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Ultrasonographic diagnosis of neurogenic heterotopic ossification in patients with severe acquired brain injury admitted to a neurorehabilitation unit.

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Manuscripts

Review

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3 **Ultrasonographic diagnosis of neurogenic heterotopic ossification in patients with severe**
4 **acquired brain injury admitted to a neurorehabilitation unit.**
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10 **Short title:** Ultrasound of heterotopic ossification
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13
14 **Abstract**

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17 **Purpose:** to illustrate the ultrasonographic (US) and Power Doppler US (PDUS) features of
18 neurogenic heterotopic ossification (NHO) in consecutive patients with severe acquired brain
19 injury, to evaluate the role of bedside US in diagnosis of NHO, to study incidence of clinical
20 NHO in this neurorehabilitation setting.
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27 **Methods:** Ninety-two (92) consecutive patients with severe acquired brain injury underwent
28 clinical and laboratory screening to pose suspect of NHO. In 6/92 patients bedside US
29 confirmed the clinical suspect of NHO. US diagnosis of NHO was then confirmed by other
30 imaging methods.
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37 **Results:** the incidence of clinical NHO was 7% (one patient with multifocal involvement). In
38 NHO of hip US demonstrated the classical pattern of zone phenomenon, and PDUS
39 demonstrated vascular signal within mineralized NHO and in outer hypoechoic area. No
40 vascular signal was observed in the central hypoechoic core. In NHO of knee and elbow only
41 a heterogeneously hypoechoic mass or hyperechoic mineralized mass were respectively
42 evident, with vascular signals within the lesions. Spectral wave analysis (SWA) demonstrates
43 low resistance vessels in NHO.
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53 **Conclusions:** bedside US is a useful and cheap tool in diagnosis of NHO. PDUS adds data
54 about neoangiogenetic activity of NHO.
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Introduction

Heterotopic ossification is the development of mature lamellar bone (by endochondral ossification) in periarticular soft tissues. This process is frequently associated with neurological disorders (spinal cord injury, hemiplegia due to stroke or traumatic brain injury, encephalitis) and it is defined neurogenic heterotopic ossification (NHO) ¹⁻³. However heterotopic ossification has been described with the same features in burns, trauma and joint arthroplasty ^{4,5}. Other terms are used to describe this phenomenon, such as paraosteopathies, myositis ossificans, heterotopic bone formation.

NHO can lead to severe limitation of the range of motion (ROM) and even complete ankylosis of peripheral joints ^{1,4}. As not-surgical treatment of NHO, even if controversial, should prevent endochondral ossification, diagnosis should be made before calcification of involved soft tissue ^{1,3,6,7}.

Though NHO could begin within few weeks after neurological injury, diagnosis is often delayed. Early clinical signs include decreased ROM, local pain and firm swelling, erythema and warmth. The clinical features of the early inflammatory phase mimic tumour, infection, deep venous thrombosis (DVT) and acute arthritis ^{1-4,8}. Laboratory findings may not be helpful in early diagnosis. In particular, a rise of serum alkaline phosphatase (AP), hydroxyprolinuria and erythrocyte sedimentation rate (ESR) is reported but it is very aspecific ^{1,3}.

Diagnostic radiographic or computed tomography (CT) findings may not appear for one month. Three-phase ^{99m}Tc bone scanning is a sensitive diagnostic tool for NHO, but its low specificity leads to difficulty in discriminating NHO from other inflammatory, traumatic or degenerative process of the skeleton. Also magnetic resonance imaging (MRI) has proved limited role in the early diagnosis of NHO because of its moderate specificity, high cost and necessity of contrast ^{1,9-11}.

