Characteristics	Ganglionic cyst	Synovial cyst (Baker's cyst)	Meniscal cyst
Related structure	Joint-related ligament and tendon Non-specific intramuscular origin	Gastrocneio-semimembranous bursa	Meniscal tear or degeneration
Knee joint association	±	+	+
Lining cell	Flat spindle-shaped cell (no synovial lining cell)	Synovial lining cell	Flat spindle-shaped cell (no synovial lining cell
Induce joint limitation of motion or nerve compression	Possible	Possible	Possible
Magnetic resonance imaging finding	Hyperintense To skeletal muscle on T2-weighted image	High signal On T2-weighted and low signal on T1-weighted image	Tear of meniscus with a connection to the cys
Recommended treatment	Excision or non-surgical option ^{a)} (e.g., needle aspiration)	Excision or arthroscopic debridement	Excision or arthroscopic debridement

and medial and lateral sural nerves. A Baker's cyst in this area can cause lower leg pain, paresthesia, and muscle weakness by the compression of the tibial nerve [5]. The authors were careful not to damage the lateral and medial sural nerves while dissecting the lateral head of the gastrocnemius muscle. Surgeons should have a precise plan to explore and dissect the popliteal fossa and to not damage vital structures; further, they should explain the possibility of a nerve injury to patients, preoperatively.

A case of an intramuscular ganglionic cyst has been rarely reported. Moreover, this cyst is easily mistaken for a Baker's cyst or a meniscal cyst in the popliteal fossa. Surgeons should be aware of the differential diagnosis among cystic masses for definite treatments. The details of the differential diagnosis are provided in Table 1. Preoperative imaging studies can be helpful, particularly MRI. Here, we have reported a rare case of an intramuscular ganglionic cyst and the distinction among the popliteal cystic masses in order to help with their diagnosis and treatment.

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Bimodal Treatment of a Huge Hypervascular Neurofibroma on the Groin

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Neurofibromatosis type I (NF-I) is an autosomal dominant genetic disorder. The corresponding vascular disease is referred to as NF-I vasculopathy, which includes aneurysms, stenoses, and arteriovenous malformations (AVMs) [1-3].



Fig. 1. Hypervascular neurofibroma in the right inguinal area. Spontaneous bleeding was noted.

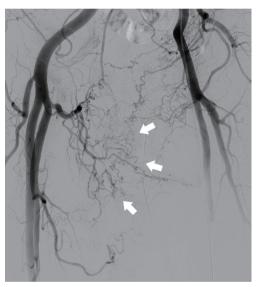


Fig. 2.

Enhanced abdominal computed tomography showing a radiolucent mass (asterisk) due to extensive vascularity separated from the inguinal portion of the mass (circle).

Although the exact cause remains unclear, it has been proposed that vascular dysplasia or cellular proliferations are the main causes of these conditions. In addition, vascular lesions in the trunk and extremities are rare [3].

Here, we report a case of a huge hypervascular neurofibroma on the groin that extended to the pelvic cavity and was initially misdiagnosed as cutaneous AVM. Two methods, namely embolization and surgical removal, were selected to treat this case, considering the hypervascular nature and relevant anatomy.



A 64-year-old male with a history of NF-I visited our institution's outpatient department and presented with swelling and spontaneous bleeding in his right inguinal area (Fig. 1). Physical examination revealed an anemic face and conjunctiva. Laboratory values showed anemia with a hemoglobin level of 9.5 g/dL; these results were thought to be caused by the bleeding nature of the mass. A contrast-enhanced computed tomography (CT) scan showed a mass with a rich vascular supply that extended from the pelvic cavity to the right inguinal area. In addition, a septation appeared to separate the pelvic portion of the mass from the inguinal mass (Fig. 2). Angiography was performed to identify the source vessel of the vascular tumor. Conventional angiography showed that the vascularity of the pelvic

Fig. 3.

Conventional angiography revealed multiple vessels from the internal iliac artery and the deep femoral artery (white arrows) that were feeding the mass.



Fig. 4. Postoperative 6-month

photograph. The defect was covered with a contralateral anterolateral thigh free flap.

