CME LIVE OPEN ACCESS

CME Journal of Clinical Case Report

Scientific Review Article ISSN: 3067- 7998

C.R.P.S. Type 1 And Type 2 Pharmacological, Rehabilitative, and Psychological Therapy in Pregnancy-Related Femoral Head Algodystrophy

Giorgio Mandala[1*] and Sofia Mandala[2]

[1]*U.O.C. Neurorehabilitation, Villa delle Ginestre Hospital. Provincial Health Authority. Palermo. Italy.*

Corresponding Author: Mandala, U.O.C. Neurorehabilitation, Villa delle Ginestre Hospital. Provincial Health Authority. Palermo. Italy.

[2]*Healthcare and Rehabilitation Center ARESS Fabiola Termini Imerese. Palermo. Italy.*

Received: 2025 Apr 14 **Accepted:** 2025 May 02 **Published:** 2025 May 15

Abstract

After a discussion of complex pain regional syndrome type 1 and type 2, the authors explain the particular characteristics of cprs type 1 in its location at the head of the femoral during pregnancy. In addition to diagnostic aspects and treatment, relevance is given to psychological support as an integral part of treatment. At the end, the most recent personal case report is explained, reported in comparison with a previous case report by the same authors.

1. Introduction

Femoral head algodystrophy, also known as Complex Regional Pain Syndrome Type I (CRPS-I), is a condition characterized by chronic pain, vasomotor and trophic alterations localized to the hip. It is distinguished from CRPS-II by the absence of documented nerve lesions. This pathology, often associated with minor trauma or immobilization, presents a variable course and a significant impact on the quality of life.

2. Epidemiology

• Incidence: 0.8-2.5 cases/100,000 inhabitants/year, with a peak between 45-55 years of age.
• Gender distribution: Prevalent in males (60%), except during the 3rd trimester of pregnancy, where it affects women more frequently.
• Risk factors: Joint trauma, surgical interventions, prolonged immobilization, pregnancy.

3. Etiopathogenesis

The pathogenetic mechanism involves an aberrant inflammatory response and microvascular alterations
• Neurogenic dysfunction: Hyperactivity of the sympathetic system and release of neuropeptides (substance P) that induce vasodilation and edema.
• Transient ischemia: Reduced local blood flow leads to hypoxia of the spongy bone, with accumulation of acidic metabolites and activation of osteoclasts.
• Role of mesenchymal stem cells (MSCs): Alterations in the viability of MSCs in the femoral head may compromise regenerative processes, promoting bone edema.

4 Clinical Presentation

4.1 Main Symptoms

• Intense pain at rest and under load, often disproportionate to the initial trauma.
• Sensory alterations: Allodynia (70% of cases) and hyperalgesia.
• Vasomotor signs: Edema, erythema, thermal variations (initial phase: warm skin; advanced phase: cold and cyanotic skin).
• Motor dysfunction: Joint limitation, muscle atrophy, tremor.

4.2 Evolutionary Phases

Acute phase (0-3 months): Bone edema, acute pain, hyperemia.
Dystrophic phase (3-9 months): Localized osteoporosis, abnormal sweating, stiffness.
Chronic phase (>12 months): Risk of evolution into osteonecrosis in 15-20% of cases.

4.3 Diagnosis

• Clinical criteria (adapted from the Budapest criteria)
• Persistent pain + symptoms in ≥3 categories:
• Sensory (allodynia/hyperalgesia)
• Vasomotor (edema, thermal alterations)
• Sudomotor/trophic (cutaneous, nail alterations)
• Motor (weakness, stiffness).

4.4 Imaging

• Magnetic Resonance Imaging (MRI): Gold standard. Shows diffuse bone edema in the femoral head and neck (T1 hypointensity, T2 hyperintensity).
• Bone scintigraphy: Increased uptake in the early phase, useful for differential diagnosis with osteonecrosis.
• Radiography: Transient osteoporosis in advanced stages, but poorly sensitive in early stages.

4.5 Differential Diagnosis

• Avascular osteonecrosis: Absence of diffuse edema on MRI and presence of signs such as the crescent sign.
• Hip osteoarthritis: Cartilage damage associated with bone sclerosis.