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3 Gray-scale ultrasonography (US) is proved to be a sensitive imaging method in soft tissues
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5 lesions, calcifications and bone-related diseases^{12,13}. Moreover US has high specificity in
6
7 early diagnosis and follow-up of NHO, allows differentiation from muscle tears, haematoma,
8
9 infection, DVT and soft tissues tumour^{1,2,8,14-20}. US has the advantage of bedside application,
10
11 it is easy to perform, repeatable and inexpensive and requires no radiation. Doppler US has
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13 proved to be a reliable tool for semiquantitative assessment of the vascularity of the soft tissue
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¹². As demonstrated in a recent work Doppler US can detect hypervascularization inside and outside HO².

The aim of this study was to evaluate the role of bedside US in early diagnosis of NHO, to define the incidence of clinical NHO in this setting and to describe US and power Doppler US (PDUS) features of NHO.

Materials and Methods

Ninety-two consecutive inpatients transferred from Neurosurgery or other intensive care unit and referred to our neurological rehabilitation unit from September 2007 to October 2008 with severe acquired brain injury and clinical suspect of NHO underwent bedside US and PDUS examination. Clinical suspect of NHO derived from reduction of ROM of a joint, with or without local swelling, rising of AP and/or ESR and C-reactive protein (CRP).

Location, etiology and severity of the neurological lesion and risk factors for NHO (like DVT, spasticity, pressure sores, and infection) was recorded.

In all US positive cases for NHO, diagnosis was confirmed with radiography or CT, and three-phase scintigraphy.

In all NHO patients a therapy was initiated with: indometacin 50 mg bid for 15 days, disodium etidronate 300 mg bid per 6 months, 6-methylprednisolone 8 mg until normalization of ESR, and cautious joint mobilization.

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3 Bedside US examination were carried out using a SonoSite (Bothell, USA) Titan with a 5-10
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5 MHz linear transducer (frequency variations depending on site and depth of the lesion). Each
6
7 US examination was carried out with both longitudinal and transverse scans by the same
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9 operator, with the same machine setting. The technical parameters of power Doppler US
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11 were: lowest pulse repetition frequency (PFR) avoiding flash artifact, highest gain level
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13 without background noise and low filter. The intensity of vascularity at PDUS analysis of the
14
15 NHO lesions was rated on a semi-quantitative 4-point scale as follows: grade 0= no flow
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17 signal; grade 1= 1-2 flow signals (only as coloured spot); grade 2= 2-5 flow signals (both
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19 coloured spot and/or definite vessels without secondary branching); grade 3= >5 flow signals
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21 or vascular tree or diffuse blush with vessel boundaries not distinguishable²¹. A spectral
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23 Doppler tracing was obtained to confirm that each color signal represented true arterial flow
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25 and spectral wave analysis (SWA) was performed in order to define two indices: Pulsatility
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27 Index (PI) and Resistance index (RI). In particular the latter, defined as (maximum systolic
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29 velocity- end diastolic velocity)/ maximum velocity, was recorded as it correlates with
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31 increasing peripheral resistance.
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41 Results

