Compression syndromes of the popliteal neurovascular bundle due to Baker cyst

Jorge E. Sanchez, BS, Nicole Conkling, BA, BS, and Nicos Labropoulos, PhD, DIC, RVT, Stony Brook, NY

Background: The purpose of this study was to perform a comprehensive search of the literature for all studies, case reports, and series describing Baker cyst compression of the neurovascular bundle in the popliteal fossa and index their findings according to the structures compressed.

Method: Case reports and series obtained after a thorough MEDLINE search were indexed according to compressed structures. Patient demographics, main findings, method of diagnosis, cyst size, outcomes, and follow-up were recorded for each publication.

Results: Signs and symptoms related to popliteal vein and tibial nerve compression were the most frequent presentation of symptomatic Baker cysts, due to the anatomic vulnerability of these structures within the popliteal fossa and their relative sensitivity to compression. Patients with tibial nerve entrapment demonstrated gastrocnemius muscle atrophy, paresthesias, and pain. Those with popliteal vein compression experienced swelling, pain, and rarely, venous thromboembolism. Isolated arterial compression, presenting with intermittent claudication, is a rare occurrence because it is a relatively stiff-walled vessel, has a higher pressure, and is located deep in the popliteal fossa. Combinations of these compressive syndromes are most frequently encountered in the context of cyst rupture and resulting compartment syndrome.

Conclusions: Baker cyst is an important pathology for the differential diagnosis of popliteal neurovascular compression phenomena. It has a wide spectrum of presentation, therefore requiring accurate diagnosis for proper patient management. Because Baker cyst is by definition a chronic disorder, long-term follow-up is necessary to monitor patient recovery and prevent recurrence. (J Vasc Surg 2011;54:1821-9.)

Popliteal cysts (Baker cysts) are a relatively common finding in patients aged >50 years, presenting with knee pain or knee pathologies that disturb the synovial fluid dynamics, including arthritis, most commonly, meniscal tears, or rarely, gout. The presence of a popliteal cyst varies by the population studied and on the imaging technique used for diagnosis. A magnetic resonance imaging (MRI) study of 400 consecutive patients referred with knee problems revealed a popliteal cyst in 77 patients (19%). In a patient population with suspected deep vein thrombosis (DVT), duplex ultrasound studies showed a lower presence of popliteal cyst. Voltes et al demonstrated a 4.1% prevalence of underlying popliteal cyst in 1000 patients undergoing venous duplex scanning, whereas Langsfeld et al established a 3.1% prevalence (95 patients) in a similar group of 3072 patients. Another retrospective study determined the incidence of popliteal cysts was 4.7% among patients aged 6 to 89 years old who were referred for symptoms consistent with knee pathology. Knee pathologies can predispose synovial fluid to accumulate within the tendon bursas of the knee, most often causing ballooning of the bursas (usually the semimembranosus tendon bursa) within the popliteal fossa. Previous investigators have studied the usual predisposition of the cyst to migrate in a medial fashion. A 2004 prospective study designed to investigate the etiology of this cyst concluded that a popliteal cyst usually develops in the postero-medial popliteal fossa due to lack of anatomic support in this area of the synovial capsule. The accumulation of fluid within the bursa in this medial anatomic location within the knee typically spares the posterolaterally located neurovascular bundle.

The developing cyst may occasionally migrate posterolaterally and cause compression of one or more components of the popliteal neurovascular bundle. Symptoms of nerve entrapment manifest as tibial or sciatic neuropathy, resulting in gastrocnemius muscle atrophy, or rarely, common peroneal neuropathy. Popliteal vessel compression can also cause a true thrombophlebitic or pseudothrombophlebitic syndrome, and in rare cases of arterial compression, claudication of the lower extremity can occur due to intermittent ischemia. The differential diagnosis for syndromes associated with compression of the neurovascular bundle include intraneural ganglion cyst of distal divisions of the sciatic nerve, adventitial cyst of the popliteal artery, thrombophlebitic syndrome caused by DVT of the lower extremity, synovial sarcoma of the knee joint, popliteal entrapment syndrome, posterior compartment syndrome of the lower leg, popliteal artery aneurysm, and Baker cyst. A popliteal cyst may be comorbid with a number of these conditions, including communication of the cyst with an adventitial cyst of the popliteal artery, development of...
a synovial sarcoma within the cyst, and also rupture of the cyst, resulting in the acute development of a compartment syndrome. It is crucial that the true cause of compression be identified because it directly affects treatment options.

