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Title: Imaging of Neuralgic Amyotrophy in the Acute Phase

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Abstract

Introduction/Aims

Hourglass-like constrictions (HGCs) occur in neuralgic amyotrophy (NA), but the earliest time at which they can be recognized by imaging is poorly understood. We aimed to determine the prevalence of abnormal imaging findings in the acute phase of NA.

Methods

Magnetic resonance neurography (MRN) and high-resolution ultrasound (US) examinations were performed at 5 sites. The investigation included 39 patients with acute NA who underwent imaging within 31 days of symptom onset. Correlation between imaging and electromyography (EMG) findings was measured.

Results

US was performed in 29 patients and MRN in 23; 16 patients underwent US only, 10 MRN only, and 13 had both. US and MRN showed nerve abnormalities within 1 month from NA onset in 90% of patients. HGCs were found in 74% (29/39) of the patients: 4 within 1 week, 8 within 2 weeks, 5 within 3 weeks, and 12 within 4 weeks. The earliest HGC on US was found within 12 hours, and on MRN within 3 days from symptom onset. MRN demonstrated a denervation edema pattern of affected muscles in 91% of the patients. The shortest time to observe an edema pattern on MRN was 8 days. EMG was performed in 30 patients and revealed fibrillation potentials in affected muscles in 22 (73%). A denervation edema pattern on MRN was significantly associated with the presence of HGCs both on MRN and US, and with fibrillation potentials on EMG.

Discussion

In the early phase of NA, US and MRN are useful diagnostic techniques for demonstrating nerve abnormalities.

Introduction

Neuralgic amyotrophy (NA), also known as Parsonage-Turner syndrome (1, 2) or brachial neuritis, may go undiagnosed in the acute setting (<4 weeks from symptom onset), as other etiologies, such as cervical radiculopathy or rotator cuff disorders, may produce overlapping signs and symptoms (3). This results in delayed referral and treatment. In the very early phase (<2 weeks), the diagnosis depends predominantly on the clinical presentation and physical examination, as electromyography (EMG) does not reliably reveal signs of active muscle denervation (fibrillation potentials) until 2-3 weeks (4). The time of onset of fibrillation potentials may depend on injury severity, muscle type, and the distance between the nerve injury site and the neuromuscular junction (4, 5).

High-resolution peripheral nerve imaging in NA may reveal focal abnormalities of involved nerves (6-14). An increasingly recognized (and possibly pathognomonic) finding for NA is the presence of intrinsic, hourglass-like constrictions (HGCs) of involved nerves or nerve fascicles (6-11). HGCs can be detected on dedicated peripheral nerve magnetic resonance (MR) imaging, MR neurography (MRN), and ultrasound (US), and are typically accompanied by focal enlargement (12-14), proximally and/or distally to the site of constriction, and by nerve signal hyperintensity (for MRN). The interval from symptom onset to the appearance of these imaging findings is currently unknown. Given the difficulty of confirming the clinical diagnosis within the first 2 weeks, knowledge of the timing of imaging findings would be particularly meaningful to increase diagnostic confidence in the acute stage.

We aimed to determine the prevalence of abnormal imaging findings of affected peripheral nerves and muscles of NA patients within the first month following symptom onset. We also explored associations among nerve imaging abnormalities and active denervation patterns on MRN and EMG.

Methods

A retrospective search of imaging databases for the clinical diagnosis of NA (1, 15) at five different sites (1=Lugano; 2=New York; 3=Budapest; 4=Nijmegen; 5=Liestal) over a 4-year period

(January 2017-December 2021) was performed following approval by each local ethics committee or institutional review board.

For the clinical diagnosis of NA, we used criteria published by Hannibal et al. (15) for hereditary neuralgic amyotrophy, disregarding the family history: 1 - (sub)acute onset; 2 - initial pain, usually with numerical rating scale (NRS) score ≥7; 3 - nerve damage distribution mainly in the upper brachial plexus; 4 - monophasic course, with slow recovery; 5 - preceding trauma, malignancy, diabetes mellitus and radiation excluded. The nerve damage could involve a single nerve (mononeuropathy) or multiple nerves (mononeuropathy multiplex).