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43 Six patients over 92 (7 %) with severe acquired brain injury had clinical and US evidence of
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45 NHO. The demographic and clinical characteristics of patients with NHO are shown in Table
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47 1 (Tab. 1). In one patient NHO was multifocal involving both hips and left elbow.
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49 US examination showed a focal disorganization of muscle in the involved area, with loss of
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51 longitudinal muscular striation and presence of a heterogeneously hypoechoic mass with the
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53 characteristics of the classical “zone phenomenon” previously described in literature, in
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55 particular in the hip^{15,18}.
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3 The zone phenomenon was always evident in NHO of the hip (2 patients) and appeared as a
4 inner hypoechoic core surrounded by a ring of hyperechoic mineralized islands, with an outer
5 hypoechoic zone adjacent to normal muscle (Fig.1). PDUS demonstrates vascular signal
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7
8 hypoechoic zone adjacent to normal muscle (Fig.1). PDUS demonstrates vascular signal
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10 (grade 2 in both patients) within mineralizing area and in outer hypoechoic area and SWA of
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12 vessels within NHO always showed $RI < 1$. No vascular signal was observed in the central
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14 hypoechoic core. No synovitic intracapsular vascular signal was observed.
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16
17 In NHO of knee (4 patients) the classical zone phenomenon was less evident. A
18
19 heterogeneously hypoechoic mass was evident within insertional area of vastus medialis
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21 muscle and proximal enthesis of medial collateral ligament (Fig. 2a). In a case mild
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23 hyperechoic deposits were observed also within distal enthesis of medial collateral ligament.
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25 PDUS showed intense vascularisation inside the lesion (grade 3 in 3 patients, grade 2 in one)
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27 (Fig. 2b) and SWA of vessels within NHO always showed $RI < 1$ (Fig. 2c).
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31 In NHO of elbow (1 patient) only mild hyperechoic deposits in olecranic recess of the joint
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33 were observed and the zone phenomenon was not evident (Fig.3). PDUS demonstrates
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35 vascular signal within mineralizing area (grade 1). In all the site SWA of vessels within NHO
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37 always showed $RI < 1$.
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41 Only hip and elbow NHO were associated with significant joint effusion (Fig. 1) but no
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43 synovitic intracapsular vascular signal was observed. In knee NHO joint effusion was always
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45 small.
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48 The dynamic examination always showed a low compressibility of the hypoechoic areas
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50 during the application of a pressure with the probe.
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53 In our case study only a NHO of the hip evolved within one year in complete ankylosis, while
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55 in the other cases an acceptable ROM was maintained.
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58 59 60 **Discussions**

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3 NHO is a complication of brain and spinal injury and its prevalence varies from 16% to 53%
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5 in traumatic patients. Scanty data are available about incidence of NHO in not-traumatic
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7 neurological disorders^{5,22}. A recent work showed 4% of incidence in a neurorehabilitation
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9 unit³. Our data shows comparable results with a similar diagnostic algorithm.

10
11
12 According with literature hip, knee and elbow were the most frequently affected joints^{1,3,22}.
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14 Severe and rapid limitation of range of motion was the most common sign of NHO in our
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16 patients, while pain was not always detectable because of sensory deficits and/or concomitant
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18 severe aphasia. In knee and elbow a firm swelling was observed in the site on NHO within
19
20 few days from diagnosis. In NHO of hip palpable mass was not appreciable because of the
21
22 depth of joint.
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26 In 4 patients AP, ESR and CRP were elevated in early stages of NHO and decreased after
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28 beginning of therapy. In 2 cases with small NHO of knee only ESR and CRP was elevated.
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30 Our data confirm the usefulness but incomplete sensitivity of these inexpensive investigation
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32 in diagnosis of NHO. In fact, as confirmed in previous works, if NHO formation is small,
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34 ALP levels may remain unchanged²². Moreover, low specificity of these laboratory findings
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36 should be considered, in particular in critical ill patients (concomitant infection, tracheostomy,
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38 recent neurosurgery, pressure sores).
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43 In all the cases plegia and muscular hypertonia or spasticity was present in the affected limbs.
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45 This risk factor is often reported in literature as cause of muscle hypoxia and increased risk of
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47 muscle tears from mobilization^{1-5,22,23}.
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51 Our gray-scale US data support the highly specific pattern of “zone phenomenon” in NHO of
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53 the hip, while in other localizations differential diagnosis with arthritis, tumour and abscess
54
55 could be more difficult.
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58 In hips NHO were localized in a plane deep to the rectus femoris and superior to the coxo-
59
60 femoral capsule, as recognized in previous MRI works¹¹. Iliopsoas muscle or iliopsoas bursa

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3 seem the first involved sites. However in coxo-femoral joint an effusion was always observed.

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5 This effusion could be secondary to the adjacent intense inflammatory process.

