associated CPN palsy from their ligament repair series [27,28]. For others, rapid spontaneous remission eliminated impact on ligament repair results [8,29–32]. Despite considerable rates of rupture and non-remission, several authors did not mention the impact of neurologic sequelae on their final results [7,21,32–34].

There is no consensus attitude towards palsy, which is seldom mentioned in reports on ligament surgery [8,26,28,35,30,31] or on overall management of such trauma cases [6,11,29,36–39]. Rosset et al. [40] distinguish several situations: in case of subtotal palsy, CPN release is recommended if early clinical and EMG recovery fails to progress and/or in all cases of lateral ligament repair. In total initial palsy, exploration and release are systematic in case of lateral ligament surgery; when rupture is discovered, the extremities are to be located to facilitate secondary interfascicular grafting. In knee dislocation with associated CPN palsy, Goitz and Tomaino [41] recommend early surgery, in agreement with Niall et al. [12]; they reported their experience in treating CPN palsy of various origins, recommending convergent attitudes in the light of the analysis of the results reported in the literature [13,14,42,16,17] and recommend early intervention in the absence of signs of clinical and EMG improvement. CPN surgery is to be guided by the macroscopic aspect and should at least comprise release from fibrotic scar tissue. In case of total rupture or a contusion segment with fascicular interruption requiring resection into healthy tissue, experience shows that long grafts fail to achieve recovery: according to Bletton et al. [13], 20 cm is the threshold beyond which functional axon regrowth is no longer obtained; Kim et al. [15] set the threshold at 6 cm and Piton et al. [15] at 15 cm. The poor prognosis for spontaneous remission of extensive anatomic lesions leads certain
authors to recommend associating nerve surgery and tendon transfer in a single operative step [14].

**Conclusion**

CPN palsy is frequent in dislocation of the knee and cruciate lesion, as a result of the trauma and displacement velocity involved. The nerve is ruptured or the lesion is irreversible in more than a third of cases, with no hope of spontaneous remission. The neurologic sequelae seriously impact the overall functional result. As with the vascular lesions, the displacement needs to be rapidly reduced to restore the anatomic trajectory of the CPN. Early surgery is recommended, comprising at least exploration and CPN release along the lateral fibular trajectory. Total rupture and contusion of more than a few centimeters are of poor prognosis for grafting. The patient should be quickly and clearly informed of this and palliative treatment proposed. Any neurologic involvement would be usefully included in assessing the overall functional results of treatment for severe rupture and dislocation of the knee.

**Conflict of interest**

None.

**References**


**Table 3** Review of the main literature series describing the macroscopic aspect of paralyzed common peroneal nerve in dislocation of the knee.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Palsies/dislocations</th>
<th>CPN explored</th>
<th>Total rupture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wright et al. [7]</td>
<td>9/19</td>
<td>7</td>
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<tr>
<td>Wascher et al. [19]</td>
<td>16/47</td>
<td>8</td>
<td>3</td>
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<tr>
<td>Richter et al. [21]</td>
<td>15/89</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Rosset et al. [40]</td>
<td>36/91</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Harner et al. [29]</td>
<td>4/33</td>
<td>11</td>
<td>1</td>
</tr>
<tr>
<td>Niall et al. [12]</td>
<td>14/55</td>
<td>23/37</td>
<td>57</td>
</tr>
<tr>
<td>Bonnevialle et al. [4]</td>
<td>23/37</td>
<td>57</td>
<td>23 = 40%</td>
</tr>
</tbody>
</table>


