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V. Esteve, M. Pou, F. Latorre*
and M. Ramírez de Arellano
Nephrology and Vascular Surgery Departments. Health Complex of Terrassa. Barcelona.*
Correspondence: *Vicente Esteve Simó. viesi@hotmail.com. Consorci Sanitari de Terrassa. Crta. Torrebonica, s/n. 08227 Barcelona. España.*

Graves disease, drug-related hypothyroidism, and nephrotic syndrome due to minimal changes disease

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To the editor: A few cases of glomerular diseases associated to thyroid diseases have been described.[1-6]

A 41-year old female went to the General Practitioner because of malaise and palpitations. On blood analysis, we only detected abnormal free T4 (fT4) (100 pmol/L) and TSH (0.01 mIU/mL) values (normal fT4 levels: 12-22 pmol/L and normal TSH levels 0.3-4.2 mIU/mL), compatible with hyperthyroidism. A Doppler ultrasound of the thyroid showed a homogeneous increase in size and diffuse increased uptake with signs of hypervascularization. A diagnosis of Graves-Basedow's disease was made and symptomatic treatment with beta-blockers and synthetic antithyroid drugs (methimazole) was initiated.

One month later she went to the hospital because of asthenia, generalized edemas and anasarca, and intolerance to cold. Blood analysis disclosed hypoalbuminemia (14.9 mg/dL) and hypercholesterolemia (350 mg/dL) with normal renal function. Urinary sediment did not show activity signs. A proteinuria of 6.5 g/day was detected. The diagnosis was oriented to pure nephrotic syndrome, as well as iatrogenic hypothyroidism due to excessive doses of anti-thyroid drugs. Treatment was initiated with levothyroxine, ACE inhibitors, diuretics and statins. The immunological investigations (including immunoglobulin levels, complement fractions, cryoglobulins, ANA, ANCA and anti-basement membrane antibodies), viral serology (HBV, HIV, HCV), and tumoral markers (AFP, CEA, Ca 12.5, Ca. 15.3, Ca 19.9) were unremarkable. A percutaneous renal biopsy was performed, which showed glomeruli with no hypercellularity, no alterations in the capillary wall, no tubular atrophy, no significant inflammatory infiltrate and negative immunofluorescence, compatible with minimal changes disease. With the treatment, the evolution was rapidly favorable, the thyroid hormones recovered, the edemas disappeared, and the proteinuria completely regressed (0.16 g/day).

The most frequent thyroid condition associated to renal alterations is Graves' disease. The most frequently reported associated renal condition in these cases is membranous glomerulonephritis with nephrotic syndrome.[3,4] However, there are sporadic cases of membranoproliferative glomerulonephritis or minimal changes disease associated to thyroid disease.[1,5,6] Some authors suggest that the incidence of the association of glomerular alterations and thyroid conditions could be higher than suspected, since the presence of constant proteinuria is not infrequent when a diagnosis of autoimmune thyroiditis is made.[2] The coexistence of the two pathologies could be explained by a common autoimmune pathogenesis. On the other hand, nephrotic syndrome secondary to structural changes of the glomerular basement membrane and tubular membrane has been reported in cases of sustained hypothyroidism.[2,7-9] Given the temporal coincidence of the diagnosis and the nephrotic flare after the iatrogenic hypothyroidism, we think that in the reported patient the underlying thyroid disease provoked a glomerular alteration, which was maintained by the situation of hypothyroidism. Most patients are controlled with steroids or other immunosuppressive drugs, although therapy with iodine or radical thyroidectomy has been efficacious in those patients with repetitive flares.[10,11] Immunosuppressive therapy was not initiated in this patient because of the rapid improvement.

In summary, we report a patient who presented minimal changes disease associated to Graves' disease in the setting of pharmacological hypothyroidism. The infrequent association of both entities and the complete remission without immunosuppressive therapy are remarkable.

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V. Esteve Simó, N. Fontseré, A. Saurina and M. Ramírez de Arellano
Nephrology Department. Health Complex of Terrassa. Barcelona.
Correspondence: *Vicente Esteve Simó. viesi@hotmail.com. Consorci Sanitari de Terrassa. Crta. Torrebonica, s/n. 08227 Barcelona. España.*

Necrotizing fasciitis and acute renal failure in mesangiocapillary glomerulonephritis