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7
8 In knees NHO involved vastus medialis fibers in its enthesal area and proximal
9
10 fibrocartilaginous enthesis of medial collateral ligament. In our cases knee effusion was
11
12 minimal, whereas a previous work reported joint effusion with synovial hypertrophy ¹⁴.

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15 In elbow NHO was evident in the olecranic recess, probably in medial head of triceps muscle
16
17 or in olecranic bursa.

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20 In our experience the low compressibility of lesions underneath the probe is a useful sign to
21
22 diagnose NHO.

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24
25 In our knowledge this is the first work in which PDUS was applied to the study of early NHO.

26
27 Our data confirm that hypervascularization of mineralizing areas is a relevant diagnostic
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29 aspect of NHO lesions, as previously observed with angiographic studies ^{1,6}. Moreover the
30
31 flow pattern indicate the presence of low resistance vessel as reported in inflammatory
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33 neoangiogenesis. The inner core of early NHO appears as a relatively avascular area. Previous
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35 observations with MRI confirmed the strong ring enhancement of early HO after
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37 administration of Gadolinium-based contrast ^{10,11}.

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41 No flow signal was observed in synovial layer within adjacent joints. This can strengthen the
42
43 hypothesis of a reactive and secondary joint effusion ²⁴.

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46 Medical management of NHO is controversial and supported with limited evidence ^{22,25}. In
47
48 our case study only a NHO of the hip evolved within one year in complete ankylosis. In our
49
50 multifocal case therapy has been relatively effective in elbow but not in hips. Previous works
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52 reported different outcome after standardized therapy in various involved sites of the same
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54 patient ⁶. NHO in knee seems to have better evolution with respect to other sites.

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57 This study has some limitations. The incidence of NHO in brain injured patients could be
58
59 underestimated because we studied by imaging only sites with clinical signs or symptoms, so
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3 subclinical NHO could be missed. Moreover, the time period of follow-up has been limited to
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5 about one year. However this diagnostic protocol has been used in previous studies and avoids
6
7 the high costs of a screening imaging protocol³.
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10 Another limitation is the heterogeneity of the diagnosis of case series, but it is related to the
11
12 neurorehabilitative setting of the study.
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15 In conclusions, NHO is a frequent cause of reduction of ROM and secondary disability
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17 following severe acquired brain injury. As early detection of NHO and prompt starting of
18
19 therapy are essential to contain related disability, bedside US seems a useful first-step
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21 diagnostic tool when clinical and/or laboratory findings induce suspicion of NHO.
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27 **References**

- 28
29 1. van Kuijk AA, Geurts ACH, van Kuppevelt HJM .Neurogenic heterotopic ossification in
30
31 spinal cord injury. *Spinal Cord* 2002;40:313-26.
32
33
- 34 2. Youssefian T, Sapena R, Carlier R, et al. Nodular osteochondrogenic activity in soft
35
36 tissue surrounding osteoma in neurogenic para osteo-arthropathy: morphological and
37
38 immunohistochemical study. *BMC Musculoskeletal Disorders* 2004;5:46.
39
40
- 41 3. Taly AB, Nair KP, Jayakumar PN, et al. Neurogenic heterotopic ossification: a diagnostic
42
43 and therapeutic challenge in neurorehabilitation. *Neurol India* 2001;49:37-40.
44
45
- 46 4. McCarthy EF, Sundaram M. Heterotopic ossification: a review. *Skeletal Radiol*
47
48 2005;34:609-19.
49
50
- 51 5. Lane JE, Dean RJ, Foulkes GD, et al. Idiopathic heterotopic ossification in the intensive
52
53 care setting. *Postgrad Med J* 2002;78:494-495.
54
55
- 56 6. Jamil F, Subbarao JV, Banovac K, et al. Management of immature heterotopic
57
58 ossification (HO) of the hip. *Spinal Cord* 2002;40:388-95.
59
60