4.6 Therapy Multidisciplinary Approach

The multidisciplinary team must take care of the person in his or her entirety, without neglecting the problems of the internal organs and the musculoskeletal system. Psychological support and therapy are fundamental in CPRS, considering the intensity of the pain and the prolonged duration of the disease.

4.6.1 Pharmacological Therapy

• Bisphosphonates (e.g., neridronate): Reduction of bone resorption and symptomatic remission in 60-70% of cases if administered early.
• Non-steroidal anti-inflammatory drugs (NSAIDs): Symptomatic pain management.
• Corticosteroids: Used in refractory cases to reduce inflammation.

4.6.2 Regenerative Therapies

• MSC infiltrations: Percutaneous injection of stem cells from the iliac crest, shown to be effective in reducing edema and accelerating healing.

4.6.3 Rehabilitation

Joint unloading: Use of crutches for 4-8 weeks to reduce pressure on the femoral head.

Physiotherapy: Passive mobilization exercises, desensitization, and graded motor imagery. Even magnetotherapy with pulsed electromagnetic fields at very low frequency (4 - 8 Hz) has proven effective if continued for at least 6-8 continuous hours, usually during the night.Passive mobilization aims to maintain the articulation of the hip and lumbar spine. It is necessary to avoid causing pain and for this reason the movements must be slow and delicate on all three planes of space, associating delicate circumductions of the hip.In order to maintain muscle tone and strength, isometric contraction exercises of the quadriceps and the hip flexor and abductor muscles must be performed

4.6.4 Psychological Support

Counseling and psychotherapy are essential for the acceptance of the pathological situation and for the favorable influences on the healing process. The cognitive behavioral approach seems to be the most effective also for the speed of its effectiveness

4.6.5 Monitoring

• Follow-up with MRI: At 3 and 6 months to assess the resolution of edema.

4.7 Prognosis

• Benign course: In 80% of cases, spontaneous resolution within 6-12 months with timely therapy.
• Complications: Evolution into osteonecrosis (15-20% of cases) or secondary osteoarthritis, especially in the presence of vascular risk factors.

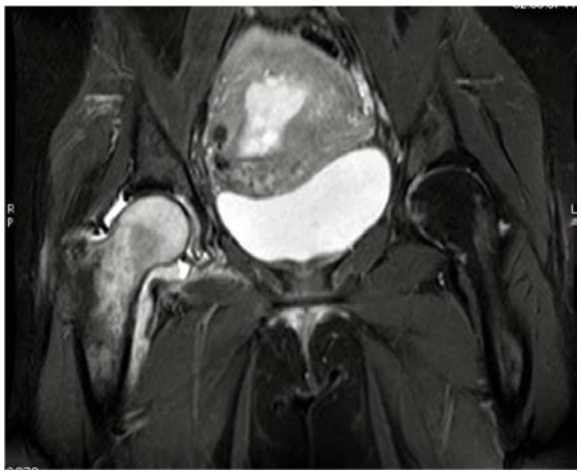

Figure 1: MRI Image of Bone Edema of the Left Femoral Head

5. Complex Regional Pain Syndrome Type Ii (CRPS-Ii)

5.1 Introduction

Complex Regional Pain Syndrome Type II (CRPS-II), also known as causalgia, is a rare and debilitating condition that develops after a peripheral nerve injury. It is distinguished from CRPS-I by the documented presence of nerve damage, while CRPS-I occurs without evidence of a specific nerve lesion.

5.2 Epidemiology

The annual incidence of CRPS-II ranges from 5.5 to 26.2 cases per 100,000 people, with a higher prevalence in females (60-80%). The average age of onset is around 45 years.

5.3 Etiopathogenesis

CRPS-II typically manifests after trauma or surgery involving a peripheral nerve. Nerve damage induces an exaggerated inflammatory response, with the release of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α) and neuropeptides (such as substance P), which contribute to the clinical symptoms.29 Furthermore, upregulation of catecholamine receptors and alterations in norepinephrine levels are observed in the affected limb.

5.4 Clinical Presentation

CRPS-II presents with intense, persistent pain that is disproportionate to the triggering event, associated wit

• ensory alterations (allodynia, hyperalgesia)

• Vasomotor changes (edema, erythema, temperature variations)

• orphic disturbances (cutaneous alterations, muscle and bone atrophy)

• Motor dysfunction (joint stiffness, weakness).