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To the editor: Necrotizing fasciitis is a condition with a high mortality.[1] which is frequently complicated by acute renal failure,[2] and seems to be favored in the presence of an underlying immunosuppression.[3] We present a patient with hypocomplementemic mesangiocapillary glomerulonephritis, who developed necrotizing fasciitis. A 62 year-old patient had a history of high blood pressure, smoking habit, dyslipidemia, and idiopathic type I mesangiocapillary glomerulonephritis, with previous serum creatinine levels of 1.9 mg/dL and nephrotic syndrome. Six months earlier, two stents were placed in the right common iliac artery and the right external iliac artery, because of stenosis due to arteriosclerosis. He went to the Emergency Room because of pain and swelling on the right inguinal area. Two hours later, the patient presented great local edema and increasing pain. On CT scan superficial and deep cellulitis, together with myositis at the proximal right thigh and internal compartment, could be seen. Blood analysis revealed leucopoenia (2,680/mm^3), serum creatinine levels of 6.2 mg/dL and CK levels of 333. In the following hours, the patient developed septic shock with respiratory distress and acute renal failure, and he was transferred to the Intensive Care Unit. Once the hemodynamic situation was stabilized, the patient underwent urgent fasciotomy with decompression of the medial and anterior compartments. The muscle had a ground-glass appearance, the fascias were enlarged and there was a small amount of gas (fig. 1). Muscular biopsy showed inflammatory infiltrate with marked neutrophilic predominance and the culture yielded *E. coli* sensitive to piperacillin-tazobactam and amikacin. Fourteen days later, fasciectomy and debridement of the necrotic ulcer were required, and signs of necrotizing fasciitis were found. At this moment, *Stenotrophomonas maltophilia* sensitive to cotrimoxazol was cultured. The evolution was very slow and the patient was discharged from the hospital after 6 months of in-hospital stay, two of them at the Intensive Care Unit. During the process, the renal function was not recovered and continuous hemodiafiltration and later hemodialysis were required.

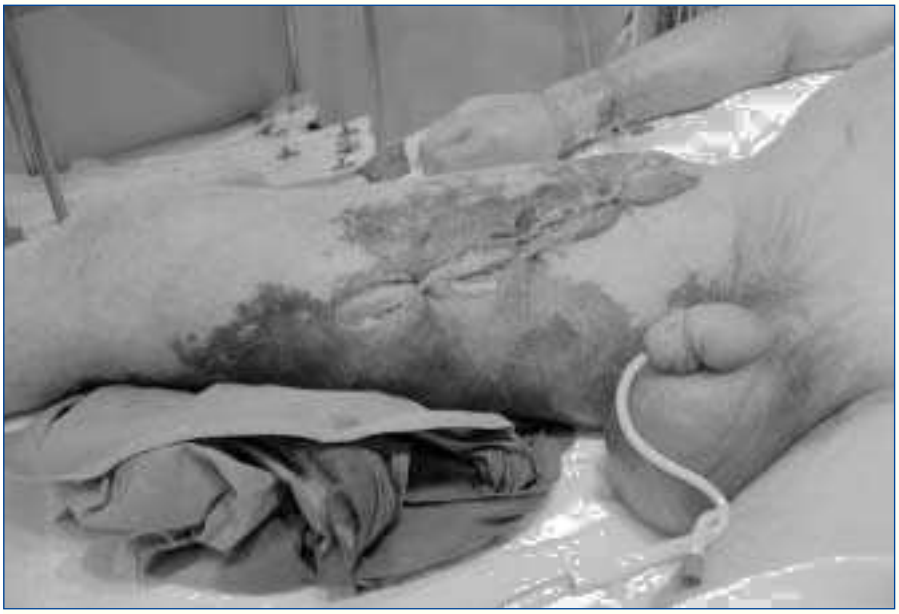

Figure 1. Right inferior limb at 24 hours after admission, after fasciotomy. Intense inflammation and edema.

Necrotizing fasciitis is an infrequent disease with a rapid onset, in which early diagnosis is paramount. It is characterized by rapid destruction of tissues, toxic systemic signs and high mortality.[1] The infection that localizes in subcutaneous tissue progressively destroys the fascia and fat tissue, and sometimes involves the skin. It can occur in immunocompetent people, however the risk is higher in immunosuppressed patients.[3] The diagnosis is made by means of a CT scan,[4] but if the clinical picture is suspected the surgical intervention should not be delayed.[1,3]

A survey performed in Ontario in 1997[3] showed that in 71% of the cases at least one underlying chronic condition was present, such as heart disease, peripheral vascular disease, pulmonary disease, renal failure, diabetes mellitus, alcohol abuse, and immunodepression from different causes. The mean age was 57.5 years, with male predominance. Acute renal failure was seen in 35% of the cases, while respiratory distress syndrome was seen in 14%. The global mortality rate was 34%, but was closed to 100% in those patients that were not operated.

In the reported case, as well as in other recently described,[5] previous renal failure and alteration of the immunity could have promoted the fasciitis. Surgical intervention in the first 24 hours played a decisive role for the resolution of the clinical picture. In spite of the loss of renal function and the prolonged hospital stay, the patient has currently an acceptable quality of life.

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