- 1
2
3 7. Banovac K, Sherman AI, Estores IM, et al. Prevention and treatment of heterotopic
4
5 ossification after spinal cord injury. *J Spinal Cord Med* 2004;27:376-82.
6
7
- 8 8. Scola FH, Parziale JR. Ultrasonographic diagnosis of heterotopic ossification mimicking
9
10 deep vein thrombosis. *J Clin Ultrasound* 1991;19:55-57.
11
- 12 9. May DA, Disler DG, Joner EA, et al. Abnormal signal intensity in skeletal muscle at MR
13
14 imaging: patterns, pearls and pitfalls. *Radiographics* 2000;20:S295-315.
15
- 16 10. Wick L, Berger M, Knecht H, et al. Magnetic resonance signal alterations in the acute
17
18 onset of heterotopic ossification in patients with spinal cord injury. *Eur Radiol*
19
20 2005;15:1867-75.
21
22
- 23 11. Ledermann HP, Schweitzer ME, Morrison WB. Pelvic heterotopic ossification: MR
24
25 imaging characteristics. *Radiology* 2002;222:189-95.
26
27
- 28 12. Kiris A, Ozgocmen S, Kocakoc E, et al. Power Doppler assessment of overall disease
29
30 activity in patients with rheumatoid arthritis. *J Clin Ultrasound* 2006;34:5-11.
31
32
- 33 13. Cho KH, Lee YH, Lee SM, et al. Sonography of bone and bone-related diseases of the
34
35 extremities. *J Clin Ultrasound* 2004;32:511-21.
36
37
- 38 14. Peck RJ, Metreweli C. Early myositis ossificans: a new echographic sign. *Clin Radiol*
39
40 1988;39:586-8.
41
42
- 43 15. Thomas EA, Cassar-Pullicino VN, McCall IW. The role of ultrasound in the early
44
45 diagnosis and management of heterotopic bone formation. *Clin Radiol* 1991;43:190-6.
46
47
- 48 16. Alouini-Mekki R, el Mhabrech H, Hasni I, et al. La myosite ossifiante circonscrite:
49
50 apport de l'imagerie. *J Radiol* 2007;88:663-8.
51
52
- 53 17. Bodley R, Jamous A, Short D. Ultrasound in the early diagnosis of heterotopic
54
55 ossification in patients with spinal injuries. *Paraplegia* 1993;31:500-6.
56
57
- 58 18. Cassar-Pullicino VN, McClelland M, Badwan DA, et al. Sonographic diagnosis of
59
60 heterotopic bone formation in spinal cord injury patients. *Paraplegia* 1993;31:40-50.

