Other potential changes in this syndrome include pre-
excitation syndrome, left ventricular hypertrophy, dilated
cardiomyopathy, retinal macular dystrophy, intestinal
pseudo-occlusion, neuropsychiatric changes, lactic acidosis,
and complications during pregnancy (prematurity, placenta
accreta).\(^6\)

Diagnosis is based on molecular studies which look for the
mtDNA mutation. Two techniques may be used, sequencing
and PCR-RFLP.

Treatment consists of the administration of coenzyme
Q10, an electron transporter of the respiratory chain which
acts as an antioxidant, protecting cell membrane phospho-
lipids and low density lipoprotein cholesterol from the
oxidative damage caused by free radicals. Administration of
Q10 at a dosage of 150 mg/day for three years delays the
occurrence of insulinopenia and hearing loss and decreases
post-exercise lactate levels.\(^9\) However, other authors have
not supported these findings.\(^10\) Statins should be used with
care because they decrease coenzyme Q10 levels. Other
treatments used in other mitochondrial syndromes include
arginine, L-carnitine, and multivitamin complexes.

In conclusion, although the prevalence of mitochondrial
diabetes in the diabetic population is low, its diagnosis is
important because it has a different prognosis and treat-
ment. It should be suspected in the presence of a personal
and/or family history of diabetes and deafness or microvas-
cular complications that do not correlate with diabetes
duration, and in slim diabetic patients with negative pan-
creatic autoimmunity.

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Lupus, Graves’ disease, and vasculitis.
A case report\(^8\)

Lupus, enfermedad de graves y vasculitis.
A propósito de un caso

Autoimmune thyroid disease (AITD), characterized by the
presence of antibodies against thyroid antigens, is associ-
ated with a number of non-organ-specific rheumatological
disorders such as systemic lupus erythematosus (SLE),
rheumatoid arthritis, and Sjögren’s syndrome.\(^1,2\) For ex-
ample, while the prevalence of subclinical hypothyroidism
(SCH, normal T4 with elevated TSH) is 4.3% in the general
population,\(^3\) it is increased to 11–13% in patients with SLE.\(^4,5\)
Similarly, the prevalence of anti-thyroid peroxidase antibod-
ies (anti-TPO) is 10–12% in the general population,\(^3,6\) but
17–23% in SLE patients.\(^4,5\) Clinical or subclinical hypo-
thyroidism is usually common in patients with SLE and accounts
for more than 87.5% of thyroid function changes in these
patients.\(^5,7\) As regards Graves’ disease (GD), it is less com-
mon in both the general population (0.5%)\(^8\) and patients with
SLE (1.7%).\(^1\)

Anti-thyroid drugs are associated with adverse events,
and most of them are mild and uncommon (less than 5%),
but, in some cases, they may cause severe autoimmune
disorders such as vasculitis, polyarthritis, or drug-induced
lupus.\(^8\) Vasculitis associated with antithyroid drugs usually
consists of a combination of polyarthralgia and skin lesions.
However, it may affect other organs such as kidney, lung,
gastrointestinal tract, or brain. We report the case of a female patient recently diagnosed with SLE and renal impairment who showed GD and worsening of lupus nephritis due to vasculitis associated with the use of dipyrone after being symptom-free for three years.

A 24-year-old female patient with an unremarkable clinical history was admitted to hospital for the recent onset of a nephrotic syndrome (24-h proteinuria, 8.75 g/day) and moderate renal failure (glomerular filtration rate, 80 mL/min). The result of a renal biopsy was consistent with class V lupus nephritis (diffuse membranous glomerulonephritis). Positive ANA and anti-DNA tests completed the diagnostic criteria. Treatment was therefore started with prednisone 60 mg/day and bimonthly intravenous cyclophosphamide pulses. The clinical course was characterized by the remission of proteinuria, asymptomatic SLE, and the occurrence of iatrogenic Cushing syndrome.

Three years later, the patient reported fine hand tremor, heat intolerance, palpitations, and weight decrease. Physical examination showed exophthalmos, diffuse goiter (60 g), and hyperreflexia. Laboratory test results included TSH 0.042 μIU/mL (normal, 0.3–5), free T4 6.85 ng/dL (normal, 0.8–2), and total T3 415 ng/dL (normal, 86–190). Anti-TPO antibodies were positive, and iodine uptake was 60% at 24 h (normal, 15–35). Anti-TSH receptor antibodies were not measured. These results confirmed the diagnosis of GD, and treatment was started with dipyrone 20 mg/day and propranolol 40 mg three times daily. After one week of treatment, the patient experienced malaise, fatigue, joint pain, and generalized edema. A repeat 24-h urine protein test showed a value in the nephrotic range. In our department, it was assumed that renal impairment was secondary to vasculitis induced by antithyroid drugs, and dipyrone was therefore discontinued. No repeat renal biopsy or measurement of antineutrophil cytoplasmic antibodies (ANCA) was performed. The disease activity index for SLE (SLEDAI) was not available in Spain) than with dipyrone and is not dose-dependent. This condition is usually characterized by renal impairment, arthritis, skin ulcers, and respiratory symptoms. Thionamides have been shown to accumulate in neutrophils, where they induce the production of toxic substances which finally act as immunogens.

In our patient, the administration of dipyrone triggered a generalized inflammatory process characterized by proteinuria and SLE exacerbation. The discontinuation of the triggering agent and the use of glucocorticoids resulted in a significant improvement.

In conclusion, frequent evaluation of the thyroid profile and the presence of anti-thyroid antibodies is recommended in patients with SLE.

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