The To and Fro Sign: The Hallmark of Pseudoaneurysm

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A pseudoaneurysm, or communicating hematoma, usually results from penetrating trauma of a native vessel with subsequent formation of a cavity contained by the adventitia, perivascular tissue and fibrous tissue originating from organization of the hematoma. Less commonly, pseudoaneurysm can result from rupture of a native vessel or leakage at the site of a surgical anastomosis. Common femoral artery pseudoaneurysms complicating catheterization procedures are by far the most common. However, pseudoaneurysms can occur anywhere. Patients with pseudoaneurysms usually present with a mass in the area of trauma that may be pulsatile. The objective of imaging is to differentiate between hematomas associated with pseudoaneurysm and those that are not. Whereas hematomas resolve spontaneously, a pseudoaneurysm can potentially rupture and therefore must be identified, closely monitored and in most instances, treated. Methods of treatment include ultrasound Doppler-guided compression and thrombin injection [1,2], transcatheter stent-graft placement, and surgical repair.

Ultrasound is the imaging modality of choice to differentiate between pseudoaneurysm and non-communicating hematoma. The characteristic appearance of pseudoaneurysm in triplex ultrasound (B-mode combined with color and pulsed Doppler spectral analysis) include the presence of a hematoma of variable echogenicity, which may represent separate episodes of bleeding and rebleeding, expansile pulsatility and detection of turbulent flow ("yin-yang" appearance) within the mass [3]. However, these findings are not always present, and may also be observed in non-communicating hematomas due to transmission of pulsations from the underlying artery.

The definitive diagnosis of pseudoaneurysm requires detection of the neck connecting the pseudoaneurysm with the injured artery, and identification within this neck of the pathognomonic "to and fro" spectral waveform pattern [4]. This pattern results from the alternating flow direction – into the aneurysm in systole and away from it in diastole.

We recently applied this sign in locations other than the CFA to differentiate between a hematoma and a pseudoaneurysm. The diagnoses were confirmed by angiography in one case and surgery in the other. In the first instance an elderly male presented to the imaging department with thigh compartment syndrome [5] following nailing of a femoral neck fracture. The diagnosis of pseudoaneurysm of a branch of the deep femoral artery was made on the basis of the presence of the to-and-fro sign between the feeding artery and the pseudoaneurysm (Figure). Angiography revealed a pseudoaneurysm and active extravasation from a branch of the deep femoral artery corresponding in location to the ultrasound finding. Symptoms resolved following transcatheter embolization of this branch. The second case involved a pulsatile mass in the left brachial region following transbrachial peripheral angiography. The diagnosis of pseudoaneurysm was made on the basis of the to-and-fro sign, and confirmed upon subsequent surgical repair.

In view of the increasing frequency of percutaneous catheterization procedures in arteries other than the CFA, as well as

Transverse ultrasound image of the proximal thigh. Within the 7 cm hematoma (h) the DFA is imaged in cross-section (open arrow). The neck of the pseudoaneurysm (n) connects the DFA with lumen of the pseudoaneurysm (arrow head). A Doppler cursor placed on the neck displays the typical to (above the zero line) and fro (below the zero line) flow.

CFA = common femoral artery
the application of ultrasound to the initial evaluation of penetrating trauma, the presence of the to-and-fro sign should be included in the sonographic examination of these cases whenever technically feasible. It is stressed that whereas detection of this sign is pathognomonic for the diagnosis of pseudoaneurysm, its absence does not entirely exclude this diagnosis.

References

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Minicapsule

How can care of acute bronchitis be improved? Bronchodilators should be offered for symptomatic relief of troublesome cough. Antibiotics should be avoided.

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Capsule

Anticancer drug from the sea

A compound derived from a marine organism defines a new class of anticancer drugs that targets the nucleotide excision repair system of cells. Ecteinascidin 743 (ET743) is an alkylating agent that tightly binds DNA in the minor groove. It was originally isolated from the Caribbean sea squirt, Ecteinascidia turbinata. In Phase II/III trials, the compound has shown remarkable activity against a number of advanced cancers, including sarcomas.

Takebayashi et al. show that ET743 DNA adducts are recognized by a cell's nucleotide repair system, which ends up killing the cell instead of repairing the damage caused by the adducts. What is particularly interesting, and relevant to cancer therapy, is that the killing is only seen if the repair occurs in transcribed genes. Nucleotide excision repair (NER) is a mechanism for removing and repairing lesions that distort the DNA helix, such as ultraviolet light-induced cyclobutane pyrimidine dimers or adducts caused by alkylating anti-cancer drugs. The link between ET743 and NER was uncovered by the authors by generating two ET743-resistant cell lines and showing that both have defects in the gene encoding xerodema pigmentosum (XPG), an endonuclease involved in NER. Restoring XPG activity in these cells restored their ability to repair DNA damage induced by ultraviolet light and also ET743 sensitivity. Cell lines deficient in other genes that, similar to XPG, are essential to both transcription-coupled and global genome NER were also found to be resistant to ET743. However, cells deficient in XPC, which encodes a protein that only recognizes damage in the global genome, were sensitive to ET743 killing. This finding suggested that ecteinascidin cytotoxicity is specifically associated with transcription-coupled NER. In each cell tested, ET743 cytotoxicity correlated with functional transcription-coupled NER activity as well as with the presence of single-stranded DNA breaks in transcribed genes. Based on these observations, the authors suggest that when the NER system detects ET743 adducts in genes it makes single-stranded DNA breaks on either side of the lesion. These breaks are what cause the cells to die.

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