- 1
2
3 19. Kramer FL, Kurtz AB, Rubin C, et al. Ultrasound appearance of myositis ossificans.
4
5 Skeletal Radiol 1979;4:19-20.
6
- 7
8 20. Kirkpatrick JS, Koman LA, Rovere GD. The role of ultrasound in the early diagnosis of
9
10 myositis ossificans. A case report. Am J Sports Med 1987;15:179-81.
11
- 12
13 21. Falsetti P, Frediani B, Acciai C, et al. Ultrasonography and magnetic resonance imaging
14
15 of heel fat pad inflammatory-oedematous lesions in rheumatoid arthritis. Scand J
16
17 Rheumatol 2006;35:454-8.
18
- 19
20 22. Vanden Bossche L, Vanderstraeten G. Heterotopic ossification: a review. J Rehabil Med
21
22 2005;37:129-136.
23
- 24
25 23. Tsur A, Sazbon L, Lotem M. Relationship between muscular tone, movement and
26
27 periarticular new bone formation in postcoma-unaware (PC-U) patients. Brain Inj
28
29 1996;10:259-62.
30
- 31
32 24. Kun EW, Barr WG. Heterotopic ossification presenting as acute arthritis. J Rheumatol
33
34 1992;19:994-6.
35
- 36
37 25. Cullen N, Bayley M, Bayona N, et al. Management of heterotopic ossification and venous
38
39 thromboembolism following acquired brain injury. Brain Inj 2007;21:215-30.
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	Sex, Age (years)	Neurologic injury (and clinical state)	site of NHO	NHO diagnosis (weeks from neurologic injury)	laboratory findings at NHO onset	PDUS grading	outcome after 1 year
1	F, 56	rupture of left middle cerebral artery arteriovenous malformation, neurosurgery for subdural hematoma (right hemiplegia and left hemiparesis with hypertonus, severe aphasia, PEG, no DVT or pressure sores)	right hip, left hip, left elbow	14 w	ESR 75 mm/h CRP 1.49 mg/dL AP 715 mg/dL bony IE 50.8 mcg/L	2 2 1	mild reduction of extension of left elbow, complete ankylosis of hips
2	M, 41	post-hypoxic encephalopathy (vegetative state, bilateral hemiplegia with hypertonus, tracheostomy, PEG, bilateral DVT, no pressure sores)	left knee	12 w	ESR 69 mm/h CRP 1.1 mg/dL AP 442 mg/dL bony IE 9.2 mcg/L	3	acceptable ROM
3	F, 68	head injury (left hemiplegia with hypertonus, no DVT or pressure sores)	left knee	18 w	ESR 28 mm/h CRP 2.79 mg/dL AP 280 mg/dL	3	acceptable ROM
4	F, 72	right intracerebral hemispheric hemorrhage, decompressive craniectomy for high intracranial pressure (left hemiplegia with hypertonus, tracheostomy, PEG, no DVT or pressure sores)	left hip	15 w	ESR 49 mm/h CRP 4.75 mg/dL AP 401 mg/dL	2	mild reduction of ROM
5	M, 40	head injury, decompressive craniectomy for high intracranial pressure (bilateral hemiparesis, tracheostomy, no DVT or pressure sores)	right knee	11 w	ESR 26 mm/h CRP 0.68 mg/dL AP 121 mg/dL	3	acceptable ROM
6	F, 76	subarachnoid hemorrhage for ruptured aneurysm of anterior communicating artery, endovascular selective embolization (bilateral hemiplegia with hypertonus, severe aphasia, tracheostomy, PEG, no DVT or pressure sores)	right knee	13 w	ESR 73 mm/h CRP 8.75 mg/dL AP 103 mg/dL	2	mild reduction of extension of right knee

Table 1**Demographic and clinical characteristics of NHO patients.**

ESR, erythrocyte sedimentation rate (normal values 2-25 mm/hour). CRP, C-reactive protein (normal values <0.5 mg/dL). AP, alkaline phosphatase (normal values 40-129 mg/dL for female, 35-104 mg/dL for male). bony IE, iso-enzyme of AP (normal values 3.8-22.4 microg/L). ROM, range of motion. DVT deep venous thrombosis. PEG, percutaneous endoscopic gastrostomy.

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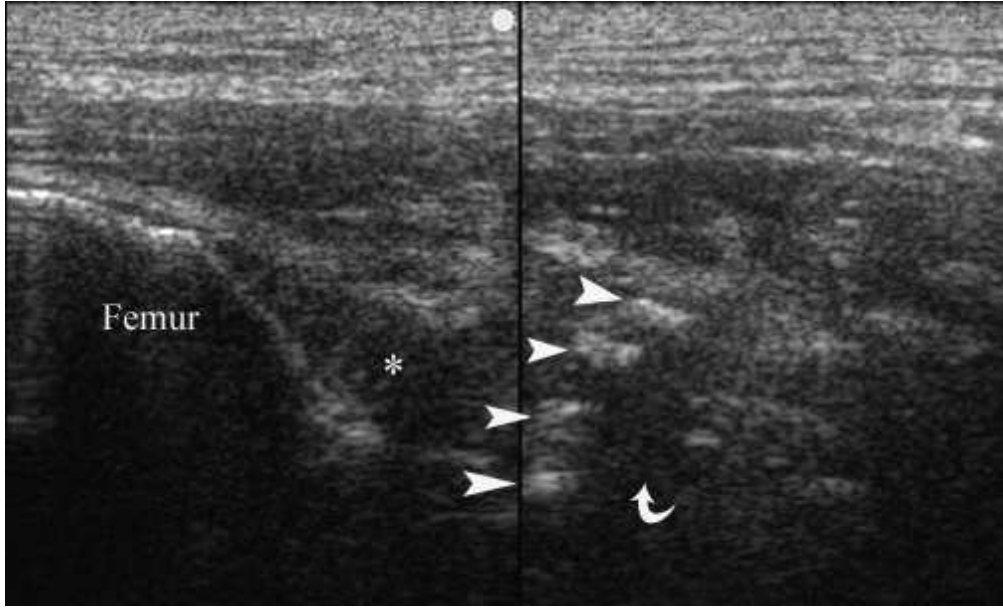