5.5 The Syndrome Typically Evolves in Three Phases

• Acute/inflammatory phase: pain, edema, hyperemia, warm and dry skin.

• Dystrophic phase: persistent pain, excessive sweating, cold skin, muscle atrophy.

• Atrophic phase: advanced atrophy, stiffness, loss of function.

5.6 Diagnosis

Diagnosis is primarily clinical and based on the Budapest criteria, which include persistent disproportionate pain and symptoms in at least three of the four categories: sensory, vasomotor, sudomotor/edema, and trophic.67 Instrumental examinations can be supportive, but are not specific.

5.7 Therapy

Treatment of CRPS-II is multidisciplinary and should be initiated early to improve prognosis

• Pharmacological therapy: analgesics, anti-inflammatory drugs, antidepressants, anticonvulsants.

• Physiotherapy: early mobilization, desensitization exercises, graded motor imagery.

Interventional therapies: nerve blocks, neuromodulation (spinal cord stimulation), sympathectomy in refractory cases.

• Psychological support: management of the emotional and behavioral impact of chronic pain.

Recent studies indicate that early diagnosis and integrated treatment can lead to significant recovery in up to 80% of patients within 18 months of symptom onset Femoral Head .

5.8 Algodystrophy in Pregnancy: Clinical Overview

5.8.1 Introduction

Femoral head algodystrophy (or CRPS-I) in pregnancy is a rare but disabling condition characterized by intense pain, bone edema, and vasomotor alterations localized to the hip. It predominantly manifests in the third trimester or postpartum, with a often benign course but potentially associated with osteoarticular complications.

5.8.2 Epidemiology

• Incidence: 0.8-2.5 cases/100,000 inhabitants/year, with a peak in the third trimester of pregnancy.

• Distribution: Prevalent in pregnant women (especially in the 3rd trimester), although non-gestational algodystrophy affects men more frequently (3:1).

5.9 Etiopathogenesis

The pathogenetic mechanism involve

• Vascular alterations: Impairment of blood flow to the femoral head due to uterine compression, with transient ischemia followed by reactive hyperemia and bone edema.

• Metabolic factors: Transfer of calcium to the fetus, hormonal imbalances (e.g., relaxin), and increased biomechanical load.

• Neurogenic inflammation: Activation of the sympathetic system and release of neuropeptides (substance P) that amplify pain and edema.

6. Clinical Presentation

6.1 Main Symptoms

• Acute pain in the hip and groin, worsened by weight-bearing and lateral decubitus.

• Joint limitation: Difficulty walking and with internal rotation movements of the hip.

• Objective signs: Edema, initial cutaneous hyperthermia (acute phase) followed by hypothermia (chronic phase).

6.2 Risk factors

• Pelvic vascular compression from the pregnant uterus.

• Coagulation disorders (thrombophilia) or ineffective fibrinolysis.

• Gestational hypertriglyceridemia.

6.3 Etiopathogenesis

6.3.1 The Pathogenetic Mechanism Involves

6.3.1.1 Evolution

• Acute phase (0-3 months): Bone edema on magnetic resonance imaging (MRI), intense pain.

• Resolution phase (6-12 months): Spontaneous regression in 80% of cases, with possible persistence of osteopenia.

7. Diagnosis

7.1 Diagnostic Tools

• Magnetic Resonance Imaging (MRI): Gold standard. Shows diffuse bone marrow edema (T1 hypointensity, T2 hyperintensity).

• Exclusion of differential diagnoses: Avascular osteonecrosis (absence of "crescent sign"), stress fractures, osteoarthritis.

8. Adapted Criteria

• Persistent pain + ≥3 of the following:

• Allodynia/hyperalgesia

• Edema or thermal alterations

• Abnormal sweating

• Functional limitation

9. Psychological Impact of Algodystrophy in Pregnancy

Algodystrophy, although not caused by psychological disorders, can generate anxiety, depression, and a sense of helplessness due to chronic pain and functional limitation.26 In pregnancy, these aspects are amplified as the woman also faces the normal emotional and physical challenges of gestation, in addition to concerns about her own health and that of the baby. Living with persistent pain and motor limitations can negatively affect mood, sleep quality, and the ability to perform daily activities, increasing the risk of secondary mental disorders.