Prior experience documented in the literature is limited to case reports and brief case series on this rare presentation of Baker cyst, and the goal of our study was to perform a systematic literature review that would index the cumulative experience with compression phenomena of the popliteal fossa. No current comprehensive reviews of the literature have been published on this subject.

METHODS

The MEDLINE database was used to conduct a complete literature review to identify existing publications of case reports or series of atypical presentations of popliteal cysts involving compression of the neurovascular bundle. Our search used the following terms: Baker’s cyst/popliteal cysts, tibial nerve compression, posterior compartment syndrome, pseudothrombophlebitis, intermittent claudication, popliteal fossa, and calf atrophy. Synovial sarcoma, adventitial cysts, and knee effusion were used as exclusionary search terms to prevent overlap with other isolated conditions responsible for popliteal neurovascular compression. In an extensive manual search, all relevant references yielded by the initial MEDLINE search were reviewed. A publication date filter was not used, and articles that appeared as early as 1964 through to the present-day were included. We limited our search to reports published in the English language.

As publications were screened, they were indexed into five categories by the syndrome manifestation of the compression: arterial compression, venous compression, nerve compression, compression causing posterior compartment syndrome, and intermittent claudication. This systematic approach allowed for a comprehensive review of the literature on the topic.
syndrome, and other unusual conditions incongruous with
the first four. Our review highlighted the major findings
and yielded a wide differential diagnosis for compression of
the neurovascular bundle in the popliteal fossa.

Because the condition in which the popliteal cyst dis-
sects laterally toward the major nerves and vessels of the
popliteal fossa is exceedingly rare in clinical experience and
in the literature, most existing publications represent only
case reports and short case series and have limited follow-
up. We present two real-patient clinical vignettes to further
illustrate the complications that arise with popliteal neuro-
vascular bundle compression.

**CASE REPORTS**

**Patient 1.** A 52-year-old man presented to the emergency
department with right calf swelling and pain in the posteromedial
popliteal fossa. Ultrasound examination demonstrated a 5 × 8-cm
cyst, and at the time, no DVT was diagnosed. The difference in
calf circumference between the affected and normal extremity
was 3 cm. The patient’s medical history was significant for a
medial meniscus injury of the affected leg that was managed
conservatively.

After initial cyst presentation in the emergency department,
the patient was sent home with a prescription for nonsteroidal
anti-inflammatory drugs and bed rest. He returned to the hospital
2 days later because of increased swelling and pain. The difference
in calf circumference between the affected and normal leg had
increased to 6 cm, swelling extending down the leg, and ecchymo-
sis of the medial malleolus was evident.

Ultrasound imaging revealed a large ruptured Baker cyst con-
taining fluid and hematoma dissecting between the medial head of
the gastrocnemius muscle and the subcutaneous tissue, as well as
between the gastrocnemius and soleus muscles, which were signif-
cantly compressed (Fig 1). Concurrently, a thrombus was identi-
fied in the popliteal vein, which was partially compressed at the
level of the cyst. He soon developed shortness of breath, and an
emergency computed tomography angiography of the lungs re-
vealed bilateral pulmonary emboli, but anticoagulation was con-
traindicated due to the expanding hematoma in the calf.

A measurement of compartment pressures was sufficient for
diagnosis of compartment syndrome. The patient underwent
placement of an inferior vena cava filter at the infrarenal level
through the right common femoral vein, and fasciotomy and
evacuation of the hematoma were performed, after which antico-
agulant therapy was commenced. An ultrasound scan before dis-
charge indicated decompression of the popliteal vein and a re-
duced, residual thrombus. No further complications developed,
and the patient was discharged 8 days after the initial admission.