Figure 1. Sagittal longitudinal US scan of left hip (patient 1). The classical aspect of the “zone phenomenon” of HO was evident in iliopsoas muscle, with a inner hypoechoic core (curved arrow) surrounded by a ring of hyperechoic mineralized islands (arrowhead), and an outer hypoechoic zone adjacent to normal muscle. Hypoechoic effusion (*) is visible within coxo-femoral joint capsule.

170x102mm (75 x 75 DPI)

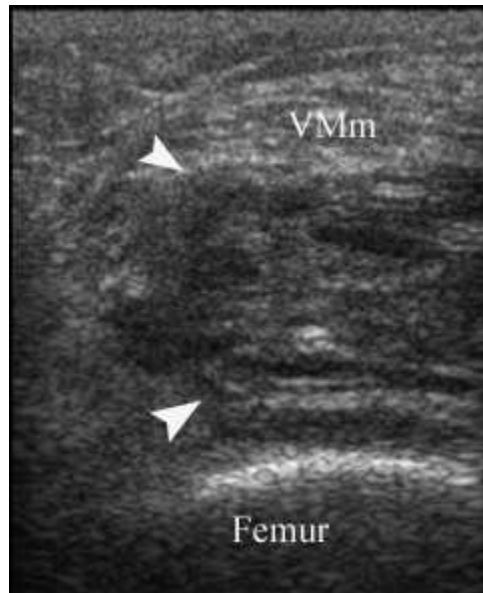


Figure 2a. Coronal transverse US scan of left knee over vastus medialis muscle (patient 2). A heterogeneously hypoechoic mass (between arrowheads) is evident within vastus medialis muscle (VMM). The cortical profile of femur appears regular.
81x99mm (75 x 75 DPI)

