Fat Mass% (FM,%), Lean Mass (LM, kg), Bone Mineral Content (BMC, mg), and Fat Mass Index (FMI: trunk fat mass/legs fat mass). Statistics. Mean $\pm$ SD, ANOVA, Scheffe's F procedure for post-hoc comparison.

Results Lean Mass was the same in the 4 groups. Despite 43 months of disease duration, Naive and Controls had the same fat mass, BMC and FMI. The two treated HIV-infected groups had lower Fat Mass and upper Fat Mass Index than Controls. Loss in Fat Mass at the expense of the legs was proportional to duration of HAART. The great variability of the FMI (SD) in these two groups suggested a poor clinical sensitivity to change. Decrease of BMC seemed treatment-dependent. Lipodystrophy appeared also age-related.

Abstract SAT0222 Table 1

	BMI	BMC	Lean mass	Fat mass	Fat mass index
Controls (1)	25	3197	58.7	16.1	2.04 $\pm$ 0.43
Naive (2)	25	3061	61.2	12.4	2.07 $\pm$ 0.38
n-CLD (3)	23	2859	58.8	9.8	2.49 $\pm$ 0.90
CLD (4)	23	2700	57.6	9.3	3.46 $\pm$ 1.10
ANOVA	P < 0.05	P < 0.05	n.s.	P < 0.05	P < 0.05
Scheffe (P < 0.05)	1-4, 2-4	1-3, 1-4, 2-4	n.s.	1-3, 1-4	1-3, 1-4, 2-4, 3-4

Conclusion DEXA allows an early diagnosis of fat maldistribution. We propose to perform this exam to all HIV-infected patients once a year before treatment and every 6 months in patients under treatment.

SAT0223 LYME MENINGORADICULITIS IN AN ENDEMIC ZONE: CLINICAL SIGNS

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Background Neuroborreliosis has been called "the new great imitator".

Objectives The aim of this study was to define the symptoms of meningoradiculitis.

Methods We collected prospective data from all patients admitted to hospital with Lyme meningoradiculitis between June 1st, 1998 and December 31st, 1999 in an endemic zone located in western France. Diagnosis was based on lymphocytic meningitis associated with either intrathecal specific antibody synthesis or high serum titers of specific IgM in ELISA and western Blot analysis.

Results Eleven patients (9 women, 2 men), with a mean age of 62 years (range: 37 to 87) were included during the study period. Two thirds of these patients were admitted to rheumatology wards during the summer. The onset of symptoms was preceded, 5 to 60 days earlier, by a tick bite in 64% of cases or erythema migrans in 55% of cases. In 18% of patients, neither a tick bite nor erythema was observed. Non specific symptoms were recorded in 82% of cases, with transient fever or flu syndrome in 45% of all cases. Cervical radiculoneuritis was observed in 4 cases, symptoms affecting several nerve roots of the lower limbs in 3 cases, femoral neuropathy in 2 cases, a

bilateral sensory radiculoneuritis T11-12 in one case and poorly localised paresthesias in the lower limbs in one case. The meningeal signs and symptoms were minimal or, more frequently, absent. A peripheral motor paresis was observed in one quarter of the cases, but without Bell's palsy or encephalic or medullary signs. In half the patients, there was associated spinal pain not relieved by rest. All patients reported nocturnal exacerbation in pain. The electrocardiographic abnormalities included first-degree atrioventricular block in 2 patients. The course of the disease was consistently favourable on ceftriaxone IV treatment, with rapid effective relief of pain.

Conclusion A diagnosis of Lyme meningoradiculitis should be considered in endemic zones in all cases of spine pain and/or radiculitis with nocturnal exacerbation, especially in the summer period.

SAT0224 PERIPHERAL NERVE INVOLVEMENT IN HCV-INFECTED PATIENTS WITH AND WITHOUT MIXED CRYOGLOBULINEMIA

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Background In contrast to cutaneous vasculitis, the morphology of which is well known, that of peripheral nerve lesions in HCV-positive mixed cryoglobulinemia (MC) patients are still under debate.^{1,2}

Objectives To evaluate the pathological processes underlying the peripheral nerve damage in HCV-infected patients, with and without MC.

Methods From 14 HCV-infected patients (10 with MC and 4 without), all with clinically peripheral neuropathy, a sural nerve biopsy was obtained. Diseases other than HCV infection were excluded. The tissue samples were processed according to standard methods to perform morphologic and morphometric evaluation, on light and electronmicroscopic study. An immunohistochemical evaluation was carried out using labelled antisera against immunoglobulins (IgA, IgG, IgM), complement proteins (C1q, C3, terminal complex C5-9), and cellular antigens (CD3, CD11a-b-c, CD20, CD45, CD68).

Results In all nerve biopsies an axonal involvement without a distinctive vasculitis was found. Scarce perivascular infiltration by mononuclear cells was observed, particularly around perineural blood vessels; extravasation of red cells was also found in the endoneurial vessels. Immunohistochemical study pointed out deposits of immunoglobulins and complement in sub-perineurial vessels, and infiltrations by mononuclear cells, mainly macrophages and CD4 T cells. Demyelinating neuropathy was only observed in patients without MC.