We included only patients who had imaging (US and/or MRN) performed within 31 days of symptom onset (typically acute, severe pain). Patients with symptom onset immediately following trauma or alternative diagnoses established after at least 3 months' follow-up were excluded. Needle EMG was performed according to the treating physician's decision and was not considered mandatory for diagnostic confirmation or study inclusion. The presence of fibrillation potentials on EMG in clinically affected muscles was considered a sign of active denervation that supported the diagnosis.

Ultrasound investigations were conducted using Logiq E9 with a ML6-15 or L8-18 linear transducer (GE Healthcare, Milwaukee WI, United States) at sites 1 and 2; Epiq 5 with a L18-5 or eL18-4 MHz linear transducer (Philips, Amsterdam, the Netherlands) at site 3; Xporte with a 6-15 MHz linear transducer (Fujifilm Sonosite Inc. Bothel, Washington, United States) at site 4; and Apollo 300 with a linear PLT-1204BT (7.2-14 MHz) transducer (Toshiba, Tokyo, Japan) at site 5. Investigators performing US had 4 (site 1), 7 (site 2), 10 (site 3), 11 (site 4), and 7 years (site 5) of dedicated neuromuscular US experience. For all patients, the clinically affected nerves were scanned in the short-axis plane along their whole length, and the brachial plexus was also examined. In perceived areas of abnormality, longitudinal scans were also obtained.

MRN at 3 Tesla was performed at site 1 (MR Signa Pioneer, GE Healthcare, Milwaukee WI, United States), site 2 (MR750, GE Healthcare, Milwaukee WI, United States) and site 5 (Ingenia, Philips, Amsterdam, The Netherlands) using receive-only surface coils wrapped around the region of interest. At all sites, fluid-sensitive, T2-weighted Dixon fat suppression and inversion recovery

sequences were acquired in multiple planes, and intravenous gadolinium contrast was administered at the discretion of the overseeing radiologist. Fluid-sensitive sequences were used to detect nerve signal and morphologic abnormalities as well as denervation, manifested as diffusely increased signal intensity of the muscle ("denervation edema pattern"). Slice thicknesses ranged from 1-2.5 mm and in-plane resolution ranged from 0.2-1 mm, depending on the anatomic region and nerve(s) of interest. MRN exams were interpreted by either neuroradiologists (sites 1 and 5, with 4 and 11 year's MRN experience, respectively), or by a musculoskeletal radiologist (site 2, 7 year's MRN experience).

"Focal nerve enlargement" (7) was defined on US and MRN as a cross-sectional area (CSA) of the nerve at least twice that of neighboring proximal and/or distal segments (with CSA on US measured inside the hyperechoic peripheral rim of the nerve). "Hourglass-like constriction" was defined on US and MRN by focally decreased caliber of an entire nerve or fascicular bundle, confirmed on US by both longitudinal and short-axis images (7). On slow cross-sectional US scanning, the site of constriction was seen as a sudden disappearance followed by re-emergence of the nerve **(video)**.

Statistical analysis

Minimum and maximum, mean, median, and quartiles were computed for patient age and time from symptom onset to imaging. Frequency data are presented using contingency tables, and pairwise associations between variables were computed using chi-square or Fisher's exact tests, as appropriate, using Cramer's V as a measure of association. All computations were completed with Stata Version 17 (StataCorp LCC, College Station, Texas, USA).