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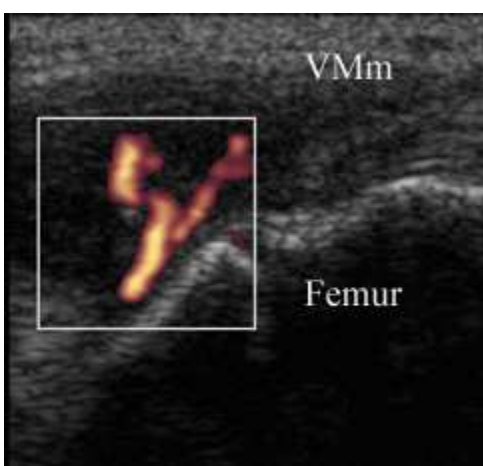
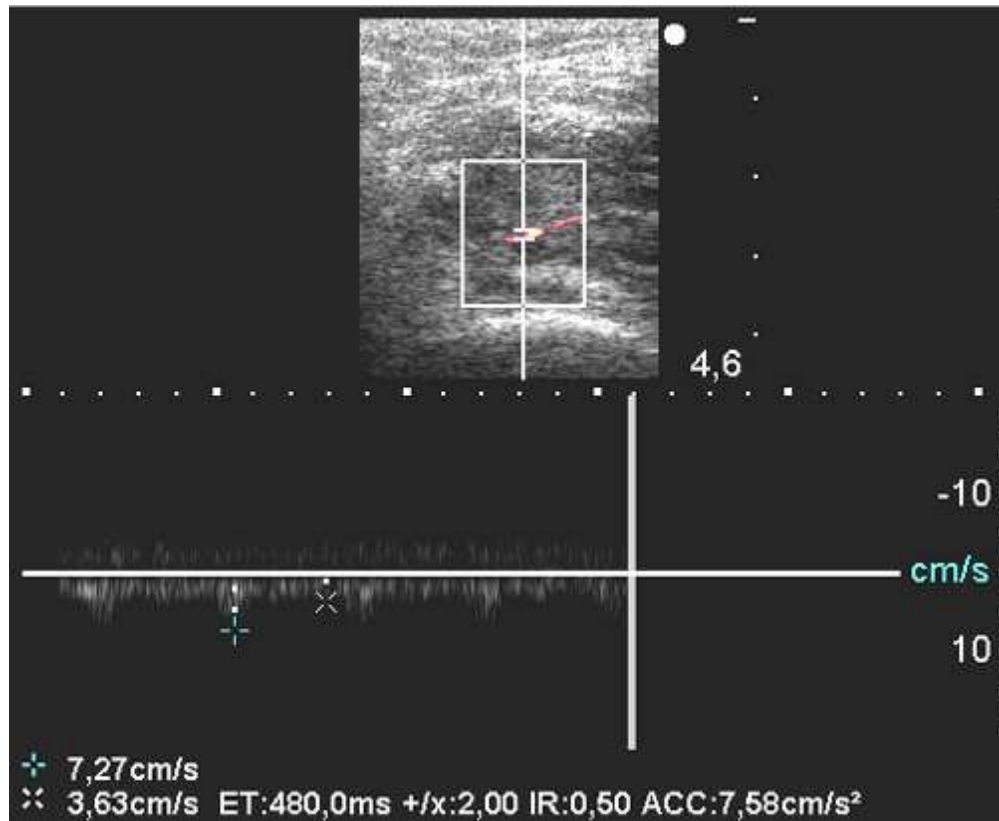


Figure 2b. Coronal transverse US scan of left knee over vastus medialis muscle (VMm) (patient 2). PDUS demonstrates a vascular tree within the hypoechoic mass of HO (grade 3). 81x77mm (75 x 75 DPI)

Peer Review



36 Figure 2c. Coronal transverse US scan of left knee over vastus medialis muscle (patient 2). PDUS
37 and SWA demonstrates low resistance flow in HO hypoechoic mass, with RI 0.50.
38 170x138mm (75 x 75 DPI)

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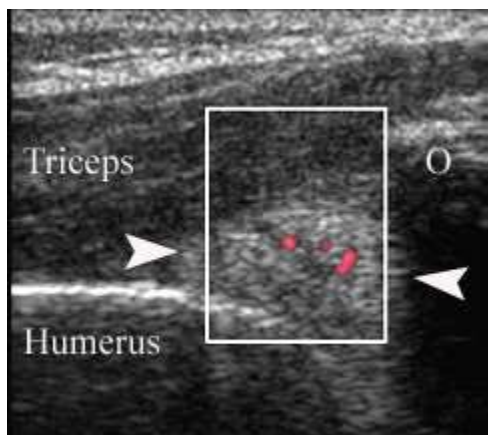


Figure 3. Coronal posterior longitudinal US scan of left elbow (patient 1). A hyperechoic mineralized mass is visible in medial side of olecranon recess (between arrowheads). PDUS demonstrates vascular signal within the mass (grade 1). O = olecranon.
81x72mm (75 x 75 DPI)

Peer Review