Table I. Cases of entrapment neuropathy due to Baker cyst

<table>
<thead>
<tr>
<th>First author (year)</th>
<th>Study type</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ji6 (2007)</td>
<td>Case report</td>
<td>Patient presented with calf atrophy and foot drop. Cyst communicated with the articular joint and was located between medial head of gastrocnemius muscle and semimembranosus tendon.</td>
</tr>
<tr>
<td>Robertson14 (2003)</td>
<td>Case report</td>
<td>Large popliteal cyst dissection and rupture proximally into posterior thigh. Sciatic nerve was compressed.</td>
</tr>
<tr>
<td>Daneyemeez34 (1998)</td>
<td>Case report</td>
<td>Traumatic neuroma was suspected, but a firmly wrapped Baker cyst was histologically diagnosed after resection.</td>
</tr>
<tr>
<td>Dash10 (1998)</td>
<td>Case report</td>
<td>Patient developed pain and swelling in knee joint and calf; aspiration of synovial fluid was cloudy.</td>
</tr>
<tr>
<td>Sun17 (1995)</td>
<td>Case report</td>
<td>Synovial cyst leading to tibial mononeuropathy with focal conduction block between popliteal fossa and ankle.</td>
</tr>
<tr>
<td>DiRisio et al34 (1994)</td>
<td>Case report</td>
<td>Tibial nerve branch compression resulting in leg pain and atrophy of gastrocnemius. Medial gastrocnemius denervation. Cyst was directly anterior to medial head of gastrocnemius and posteromedial to joint capsule. Report suggests that the nerve can compress a branch of the tibial nerve when patient assumes certain positions. At the time of surgery, the cyst was not compressing the nerve.</td>
</tr>
<tr>
<td>Kashani9 (1985)</td>
<td>Case report</td>
<td>Degenerative joint disease complicated by Baker cyst and thrombophlebitis; tibial entrapment neuropathy presented 5 weeks later, with decreased sensation and dysesthesia.</td>
</tr>
<tr>
<td>Zygmunt16 (1982)</td>
<td>Case reports (n = 2)</td>
<td>Popliteal cyst presenting in patients with polyarthritids; one patient had tibial nerve entrapment and the other had common peroneal nerve entrapment.</td>
</tr>
<tr>
<td>Nakano12 (1978)</td>
<td>Case series (n = 5)</td>
<td>Popliteal cyst presenting with motor weakness and diminished sensation in four patients with rheumatoid arthritis and presented with foot-drop in a patient after trauma to the knee.</td>
</tr>
</tbody>
</table>

*EMG, Electromyography; F, female; M, male; MRI, magnetic resonance imaging; US, ultrasound.*
He was seen at the clinic a week later with no complaints and only minor discomfort from the incisions at the fasciotomy site. Follow-up assessments at 1 month and 6 months (clinically and with ultrasound) revealed mild chronic changes, with venous wall thickening and some remaining thrombus, with mild segmental reflux. At the 1-year clinical follow-up, the patient complained of mild swelling and pain that was worse toward the end of the day when standing for long periods of time.

**Patient 2.** A 63-year-old woman presented at the clinic with pain and swelling on the left lower leg that progressively worsened, as well as burning and tingling sensations in the affected limb. Physical examination was unremarkable, so no action was taken at that time, and rest was advised. Eight months later, the pain significantly increased without notable swelling. The patient was referred by her primary care physician to the hospital for a vascular consult and evaluation and a neurology workup for her pain and paresthesias. Other than her age, she had no major risk factors for DVT.

An ultrasound study performed to rule out chronic venous disease and DVT revealed no evidence of reflux or venous obstruction; however, a large cyst (7.2 × 6.4 cm) was found in her left popliteal fossa, extending posterolaterally near the neurovascular bundle (Fig 2). The cyst had a mixed appearance, with fluid and clotted blood. The patient was referred to an orthopedic surgeon and a neurologist, who determined that she had tibial neuropathy as a result of compression from the cyst over the neurovascular bundle. The cyst was surgically resected, and a week later when the patient recovered from the surgery, only mild symptoms remained, likely due to the long-term nerve compression.

At 3 and 8 months of follow-up, she had very mild symptoms that did not affect her quality of life, and both of her legs were close to symmetric. An ultrasound scan did not demonstrate the presence of a cyst or hematoma in the popliteal fossa, and the neurovascular bundle was free from any compressing phenomena.

**RESULTS**

A total of 30 publications fit the criteria for our search, representing 73 described patients. Because the cumulative experience in the literature is limited, a meta-analysis was not possible. Also, the variability in the reported sizes of the cysts throughout the literature prevented determination of the minimum cyst size required for symptomatic presentation. However, our thorough review provided insight into the relative occurrence of each type of compression phenomenon. Relevant information from each report was extracted and organized by specific compression syndrome.