Conclusion Our data suggest that in HCV-infected patients different physiopathological processes are involved in induction of peripheral nerve damage. A potential direct pathogenic role of HCV should be investigated.

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Miscellaneous rheumatic diseases

AB0207 REFLEX SYMPATHETIC DYSTROPHY ASSOCIATED WITH PHENOBARBITAL

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Background Reflex sympathetic dystrophy (RSD) is a complex syndrome characterised by pain and oedema of one or more extremities, trophic skin changes and vasomotor instability. Long-term outcome is variable and can induce severe consequences. Barbiturates has been related with RSD in 15% of the cases. **Objectives** The aim of our work is to define long-term outcome and response to therapy of three patients with RSD following barbiturates use.

Methods

Results Main results are expressed in the next Table 1:

Abstract AB0207 Table 1

	Sex and age	Distribution	Response to barbiturates discontinuation	Response to steroids/calcitonin	Sequels (1 year)
Patient 1	Male/ 70	Both upper limbs	No	No	+
Patient 2	Female/ 60	Both upper limbs	No	No	+
Patient 3	Female/ 54	Right upper limb	±	+	±

Conclusion RSD can be induced with barbiturates therapy. In our experience barbiturates discontinuation is insufficient for improving and response to other therapies can be limited with significant long-term disability.

AB0208 CYCLOPHOSPHAMIDE MAY INDUCE REMISSION OF RELAPSING POLYCHONDritis REFRACTORY TO CORTICOSTEROIDS

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Background Relapsing polychondritis (RP) is so rare that controlled therapy trials have not been carried out. Therefore, strategies for the treatment of RP refractory to moderate- or high-dose corticosteroids (CS) have not been established.

Objectives To report a severe case of RP, refractory to CS, in which complete remission was induced by cyclophosphamide (CP) therapy.

Methods Retrospective clinical and laboratory analysis of one patient.

Results A 55-year-old Caucasian woman developed ear and nose cartilage inflammation, for the first time, when she was 31. At that time a biopsy of auricular cartilage confirmed the clinical diagnosis of RP. The disease responded to an initial short-term CS therapy (20 mg prednisolone/day), but a maintenance dose

of 5–10 mg prednisolone/day was required to control symptoms and prevent relapses ever since. Meanwhile, she developed diabetes mellitus and cataract. Additionally, she had chronic anaemia due to thalassemia minor. Sixteen months ago, after strenuous physical exercise, a severe relapse of the disease occurred suddenly. The patient presented with acute arthritis of the knees and wrists, chondritis of the auricular, nasal, and laryngotracheal cartilages, as well as symptoms of eye inflammation. She complained of severe arthralgias, hoarseness, cough, eye irritation, tinnitus, and vertigo. Physical examination revealed fever (38°C), tenderness of thyroid cartilage and the anterior trachea, bilateral episcleritis and cataract, cochlear and vestibular dysfunction, and effusion of both knees. Laboratory investigation showed an erythrocyte sedimentation rate of 41 mm, C-reactive protein 15.5 mg/dl (normal <0.9 mg/dl), glucose 216 mg/dl, hematocrit 35%, haemoglobin concentration 10.3 g/dl, white blood cell count 10,540/μl with 71% polymorphs, platelet count 477,000/μl, negative rheumatoid factor, microscopic hematuria, and inflammatory synovial fluid, containing 15,800 white cells/μl (95% polymorphs, 5% lymphocytes), but no crystals or microorganisms. The manifestations of laryngotracheal, ocular, and inner ear inflammation were refractory to 60 mg prednisolone/day for four weeks. Then, oral CP (1.5 mg/Kg/day) was added and a quick improvement ensued. Within four months of CP therapy complete clinical remission of the disease and normalisation of laboratory tests was observed. Subsequently, CP was discontinued and prednisolone was gradually tapered off over the next six months. Eventually, five months after stopping CS, the disease still remains quiescent without therapy.

Conclusion Although RP may have a remitting course, the remission in our patient has most likely been induced by CP in combination with prednisolone. Our observation suggests that CP is an effective therapy for severe cases of RP refractory to CS alone. Moreover, CP seems capable of inducing sustained remission of this disease.

AB0209 LONG TERM ANALYSIS AND EVALUATION OF SIDE EFFECTS RELATED TO CYCLOSPORINE TREATMENT OF 59 PATIENTS WITH RHEUMATIC DISEASES

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Background Cyclosporine has been used in the area of rheumatology, and various published articles describe evidence of side effects in short and long term, which have lead to withdrawal of the treatment.

Objectives Evaluate the presence of side effects in 59 patients that received cyclosporine treatment in dosis of 2.5–3 mg/Kg/day.

Methods This is a prospective study which carried out 59 patients that received cyclosporine treatment: 45 females with an average age of 57 years (range 23–80) and 15 males with an average age of 50.6 years (range 31–72) with the follow disease: 41 Rheumatoid Arthritis, 6 Psoriatic Arthritis and tha remainder included sufferers of Systemic Lupus Erythematosus, Scleroderma, Ankylosing Spondylitis and Cryoglobulinemia. Female patients suffering from the illness for an average of 19.95 years, were treated for a total period of 22.11 months. Male patients suffering from the illness for an average of 8.7 years, were treated for a total period of 19.4 months.