Results

Patient characteristics are summarized in the **supplementary table**. Thirty-nine patients (15 female; 24 male) with a median age of 42 years fulfilled inclusion criteria: 16 underwent US only, 10 MRN only, and 13 both. Median time from symptom onset to US was 19 days (interquartile range (IQR) 10-26; shortest interval 12 hours). Median time to MRN was 25 days (IQR 13-33; shortest

interval 3 days). Median time to needle EMG was 26 days (IQR 13-31). Clinically affected nerves were: suprascapular (SN)=16; anterior interosseous (AIN)=14; long thoracic (LTN)=12; posterior interosseous (PIN)=11; axillary (AXN)=9; musculocutaneous (MCN)=4; phrenic (PH)=3; spinal accessory (SAN)=3; and the C5 nerve root=1.

Based on review of the original reports, HGCs were detected on either US or MRN in at least one clinically affected nerve in 29/39 (74%) patients. Of the 29 patients with HGCs detected in at least one clinically affected nerve, HGCs were identified in initial reports in 4 patients within 1 week, in 8 patients within 2 weeks, in 5 patients within 3 weeks, and in 12 patients within 4 weeks after symptom onset. The earliest HGC on US was found within 12 hours from symptom onset **(Fig. 1A, 1B)**, and on MRN at 3 days from symptom onset.

Overall, focal nerve enlargement (without a clearly identifiable associated HGC) of at least one clinically affected nerve was detected in 18/29 (62%) patients who underwent US and in 19/23 (82%) patients who underwent MRN (**Table 1**). HGCs **(Fig. 1, 2, 3)** were detected in at least one clinically affected nerve in 18/29 (62%) patients undergoing US, and in 16/23 (69%) patients undergoing MRN (**Table 1**). Nerve abnormalities (focal nerve enlargement with or without HGCs) were seen on ultrasound on MRN in 35/39 (90%) of patients within 1 month of symptom onset. Needle EMG was performed in 30 patients and revealed fibrillation potentials in affected muscles in 22/30 (73%) patients (**Table 1**).

On MRN, denervation edema pattern (**Fig. 2A, 2D; Fig. 3A**) of clinically affected muscles was observed in 21/23 (91%) patients (**Table 1**). The shortest time to observe an edema pattern was 8 days in the forearm in a patient with anterior interosseous nerve involvement. Denervation edema pattern on MRN was associated with the presence of HGCs on MRN ($p=0.025$, Cramer's $V=0.466$) or US ($p=0.045$, Cramer's $V=0.519$) and of fibrillation potentials on EMG performed at least 3 weeks after onset ($p=0.004$, Cramer's $V=0.664$).

Discussion

Nerve ultrasound and MRN showed nerve abnormalities (focal nerve enlargement, with or without HGCs) within 1 month from the onset of NA in 35/39 (90%) of the patients. In 8 patients,

HGCs were seen in the very early phase (within 2 weeks), and in one case at the emergency ward within 12 hours (**Fig. 1**). Overall, HGCs, an emerging diagnostic biomarker of NA (6-11), were detected in 74% of the patients.

A muscle denervation edema pattern was identified on MRN in 3 patients within the first 10 days from symptom onset **(Fig. 2A, 2D)**. Previous reports showed that, in animal models, T2-weighted MRI was sensitive to denervation as early as 24-48 hours after injury (16-19). We demonstrate that HGCs not only occur early in the disease course (in 4 patients by the first week), but also correlate with active muscle denervation, seen either by MRN (edema pattern) or needle EMG (fibrillation potentials) performed at least 3 weeks after disease onset. This finding is clinically relevant as EMG does not reliably show signs of muscle denervation until 2-3 weeks after axonal damage (4). Identifying imaging abnormalities in the acute phase of NA facilitates rapid diagnostic confirmation that would support early use of immunomodulatory therapy. Although no randomized clinical trials have been performed to validate use of immunomodulatory medications (20), their potential efficacy may be greatest soon after symptom onset (21-24).

Whether to use US or MRN to diagnose peripheral neuropathy depends on multiple factors, including cost, availability, and personal experience. While this study did not directly compare the efficacy of MRN versus US to detect nerve abnormalities, deeper coursing nerve segments, such as those of the axillary and long thoracic nerves, are typically more easily identified on MRN than US, assuming that motion artifacts do not degrade MR quality. However, the diagnostic performance of US for nerves located in the neck is overall high (25).