> portion of the mass was supplied by two major feeding vessels, which originated from the deep femoral artery and the medial femoral circumflex artery. The inguinal portion of the mass was thought to be supplied by the small feeding vessel from the medial femoral circumflex artery (Fig. 3). We planned a bimodal approach to remove the mass on the basis of the impression of the cutaneous AVM associated with neurofibromatosis. First, radiographic intervention was performed to embolize the two major feeding vessels that supplied the pelvic portion of the mass. Transfemoral catheterization was applied to branches of the right deep femoral artery and medial femoral circumflex artery, which were superselected and embolized with particles measuring 255–250 µm (Contour, Boston Scientific, Cork, Ireland). Nonvisualization of the hypervascular mass was confirmed after embolization. However, spontaneous bleeding was persistent in the inguinal area, and surgical excision was planned three days after the patient was stabilized.

> During the operation, the medial femoral circumflex artery that branched from the deep femoral artery was dissected, clipped, and prepared as a recipient artery for the free flap. The mass was dissected from the periphery to the central portion and detached from the adductor brevis muscle. The inguinal mass was completely segregated from the pelvic mass.

The anterolateral thigh free flap was harvested from the contralateral thigh to cover the resultant defect. The medial femoral circumflex artery that previously supplied the inguinal portion of the mass was prepared as a recipient artery for anastomosis along with the two venae comitantes. After anastomoses of one artery and two veins with the aid of a microscope, the flap margin was approximated and moderately compressed with bolster sutures, and drains were inserted in several planes to minimize postoperative bleeding.

Although there was hematoma accumulation on postoperative day 3, evacuation of the hematoma was successfully preformed and the flap survived completely (Fig. 4). Laboratory values recovered to normal within two weeks, there was no sign of recurrence during the two years of follow-up, and sustained regression of the embolized pelvic portion of the mass was also maintained (Fig. 5).

Histopathological examination confirmed the diagnosis of hypervascular neurofibromatosis with no evidence of arteriovenous anastomoses within the mass.

NF-I is an autosomal dominant condition that presents with multiple symptoms of café au lait spots, iris hamartoma, and skin neurofibroma [3]. Other clinical features include short stature, skeletal abnormalities, and various vascular pathologies that affect medium- to large-sized vessels [1,4,5].

Vascular manifestations of NF-I are referred to as NF-I vasculopathies. The pathogenesis is currently unknown, but this condition can manifest as aneurysms, stenoses, and AVMs [1]. Most patients with NF-I vasculopathy do not recognize the symptoms but usually have multiple simultaneous vascular abnormalities [1,2,4]. The aneurysm and stenosis pathophysiologies are thought to be similar to the pathophysiology of cutaneous neurofibroma, which consists of cellular proliferation, degeneration, healing, smooth muscle loss, and fibrosis [1,2]. An article reported that the most common vasculopathy in NF-I patients is isolated renal artery disease, followed by mesenteric artery stenosis/aneurysm and aortic dissection. AVM does not occur as frequently, but most cases arise in the cervical vertebral artery and lumbar vertebral artery, and an occurrence in the trunk, extremities, and face is very rare [3]. Systemic manifestations from this vascular pathology, such as hypovolemic shock, are rare, but bleeding is difficult to control once it has started.

NF-1 vasculopathy conditions such as aneurysm or psuedoaneurysm are generally treated with transcatheter embolization of the feeding artery, which is usually limited to one or two major vessels. However, AVM has multiple tortuous vessels that feed the mass, which makes vascular intervention difficult. Moreover, in the case that the mass is superficially located and the feeding vessel is too small to perform selective embolization, a surgical approach is preferred.

Although the pathologic diagnosis was not AVM in our case, the massive bleeding nature and clinical diagnosis from the preoperative evaluation included a differential diagnosis of AVM. Based on the finding that the inguinal portion and the pelvic portion of the mass were septated, a bimodal approach combining selective embolization of the pelvic mass and surgical excision of the inguinal portion of the mass was successfully performed.

Here, we have reported successful treatment of a severe case of hypervascular neurofibroma with intractable bleeding, which was successfully controlled with selective embolization and surgical removal followed by reconstruction with a free flap. Moreover, because a hypervascular neurofibroma has clinical features similar to those of an AVM, our bimodal approach could be a good option for the treatment of a huge AVM that is caused as a complication of neurofibromatosis.

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Giant Extrapleural Solitary Fibrous Tumor of the Thigh

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A solitary fibrous tumor (SFT) is an uncommon tumor that arises from primitive fibroblast-like cells in the connective tissue [1]. It characteristically

Fig. 5.

Postoperative 6-month enhanced abdominal computed tomography. The regression of the embolized pelvic portion of the mass is maintained.