<table>
<thead>
<tr>
<th>Method of diagnosis</th>
<th>Patient demographics</th>
<th>Size of cyst</th>
<th>Treatment/patient follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI</td>
<td>58 F</td>
<td>Initial cyst: 6- × 4-cm recurrent cyst: 4 × 3 cm</td>
<td>Cyst decompression, followed by recurrence in 6 weeks; post-op resection course uncomplicated</td>
</tr>
<tr>
<td>Doppler US</td>
<td>89 F</td>
<td>Pre-op MRI: 3.5 × 4 × 13 cm</td>
<td>Surgical removal; post-op course uncomplicated</td>
</tr>
<tr>
<td>MRI</td>
<td>70; 50 F; M</td>
<td>Approx 4- × 2-cm cyst after excision</td>
<td>Excision of cyst, full relief of symptoms and recovery in 3 weeks</td>
</tr>
<tr>
<td>EMG diagnosed nerve entrapment, Baker cyst diagnosed on analysis of effused fluid</td>
<td>22 M</td>
<td>...</td>
<td>Cyst was surgically resected; 3-month follow-up showed dramatic improvement in EMG and motor and sensory function</td>
</tr>
<tr>
<td>Color Doppler duplex US</td>
<td>53 M</td>
<td>2.3 × 2.7 × 4 cm</td>
<td>Intra-articular corticosteroid injections, neuropathy had improved at follow-up</td>
</tr>
<tr>
<td>Electrophysiologic tests, MRI, surgical exploration</td>
<td>... M</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>MRI for presence of cyst; EMG for entrapment symptoms</td>
<td>50 M</td>
<td>2-cm cyst medial to left posterior tibial n. (at time of surgery)</td>
<td>Synovectomy and decompression of the branch of the left posterior tibial n. to the medial head of gastrocnemius; return of function at 3 months and improvement in muscle bulk at 1 year</td>
</tr>
<tr>
<td>Venography revealed DVT, nerve conduction and EMG studies showed tibial neuropathy</td>
<td>60 M</td>
<td>...</td>
<td>Bed rest, analgesics, anticoagulation for DVT; knee joint aspiration and intra-articular predinsone injection to treat entrapment neuropathy with some improvement in EMG at 3 months</td>
</tr>
<tr>
<td>Physical examination</td>
<td>55; 86 M</td>
<td>...</td>
<td>Synovectomy and extirpation of cyst; follow-up was unremarkable for both patients</td>
</tr>
<tr>
<td>US, radionuclide scans, contrast arthrograms</td>
<td>51-66 3 M, 2 F</td>
<td>...</td>
<td>Intra-articular steroid injections to relieve pain and gradual return of motor and sensory function in four patients; one patient required synovectomy</td>
</tr>
</tbody>
</table>
The anatomic relationship of structures in the popliteal fossa and the effect of lateral cyst migration are illustrated in Fig 3.

Tibial neuropathy due to popliteal cyst entrapment, illustrated in patient 2, was documented in 16 patients (22% of the reports in the literature) and is a common manifestation of neurovascular compression because it is the most superficial and medial structure in the popliteal fossa (Table I). The results of our search demonstrated that pseudothrombophlebitis secondary to venous compression is most frequently reported, with 46 patients total (63%; Table II). Popliteal vein compression can often mimic the symptoms of DVT or ultimately result in thrombosis, or both, as seen in patient 1.

No evidence of thrombus is detected in most patients with Baker cyst presenting with pseudothrombophlebitic symptoms; however, DVT was found in three patients. One patient in the report by Chaudhuri et al presented with true thrombosis of the calf, which was most likely due to the increased pressure in the vein caused by the popliteal cyst. Both patients described by Gordon et al were treated for DVT based on filling defects detected by venography. None of the patients encountered in our search experienced venous thromboembolism. Finally, isolated arterial compression is the least common phenomenon reported in the literature, with only five patients representing 7% of the patients uncovered in our search (Table III). Two patients with arterial occlusion also exhibited nerve symptoms, but it is not clear whether these were due to compression by the cyst or ischemia. None of the case reports described concurrent symptoms of venous compression.