Study limitations include its retrospective nature, the limited number of patients, and the fact that EMG was not considered mandatory to confirm the diagnosis. This study cannot determine the earliest time when HGCs develop, but demonstrates that they do occur very early, at least within several hours to days from symptom onset. Another limitation was the variability in imaging protocols used among the different sites, particularly for MRN. Although there was relative uniformity with regards to spatial resolution obtained for 3-D pulse sequences, which are important to delineate small branch nerves, the signal-to-noise ratio likely varied given different surface coil setups and MR systems used. Additionally, images were interpreted by radiologists with differing experience with

MRN and this may have influenced the diagnostic sensitivity of HGC detection. For instance, at site 2, where MRN has been optimized specifically to detect HGCs in NA, constrictions were identified in 84% (11/13) of the patients studied, a higher percentage compared to other sites. Finally, this study was retrospective and not conceived to investigate prognosis, and therefore we did not systematically follow recovery in patients. The presence of HGCs of nerves detected in the operating room has been associated with poorer prognosis (2), but this likely reflects a selection bias, as surgical exploration has been most commonly performed in patients without signs of recovery (6, 26-34). We believe that dedicated studies are needed to determine whether the number and/or severity of HGCs are prognostic indicators, and if patients without spontaneous recovery can benefit from surgery (34).

In conclusion, early and reliable diagnosis of NA may be facilitated by MRN and US, abnormalities of which correlate with muscle denervation. We recommend the use of US and/or MRN as first line complementary tools within the first two weeks after symptom onset, with EMG performed subsequently.

Table 1. Summary of Imaging and EMG Findings

PATIENTS	**SITE 1 (N=9)**	**SITE 2 (N=13)**	**SITE 3 (N=14)**	**SITE 4 (N=2)**	**SITE 5 (N=1)**	**TOTAL (N=39)**
US	9	3	14	2	1	29
Focal nerve enlargement	7	2	6	2	1	18 (62%)
HGCs	4	2	11	0	1	18 (62%)
MRN	9	13	---	---	1	23
Focal nerve enlargement	5	13			1	19 (82%)
HGCs	5	11			0	16 (69%)
Muscle denervation	7	13			1	21 (91%)
EMG	9	10	9	1	1	30
Fibrillation potentials	6	9	6	1	0	22 (73%)

HGC = hourglass-like constriction; EMG = electromyography; MRN = magnetic resonance neurography; US= ultrasound.

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List of abbreviations

HGC - hourglass constriction

NA - neuralgic amyotrophy

MRN - magnetic resonance neurography

US - ultrasound

EMG - electromyography

NRS - numerical rating scale

IQR - interquartile range

SN - suprascapular nerve

AIN - anterior interosseous nerve

LTN - long thoracic nerve

PIN - posterior interosseous nerve

AXN – axillary nerve

MCN - musculocutaneous nerve

PH – phrenic nerve

SAN - spinal accessory nerve

SS - supraspinatus muscle

IS - infraspinatus muscle

Da – deltoid muscle, anterior head

Dm - deltoid muscle, middle head

Dp – deltoid muscle, posterior head

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Figure 1. Suprascapular and phrenic nerves on US.

A and B: US of patient #7 at 12 hours after symptom onset. **(A)** The suprascapular nerve (SN, white arrow) is enlarged (CSA 4.5 mm^2) in the lateral part of the neck, as the nerve courses behind the omohyoid muscle. The diagnostic cut-off proposed by Gruber et al.(12) for the nerve in this location is 4.2 mm^2. **(B)** The longitudinal view shows a partial HGC of the nerve (2.2 mm at point 1, 2.4 mm at point 2, 1.5 mm at point 3). Gentle palpation of the nerve at the constriction site (point 3) triggered severe neuropathic pain radiating towards the scapula.