10. Need for Specific Psychological Support

Psychological support does not aim to treat the organic cause of algodystrophy, but to reduce emotional distress and improve disease management. In pregnancy, a multidisciplinary approach that includes:

• Early psychological counseling to help the woman process emotions related to pain and prognostic uncertainty.
• Support in adapting to the disease, promoting effective coping strategies and reducing anxiety and depression.
• Management of stress related to pregnancy and illness, preventing psychological complications such as postpartum depression.

11. Methods of Psychological Intervention

• Individual interviews with psychologists specializing in chronic diseases and pregnancy.
• Cognitive-behavioral therapies (CBT) to modify dysfunctional thoughts related to pain and improve adaptation.
• Emotional and informational support, providing clear explanations of the pathology and therapeutic possibilities to reduce uncertainty and fear.
• Involvement of the family or partner to create an effective social support network.

12. Benefits of Psychological Support

• Improved quality of life and therapeutic compliance.
• Reduction of anxious and depressive symptoms secondary to the disease.
• Promotes better psychological adaptation to pregnancy complicated by algodystrophy.
• Supports the prevention of postpartum psychological disorders and facilitates mother-child bonding.

13. Our Experience

The primary forms concern a quarter of the cases in disturbed or anxious personalities with a depressive-anxious psychometric evaluation. Favorable factors are diabetes and hyperuricemia, hypertriglyceridemia, bed rest. The secondary forms can follow a trauma even of modest entity, to central or peripheral neurological affection, to ischemic heart disease, to prolonged pharmacological treatments with barbiturates and pregnancy. [1-3].

14. Materials and methods of the study

to evaluate the accuracy of the administration of anamnestic and clinical tests in the screening of people to be sent for the execution of nuclear magnetic resonance of the femoral head during or after pregnancy. Furthermore, in order to control the frequency of presentation of the pathology in a hospital department of obstetrics as a cause of pain in the pelvis or in the others or in the lower limbs, we selected all the cases of pain in the region of the pelvis and the lower limbs for which a physiatric consultation was requested and all the pregnant or puerperium patients visited in the physiatric clinics of our hospital.

15. Selection

15.1 The Selection Was Made on The Basis of The Following Inclusion Criteria

15.1.2 Anamnestic Data

• pain with inguinal localization in the thigh or gluteal area
• accentuation of pain with load
• improvement of pain with rest worsening of painful disorders in the peripartum period.

15.1.3 Clinical Data

• pain with rotations Coxo femoral with flexed hip
• negativity of the radicular tension tests the following
• negativity of the succussion and lumbar palpation tests.

All women in whom the pain, even if irradiated to the buttocks or lower limbs, had a prevalent lumbosacral localization and the cases in which the anamnestic and clinical data did not correspond to the inclusion criteria were excluded from the study. The selection was always performed by the same operator; the definitive diagnosis was obtained by performing a nuclear magnetic resonance of the femoral heads evaluated by an independent operator. Pain measurements were reported with a N.R.S. scale and with the M.P.Q. questionnaire and the measurement of hip articulation in flexion, extension and rotation and the values of the mini Mental test score [1-4].

16. Results

on an average of 3500 admissions to the obstetrics department in a period of 8 years from 1998 to 2005, the deliveries were on average 3,330 per year the requests for specialist physiatric consultations on an annual average of 48 the selected cases that met all the inclusion criteria were 18. Of these in thirteen cases the diagnosis was of reflex algodystrophy of the femoral heads, in particular in two cases the localization was bilateral and in one woman the pathology occurred in both the first and second pregnancy. In a second subsequent three-year case study from 2021 to 2023, 8 more cases were identified that were not included in the previous study concluded in 2006. The higher number of cases identified with the second case study indicates that the diagnostic criteria and attention to this pathology have improved over the years [4].

In conclusion, from the study conducted the algodystrophy of the femoral heads in pregnancy although representing a pathology with a low frequency of presentation and therefore little known is however underestimated. In the cases resulting from the hospital discharge forms, since it is often confused with sciatica, a careful clinical examination allows us to reach a diagnostic accuracy of 73.3% in our cases, thus preventing complications which, although infrequent, are highly disabling.

