Adventitial Cystic Disease of the Radial Artery
Two Case Reports and a Review

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Abstract
A 29-year-old female presented with pain and a palpable mass in the left wrist. Imaging demonstrated a multiloculated cystic mass adjacent to the radial aspect of the scaphoid, which was intimately associated with and appeared to arise from the wall of the radial artery and its dorsal branch. The mass was surgically resected. The histological analysis confirmed the presence of adventitial cystic disease (ACD) of the radial artery. In addition, within a year time span, a second 34-year-old male patient presented with a palpable mass in the right hand. Imaging demonstrated a cystic mass encasing the dorsal carpal branch of the radial artery and its terminal vessels to the thumb and index finger. The diagnosis of ACD was raised based on MR imaging. Histological analysis confirmed the presence of an adventitial cyst. ACD of the arteries is a rare disorder of unknown etiology, which usually involves the popliteal artery. Less common sites of involvement include the external iliac, common femoral, radial, and ulnar arteries. To our knowledge, there have only been six previous case reports of adventitial cystic disease involving the radial artery. We report two additional cases of adventitial cystic disease of the radial artery including a review of the imaging features, histology, differential diagnosis, potential pathogenesis, and treatment.

Case Report 1
A 29-year-old female presented with a history of a progressively enlarging mass for several months along the radial aspect of the wrist, which was causing increasing discomfort. The patient had no history of trauma or other prior medical issues. On examination, the patient had a palpable mass in the radial aspect of the wrist, which was not significantly tender to palpation. There was no erythema or warmth on inspection. Plain radiographs were obtained, which showed a soft tissue mass adjacent to the radial styloid and scaphoid without evidence of soft tissue calcification or osseous abnormality (Fig. 1). An MRI was performed for further evaluation; it showed a 2 cm diameter multiloculated, cystic mass centered about the radial aspect of the scaphoid, which extended from the radial styloid to the first carpometacarpal joint (Fig. 2). The cystic mass was intimately associated with and appeared to be arising from the wall of the distal radial artery, encasing it. Surgical resection was performed. The operative report noted that the mass was intimately wrapped around the main branch of the radial artery, and the mass had to be decompressed in order to dissect it off of the artery. Additionally, the cystic mass had a connecting stalk to the scaphoid-trapezium-trapezoid (STT) joint. Grossly, the specimen consisted of multiple irregular fragments of iliofemoral artery stenosis caused by the encroachment of the lumen by the cystic changes in the adventitia. Occasionally, patients have presented with a mass lesion in other sites, including a few case reports of involvement in the brachial, ulnar and radial arteries, the axillary artery, and the saphenous veins in the ankle. To our knowledge, there are only six previous case reports of adventitial cystic disease involving the radial artery. We report two additional cases of adventitial cystic disease of the radial artery including a review of the imaging features, histology, differential diagnosis, potential pathogenesis, and treatment.
soft grey white fibromembranous tissue. The histological analysis using H&E, trichrome and elastin stains showed a fibrous wall cyst with mucoid degeneration and elastic fiber disintegration (Fig. 3). These findings in correlation with surgical and radiological appearance confirmed the presence of cystic adventitial disease of the radial artery. Following the surgery, the patient did well without recurrence of symptoms or need for follow-up imaging.

**Case Report 2**

A 34-year-old male presented with a history of a painless palpable mass over the first and second metacarpals, which progressively became more prominent over the course of several months. No tenderness to palpation, erythema, or warmth was present. MR imaging was performed, which demonstrated a 1.5 cm cystic mass encasing the dorsal branch of the radial artery and its terminal vessels to the thumb and index finger (Fig. 4). Surgical resection was performed and histological analysis demonstrated positive trichrome and elastic stains consistent with adventitial cystic disease (Fig. 4).

**Discussion**

Involvement of upper extremity vessels by adventitial cystic disease is extremely rare, including a few case reports of occurrence in the radial artery. To the best of our knowledge, only six other cases of adventitial cystic disease of the radial artery have been discussed.1-3

The clinical presentation of ACD in the radial artery is

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**Figure 1** Radiographic PA view of the wrist shows a soft tissue mass (white arrow) adjacent to the radial styloid and scaphoid without evidence of associated calcification or pressure erosion.
usually with a palpable mass, sometimes described as pulsatile and painful. This is in contrast to ACD in the popliteal artery, where claudication is the most common clinical presentation. The explanation for the difference in clinical presentation between lower and upper extremity ACD has not been discussed in the literature, likely because it is such a rare lesion in the upper extremity, that perhaps there are not enough cases to accurately make this distinction.