(C) and (D): US of the same patient (#7) 10 days after sudden onset of orthopnea. Several days after initial symptom onset, the patient developed a bilateral phrenic nerve palsy **[video]**. **(C)** The left phrenic nerve (white arrow) is enlarged. **(D)** The longitudinal view of the left phrenic nerve better demonstrates the caliber changes (1.1 mm at point 1, 2.7 mm at point 2, 2.0 mm at point 3).

Omo=omohyoid muscle; SN=suprascapular nerve; CA= carotid artery;

Figure 2. **Suprascapular and axillary nerves on MRN.**

A and B (suprascapular nerve): Brachial plexus MRN of patient #26 at 9 days after onset. Coronal T2-weighted fat suppressed MR image **(A)** demonstrates early denervation edema pattern of the supraspinatus and infraspinatus muscles. Curved multiplanar reformatted T2-weighted fat suppressed image **(B)** shows three intrinsic constrictions (arrows) of the suprascapular nerve.

C and D (axillary nerve): Brachial plexus MRN of patient #27, 13 days after onset. Curved multiplanar reformatted 3-D fat suppressed image **(C)** of the left axillary nerve shows focal, intrinsic constriction of the anterior division (arrow) after the axillary nerve bifurcation. Note the normal appearance of the axillary nerve posterior division (bracket). Axial T2-weighted fat suppressed image **(D)** demonstrates selective early denervation edema pattern of the left deltoid anterior head, with sparing of the middle and posterior heads.

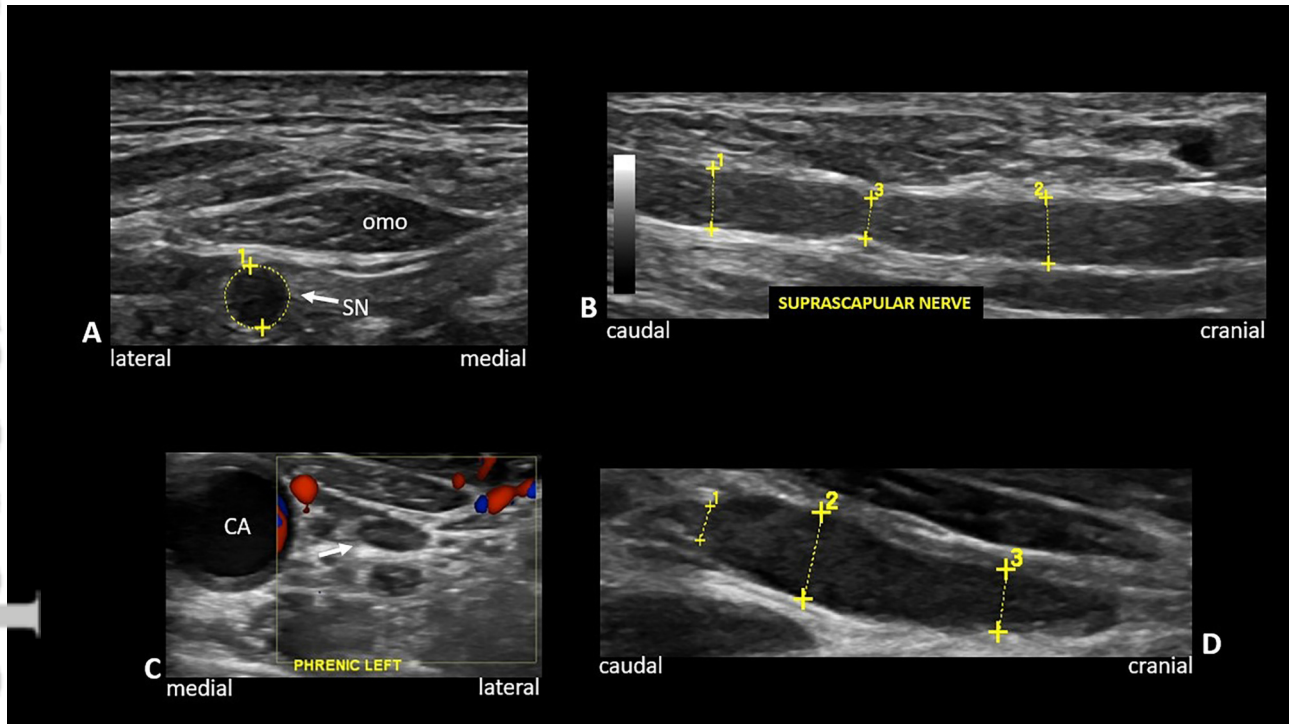
SS=supraspinatus muscle; IS=infraspinatus muscle; Da=deltoid, anterior head; Dm=deltoid, middle head; Dp=deltoid, posterior head.