Cases demonstrating cystic rupture and the development of compartment syndrome, an emergency condition causing compression of all neurovascular and muscular structures, are described in Table IV and represent 4% of the total. Compartment syndrome, which developed emergently in patient 1, requires immediate surgical intervention to prevent permanent morbidity to the affected limb. Reports of other comorbid conditions, such as an adventitial cyst, synovial sarcoma, or multiple compression phenomena are exceedingly rare, comprising 4% of published case reports meeting our search criteria (Table V).

The cases encountered in the literature spanned several decades, and various techniques were used to identify and diagnose the Baker cyst lesion. Methods included MRI, Doppler ultrasound imaging, fluid analysis, electromyography, physical examination, and contrast imaging (including arthrography and venography). The most frequently used methods for diagnosing Baker cyst nerve entrapment were MRI and Doppler ultrasound imaging. For vascular compression syndromes, contrast imaging appeared most often among diagnostic methods.

**DISCUSSION**

The typical presentation for laterally migrating cysts includes any neural or vascular symptoms, or combination...
Compression occurs as the cyst grows in size and occasionally expands laterally to compress the tibial nerve, which is the most medial and superficial component of the neurovascular bundle. Rarely, the cyst expands superiorly and compresses the sciatic nerve. For tibial nerve compression, gastrocnemius muscle atrophy can be observed. In addition, as the cyst develops, the patient may experience other clinical symptoms suggestive of neuropathy, including pain, burning sensations, and paresthesias. As shown in the above patients, however, relief of pressure can resolve these symptoms and restore tibial nerve function.

Owing to the anatomic location of the nerve, entrapment neuropathy is a frequently observed symptom of this condition; however, the vascular components of the popliteal vasculature are also subject to compression, including the vein, which is next most medially located and easily compressible compared with the artery, which is most lateral and least frequently involved. In rare instances in which the popliteal artery is compressed by the Baker cyst in a pulsatile fashion, the patient can experience lower extremity claudication due to intermittent ischemia to the limb. Compression of the vein can lead to pseudothromboophlebitic symptoms, including swelling, lymphedema, pain, and discomfort. If there is bleeding into the cyst or cystic rupture, laboratory tests may reveal a positive D-dimer, which could lead to misdiagnosis and unnecessary and possibly harmful treatment with anticoagulant therapy. The resultant compartment syndrome from the accumulating hematoma can result in muscle death and limb loss and requires immediate surgical intervention. If true thrombosis of the vein occurs, as in patient 1, the long-term clinical course may ultimately lead to postthrombotic syndrome, which develops in as many as one-third to one-half of patients with DVT. Chronic insufficiency of lower limb veins causes significant morbidity, including swelling, skin changes, and chronic pain.

Combinations of these are most frequently encountered in the development of compartment syndrome secondary to a ruptured cyst. As the cyst expands, it can potentially dissect between the gastrocnemius and soleus muscles or between the medial and lateral heads of the gastrocnemius muscle, and ultimately rupture. Cyst rupture often mimics thromboophlebitic symptoms, including swelling and pain of the affected limb. We identified several patients in our center with cyst rupture and concomitant DVT but did not include them in this study because the cyst was not compressing the popliteal vein and was not directly responsible for the thrombus.

### Table II. Continued

<table>
<thead>
<tr>
<th>Method of diagnosis</th>
<th>Age</th>
<th>Sex</th>
<th>Size of cyst</th>
<th>Treatment/patient follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI</td>
<td>67</td>
<td>F</td>
<td>Dimensions of original cyst unknown</td>
<td>Bed rest, leg elevation were advised; patient recovered spontaneously</td>
</tr>
<tr>
<td>Arthrography and venography</td>
<td></td>
<td></td>
<td>Dimensions not given, but arthrographs and MRIs shown</td>
<td></td>
</tr>
<tr>
<td>Arthrography or US X-ray</td>
<td>66, 83</td>
<td>F</td>
<td>Dimensions not given, but plain films provided</td>
<td>1. Conservative, reaspiration, responded well. 2. Antibiotic therapy for Neisseria infection, conservative treatment</td>
</tr>
<tr>
<td>Arthrography, US, CT, radionuclide scanning</td>
<td></td>
<td></td>
<td>Dimensions not given, but US images shown</td>
<td></td>
</tr>
<tr>
<td>US, arthrogram showed ruptured cyst</td>
<td>77/63</td>
<td>M/F</td>
<td>Dimensions not given, but US images shown</td>
<td>Aspiration of cyst (eventually ruptured), steroid treatment and anticoagulant therapy</td>
</tr>
<tr>
<td>US, joint fluid needle aspiration</td>
<td>64</td>
<td>M</td>
<td>2 × 5 cm</td>
<td></td>
</tr>
<tr>
<td>Arthrogram and sonogram</td>
<td>46</td>
<td>M</td>
<td>Dimensions not given, but plain films provided</td>
<td></td>
</tr>
<tr>
<td>Physical examination</td>
<td>45-74</td>
<td>M</td>
<td>Dimensions not given, but plain films provided</td>
<td>Average follow-up: 2 years</td>
</tr>
</tbody>
</table>
The relative reporting of each encountered compression syndrome reflects the anatomy and compressibility of the neurovascular bundle contents; however, their incidence may be disproportionately represented due to reporting bias of this rare condition and absence of prospective data. Because the consequences of undiagnosed DVT are potentially catastrophic and anticoagulant therapy is not without complications, identifying the cause of thrombophlebitic symptoms in the patient is medically urgent, and therefore, the differential diagnosis becomes paramount.