Imaging of ACD is best demonstrated with magnetic resonance imaging (MRI), which has excellent contrast resolution for differentiating the fluid content of lesion from the adjacent soft tissue and osseous structures. The fluid contents of the adventitial cyst are typically hyperintense on T2-weighted images and variable in signal on T1-weighted images depending on the amount and composition of its internal content.6 The cyst will typically have thin smooth septations. Intravenous contrast is not usually necessary to evaluate this lesion. However, if there is concern for underlying neoplasm or superimposed infection with abscess formation, contrast should be administered to further characterize the lesion. Adventitial cysts will typically show a rim of enhancement in the thin walls of the cysts and no enhancement of its internal mucinous content.7 A neoplasm will show areas of central enhancement, and an abscess will show a peripheral usually thick rim of enhancement as well as perilesional inflammatory changes. The combination of clinical presentation, laboratory tests, and MRI are usually sufficient to differentiate between these potential differential diagnoses.

In contrast, a ganglion may clinically present identical to ACD as a palpable or painful mass with very similar MRI features. The signal intensity of the cystic fluid of an adventitial cyst is indistinguishable on MRI from a ganglion. There can sometimes be communication of the joint with the adventitial cyst as previously discussed in this case report, which has also been reported in popliteal artery adventitial cysts communicating with the knee joint.8,9 Therefore, the presence of communication of the cystic mass with an adjacent joint does not help in distinguishing between an adventitial cyst and a ganglion, as both lesions can demonstrate this finding. For these reasons, a ganglion will usually be the main consideration in the differential diagnosis for adventitial cystic disease. The most helpful MRI finding to differentiate a ganglion from an adventitial cyst is the location of the cystic mass. In the setting of cystic adventitial disease, the cyst is intimately associated with the vessel arising from its wall and encasing it. A very useful MRI finding is demonstration of pulsation artifact along the phase encoding axis arising from the lumen of the involved vessel, which is encased by the adventitial cyst. A ganglion cyst may abut and exert mass effect upon an adjacent vessel but will not typically encase it within its lumen (Fig. 5A).

The other main differential diagnostic consideration is the possibility of an aneurysm or a pseudoaneurysm. Patients with pseudoaneurysm usually have a traumatic event or procedure to the area of clinical concern. This may or may not be helpful in differentiating from an adventitial cyst since both lesions may theoretically be the result of trauma, as discussed in the proposed theories of the pathogenesis of adventitial cysts later in this discussion. On imaging (MRI or ultrasound), it may be difficult to distinguish between ACD and a partially thrombosed pseudoaneurysm based on
morphology; however, a multilobulated cystic appearance is more characteristic of ACD whereas a partially thrombosed aneurysm has a more uniform unicameral cystic pattern. MR angiography demonstrates pseudoaneurysm as an eccentric saccular mass with an irregular thickened wall communicating with an artery. On contrast MR angiography, the mass may contain central vascular flow indicated by hyperintense signal on all sequences secondary to contrast in the lumen of the pseudoaneurysm. Varying MR signal characteristics in the pseudoaneurysm reflects the status of adventitial cystic disease. The media layer of the artery contains elastic fibers represented in red. The adventitial layer is represented in purple. A, A ganglion cyst may exert mass effect upon an adjacent artery but its wall (blue) will not typically be elastin stain positive. B, The elastin stain (red) demonstrates disrupted elastic fibers throughout the entire cyst wall in ACD. C, A pseudoaneurysm may only have a positive elastin stain at its neck (the site of traumatic disruption of the arterial wall). See color illustration at www.nyuhjdbulletin.org.
vascular flow, hemorrhage, and thrombosis. In equivocal cases, Doppler ultrasound may be of help because it will detect turbulent flow into the lumen of the pseudoaneurysm. Typically, blood flow into the pseudoaneurysm concentrates along one wall and flow out of the pseudoaneurysm concentrates along the opposite wall. This creates a characteristic “yin-yang” appearance on color Doppler, where one half of the lumen appears red and the other half appears blue. Another characteristic pattern of blood flow of a pseudoaneurysm is a “to-and-fro” pattern of flow in the neck, which is seen on spectral Doppler waveform. This refers to systolic flow into the pseudoaneurysm appearing on one side of the pulsed Doppler baseline and diastolic flow out of the pseudoaneurysm appearing on the other side of the baseline. Turbulent flow inside a pseudoaneurysm will translate on MR imaging as an area of internal signal void.