17. Conservative Approach

Joint unloading: Use of crutches to reduce pressure on the femoral head.

18. Physiotherapy

• Passive mobilization and desensitization.
• Magnetotherapy to reduce edema.

19. Drugs

• Bisphosphonates (e.g., neridronate): Effective in 60-70% of

cases if administered early, with reduction of pain and edema.
• Vitamin C: Used prophylactically to prevent post-traumatic algodystrophy.

20. Monitoring
• Control MRI: At 3 and 6 months to assess the regression of edema.

21. Prognosis and Complications
Favorable course: In 80% of cases, complete recovery within 6-12 months.
Risks:
• Evolution to osteonecrosis (15-20% of cases).
• Fragility fractures due to transient osteopenia.

22. Special Considerations in Pregnancy
• Limited diagnostics: Avoid X-rays and bone scintigraphy; prefer MRI.
• Safe therapies: Promote non-pharmacological approaches (physiotherapy) and evaluate the risks/benefits of bisphosphonates.

23. Conclusions
CRPS is a complex, chronic, and potentially disabling pathology that requires a timely and multidisciplinary therapeutic approach. Current research suggests that, contrary to what was previously believed, early and targeted management can significantly improve patients' quality of life and functionality. Femoral head algodystrophy in pregnancy requires a multidisciplinary approach, with emphasis on early diagnosis via MRI and conservative therapies. Although often self-limiting, failure to manage it can lead to osteoarticular complications. Future studies should clarify the role of vascular and metabolic imbalances typical of pregnancy in pathogenesis [5].

Femoral head algodystrophy in pregnancy requires an integrated approach that includes not only clinical and pharmacological management, but also psychological support. Timely and targeted psychological intervention is essential to reduce emotional distress, improve adaptation to the disease, and optimize both maternal and fetal outcomes [6-9].

Bibliography

1. Gougeon, J., Eschard, J. P., Moreau-Hottin, J., Fmncon, J., David-Chausse, J., Doury, R. (1982). "L'algoditrophie: developpement, formes polyarticulaires, for¬mes recidivantes ". *Rev. Rhum. Mal. Osteoartic. 49*: 809-814.
2. Pélissier, J., Viel, E., Chauvineau, V., Ricard, C., Romain, M., & Hérisson, C. (2003). Algodystrophy or complex regional pain syndrome type I. Encycl Med Chir , 26-293.
3. Roberts, W. J. (1986). A hypothesis on the physiological basis for causalgia and related pains. *Pain, 24*(3), 297-311.
4. Stanton-Hicks, M., Jänig, W., Hassenbusch, S. A., Haddox, Boas, R., & Wilson, P. (1995). Reflex sympathetic dystrophy: changing concepts and taxonomy. *Pain, 63*(1), 127-133.
5. Pham, T., & Lafforgue, P. (2003). Reflex sympathetic dystrophy syndrome and neuromediators. *Joint Bone Spine, 70*(1), 12-17.
6. Gobelet, C., Meier, J. L., Schaffner, W., Bischof-Delaloye, A., Gerster, J. C., & Burckhardt, P. (1986). Calcitonin and reflex sympathetic dystrophy syndrome. *Clinical rheumatology, 5,* 382-388.
7. Devogelear, J. P., Dall'Armellina, S., Hoax, J. P., Nagan de Deux-chaisnes, C. (1988). " Dramatic improvement of intractable reflex sympathetic dystrophy syndrome by intraveinous infusions of the second generation bypho¬sphonate apd " *J. Bone Miner. Res,*3: 213.
8. Adami, S., Fossaluzza, V., Gatti, D., Fracassi, E., & Braga, V. (1997). Bisphosphonate therapy of reflex sympathetic dystrophy syndrome. *Annals of the rheumatic diseases, 56*(3), 201-204.
9. Mandalà, G., Mazzola, F. G., Valenti, E. (2006). "L'algodistrofia riflessa della testa femorale in gravidanza: accuratezza dei test anamnestici e clinici per uno screening" *Eur. Med. Phys. 42* (Suppl. 1 to n. 3) 445-447.