Figure 3. Spinal accessory and posterior interosseous nerves.

A and B (spinal accessory nerve from patient #1): MRN **(A)** performed 30 days after onset shows multiple constrictions of the spinal accessory nerve in the neck (white arrows), confirmed 24 hours later by US scan **(B)** of the same nerve.

C (radial nerve and posterior interosseous nerve, PIN): US of patient #25, 28 days after onset. Longitudinal view of the posterior interosseous nerve (PIN) from the elbow (proximal, right corner) to the mid forearm (distal, left corner). The PIN is swollen and shows two subsequent constrictions (white arrows), one proximal to and one as the nerve enters the supinator tunnel. Dynamic cross-sectional scanning of the PIN from proximal to distal is shown in the **video**.

Trap=trapezius muscle; sup=supinator muscle; rad=radius; hum=humerus.



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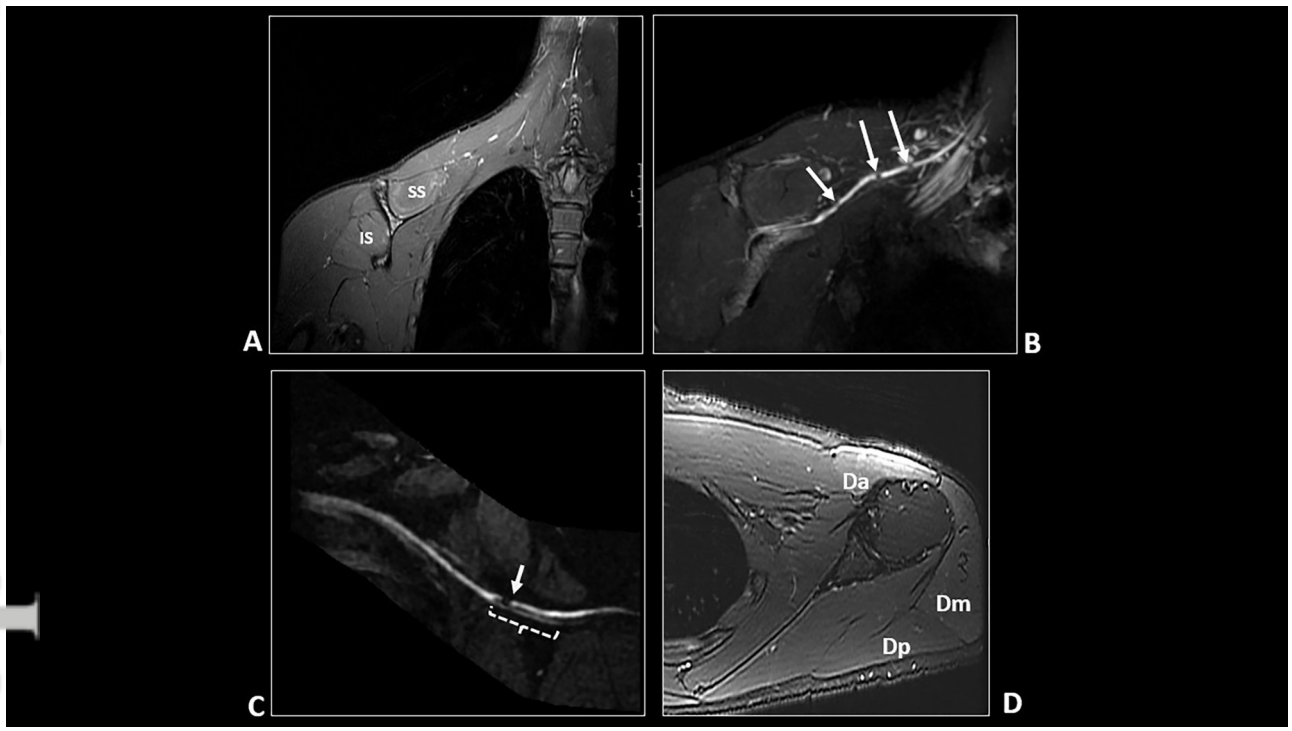