Isolated tibial neuropathy due to Baker cyst, which is comparatively less serious and emergent, may be misdiagnosed or undetected and therefore under-represented in the literature. The decision to submit a case report, then, is at the discretion of the encountering physician and is not necessarily representative of the overall clinical experience with this syndrome.

CONCLUSIONS

Accurate diagnosis is crucial for determining the course of treatment for the patient. Intervention can be surgical or conservative, depending on whether there is precipitating knee injury ultimately responsible for the development of the cyst. Because rupture of the cyst may lead to the development of a critical compartment syndrome, patients with an elevated risk to develop venous thrombosis and pulmonary embolism should be carefully monitored because their management and treatment is informed by their potential to bleed into a synovial cyst. A complete differential diagnosis for pseudothrombophlebitic syndrome of the lower limb that includes a Baker cyst should involve venous duplex scanning and ultrasound imaging to rule out thrombosis and avoid unnecessary anticoagulation and potential bleeding into the cyst. In our patients, development of compartment syndrome due to cyst rupture necessitated fasciotomy, but nonsurgical management of unruptured cysts may be achieved. Management of a prior knee injury that resulted in cyst development may prevent or abate growth and spread of the popliteal cyst.

Because of the rarity of this condition, the cumulative experience with Baker cyst compression phenomena is short, and therefore, no particular treatment recommendations can be made. The cases recorded in the literature are also limited in their length of follow-up, with a maximum reported of up to 2 years. Baker cyst is by definition a chronic condition, and in the prevention of cyst recurrence, ultimately patient management must include long-term monitoring of symptoms.

Table III. Case reports of popliteal artery compression due to Baker cyst

<table>
<thead>
<tr>
<th>Year</th>
<th>Findings</th>
<th>Method of diagnosis</th>
<th>Patient demographics</th>
<th>Treatment/patient follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prichard21 (1990)</td>
<td>Transmittal of pulsation through cyst mimic popliteal artery aneurysm</td>
<td>US of popliteal fossa showed a mass associated with the artery, arthrography showed torn medial meniscus and Baker cyst</td>
<td>52 F</td>
<td>Surgical excision of cyst, normal pulses and no vascular symptoms at 6 months</td>
</tr>
<tr>
<td>Olcott23 (1986)</td>
<td>No popliteal or pedal pulses felt as caused by artery compression by Baker cyst</td>
<td>Arteriogram and surgical exploration of popliteal fossa</td>
<td>58 M</td>
<td>Artery surgically freed from cyst (pathologically examined and confirmed to be a popliteal cyst), complete resolution of symptoms</td>
</tr>
<tr>
<td>Krag22 (1982)</td>
<td>Pain in left calf, numb left foot, and claudication upon walking resulting from direct pressure on the artery by the cyst</td>
<td>Arteriogram and surgical exploration of popliteal fossa</td>
<td>56 M</td>
<td>Cysts excised, artery opened and cleared of thrombotic material; patient’s symptoms improved.</td>
</tr>
<tr>
<td>Schlenker24 (1974)</td>
<td>Acute ischemia, pain and numbness of the leg secondary to occlusion of the popliteal artery (and nerve) by two popliteal cysts</td>
<td>Arteriogram and subsequent surgical exploration of popliteal fossa</td>
<td>39 M</td>
<td>Affected artery and cyst resected and graft from small saphenous vein used for reconstruction, some recovery but distal pulses were still weak</td>
</tr>
<tr>
<td>Robb25 (1960)</td>
<td>Pain in ankle upon walking and weak right distal pulses, surgical resection revealed cyst compressing artery and partial thrombosis of artery</td>
<td>Arteriogram</td>
<td>1 cm in diameter at largest point</td>
<td></td>
</tr>
</tbody>
</table>