Histological diagnosis of adventitial cystic disease is based on H&E, trichrome, and elastin stains. H&E staining demonstrates mucoid degeneration within the fibrous wall of the cyst. The fibrous nature of the cyst wall is confirmed with the trichrome stain. The elastin stain demonstrates disrupted elastic fibers throughout the entire cyst wall, which indicates that the cyst emanated directly from the adventitial layer of an artery (Fig. 5B). In contrast, a pseudoaneurysm may only be elastin stain positive at the site of its neck, point of traumatic disruption of the arterial wall (Fig. 5C). A ganglion cyst will typically not stain positive with elastin, as it would need to erode into the vessel to cause disruption of its elastic fibers, in which case the elastin stain would only be positive at that specific site of contact and erosion of the ganglion into the arterial wall.

The etiology of ACD is unknown. There are several proposed etiologies, including traumatic, systemic, ganglion, and developmental. The traumatic theory postulates that repeated trauma results in stretching and distortion of the vessel wall, which leads to destruction and cystic degeneration of the adventitia. This theory fails to explain the occurrence of ACD in the absence of history of trauma. The systemic theory suggests that there is an underlying mucinous or myxomatous systemic degenerative condition in patients with ACD. However, long-term follow-up of patients with ACD has failed to demonstrate systemic manifestation of disease. The ganglion theory suggests that adventitial cysts arise as capsular synovial structures that track along vascular structures to involve the adventitia of the adjacent vessel, either during development or later in life. The developmental theory suggests that mucin-secreting cells derived from mesenchymal elements of the adjacent joint become included in the adventitia of the artery or vein during development. This theory is supported by the reported demonstration of a communication between the cyst and the adjacent joint in many cases of ACD. In one of our cases, there was communication of the cystic mass with the adjacent scaphoid-trapezium-trapezoid (STT) joint, seen on MRI and during surgical resection of the mass. A combination of the developmental theory and the ganglion theory has also been suggested in which a joint-related ganglion like structure is incorporated into the developing vessel during embryological development which enlarges over the years due to secretions from synovial rests within the cyst. However, adventitial cysts have a higher concentration of hyaluronic acid than synovial fluid, which supports the developmental theory that inclusion of mucin secreting mesenchymal cells has occurred during embryological development.

Treatment of adventitial cystic disease is preferably cyst decompression and excision with preservation of the involved vessel. Percutaneous aspiration of the cyst is usually not successful because the cyst fluid reaccumulates with time. In extensive cases where there is no possibility of excision without compromising the involved vessel or in cases that are refractory to decompression and excision, replacement with a venous graft is the recommended treatment option.

In conclusion, adventitial cystic disease of the radial artery is rare, and only six other cases have been reported. We report two more cases, which were surgically resected, one of which was shown intraoperatively to communicate with the adjacent scaphoid-trapezium-trapezoid joint. Although the etiology of this lesion is unknown, there are several proposed mechanisms, many of which are not supported by the evidence available. The most plausible theory and the one we support is that ACD develops as a result of aberrant inclusion of mucin secreting mesenchymal cells in the adventitial wall of a vessel during joint development. In both of our cases, the radiologists had a key role in the diagnosis of ACD of the radial artery given the typical encasement of the involved vessel by the adventitial cysts on MR images prompting the performance of elastin and trichrome stains. Communication with the surgeon and the pathologist prior to surgical excision and histological analysis of the specimen is crucial in order to obtain the appropriate stains in cases in which there is high preoperative suspicion of an adventitial cyst.

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Disclosure Statement
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