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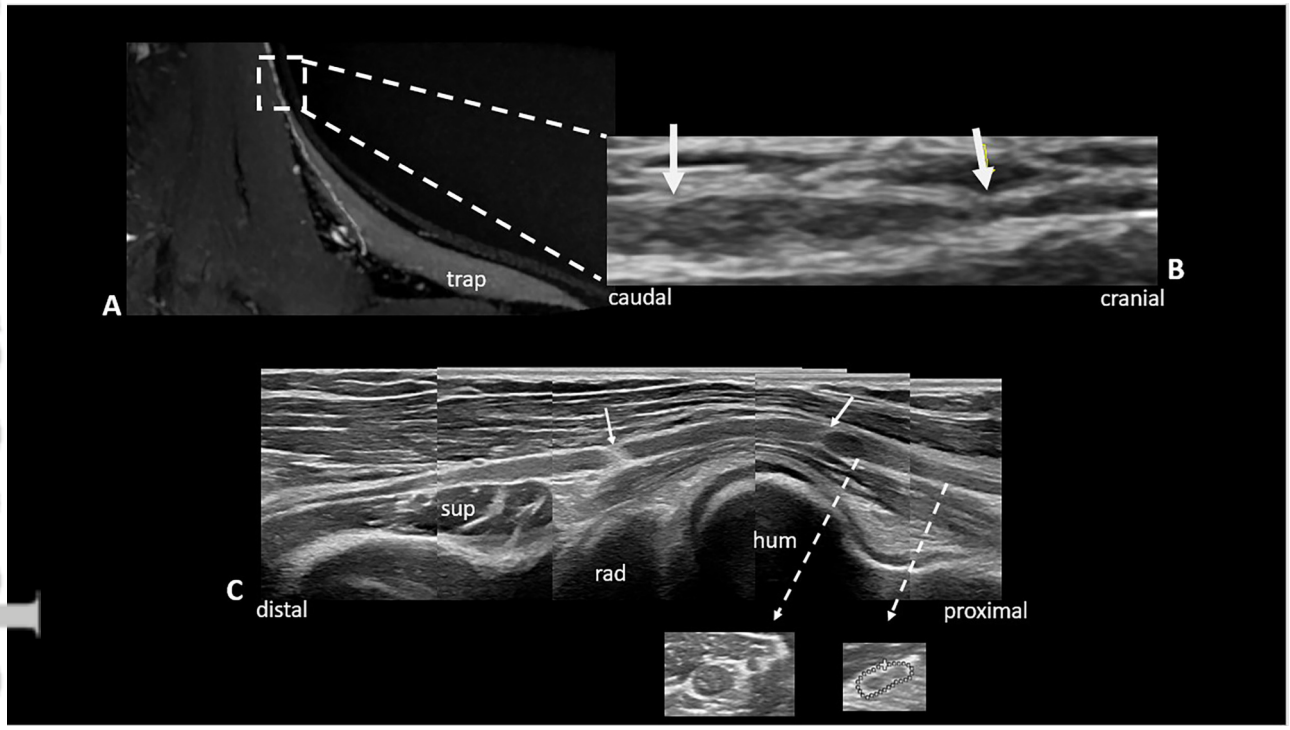

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