F, Female; M, male; US, ultrasound.
Despite the limitations of this review, it does illustrate the importance of including this syndrome in the differential diagnosis of patients presenting with symptoms that could be sequela of popliteal fossa compression syndromes. It also demonstrates that patient follow-up is necessary to monitor recovery of nerve function and vascular status. The patients in our experience, as described above, regained motor and sensory function of the affected lower limb, indicating recovery from tibial neuropathy; however, the possibility of development of long-term complications suggestive of venous insufficiency remains.

**AUTHOR CONTRIBUTIONS**

Conception and design: NL
Analysis and interpretation: JES, NC, NL
Data collection: JES, NC, NL
Writing the article: JES, NC, NL
Critical revision of the article: JES, NC, NL
Final approval of the article: JES, NC, NL
Statistical analysis: NL
Obtained funding: Not applicable
Overall responsibility: NL

---

**Table IV. Case reports of popliteal cyst causing posterior compartment syndrome**

<table>
<thead>
<tr>
<th>Year</th>
<th>Findings</th>
<th>Method of diagnosis</th>
<th>Patient demographics</th>
<th>Treatment/patient follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dunlop(^{38}) (1997)</td>
<td>Patient was treated with anticoagulants for a suspected DVT, which resulted in bleeding from Baker’s cyst into the calf causing compartment syndrome</td>
<td>Compartment syndrome was clinically suspected after anticoagulation</td>
<td>58 M</td>
<td>Compartment syndrome was treated by fasciotomy and excision of necrotic gastrocnemius muscle; discharge after 5 weeks with full recovery and no deficits</td>
</tr>
<tr>
<td>Petros(^{39}) (1990)</td>
<td>Patient was treated with anticoagulants for a suspected DVT, which precipitated compartment syndrome</td>
<td>Venography and arthrography</td>
<td>35 M</td>
<td>Compartment syndrome was treated by four-compartment fasciotomy, swelling and gastrocnemius hemorrhage prolonged recovery; after 4 months there were some deficits in the sensory distribution of the left saphenous nerve</td>
</tr>
<tr>
<td>Scott(^{33}) (1977)</td>
<td>Patient’s calf findings were misdiagnosed with septic arthritis and thrombophlebitis. The final diagnosis was Reiter syndrome with bilateral popliteal cysts complicated by left-sided posterior compartment syndrome, secondary to dissecting popliteal cyst</td>
<td>Arthrogram</td>
<td>51 M</td>
<td>Decompression by knee aspiration was carried out, which was enough to relieve symptoms</td>
</tr>
</tbody>
</table>

*DVT,* Deep vein thrombosis; *F,* female; *M,* male.

<table>
<thead>
<tr>
<th>Year</th>
<th>Findings</th>
<th>Method of diagnosis</th>
<th>Patient demographics</th>
<th>Size of cyst</th>
<th>Treatment/patient follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ayoub(^{37}) (2000)</td>
<td>Bilateral cysts, with synovial sarcoma arising within causing discomfort but no compression syndromes. Recurrence of cyst with synovial sarcoma within</td>
<td>US, then MRI</td>
<td>13 F</td>
<td>6 × 3 cm (MRI)</td>
<td>Excision, patient continues to be monitored</td>
</tr>
<tr>
<td>Schroë(^{35}) (1988)</td>
<td>Communication of adventitial cyst with synovial cyst</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Ikeda(^{42}) (1984)</td>
<td>Popliteal cyst in left leg. Vein compression caused venostasis, artery compressed when knee flexed</td>
<td>X-ray</td>
<td>53 M</td>
<td>4.5 cm (palpation)</td>
<td>Excision, uneventful post-op</td>
</tr>
</tbody>
</table>

*F,* Female; *M,* male; *MRI,* magnetic resonance imaging; *US,* ultrasound.
REFERENCES


Submitted Mar 24, 2011; accepted Jul 20, 2011.