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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.

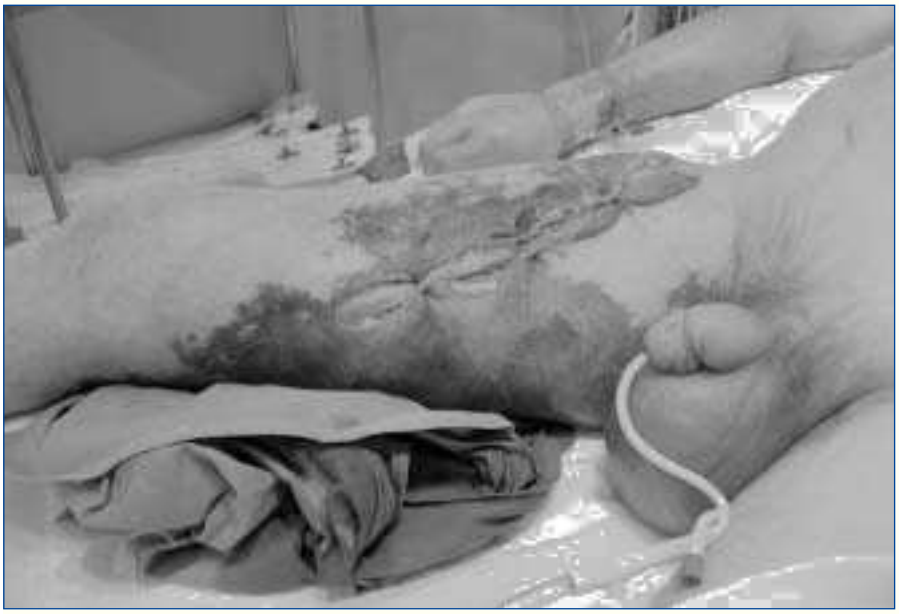

Figure 1. Right inferior limb at 24 hours after admission, after fasciotomy. Intense inflammation and edema.

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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Wegener granulomatosis and cancer

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To the editor: A 50 year-old male is presented, who is on periodic hemodialysis since April of 1984 because of chronic renal failure after rapidly progressive glomerulonephritis secondary to Wegener' s granulomatosis.

Wegener's granulomatosis was first described in 1931 by Klinger.[1] In 1936, Wegener clearly defined the clinical and pathological picture.[2] The etiology remains unknown. It is a systemic necrotizing vasculitis, which involves small arteries and veins, above all in the upper and lower respiratory tracts and the kidney.[3]

Renal involvement manifests with proteinuria, hematuria and red cell casts, and progressive renal failure. The most frequent pathological finding is ~~a~~ focal necrotizing glomerulonephritis. Interstitial nephropathy and vasculitis, as well as proliferative glomerulonephritis, can also be found.[4]

In January of 1984, a renal biopsy was performed and extracapillary necrotizing glomerulonephritis was observed. A biopsy of the nasal cavities was normal.

The patient received immunosuppressive therapy with steroids and cyclophosphamide, but the renal function did not recover.

In February of 1995, the patient presented a flare of vasculitis, which consisted in the development of a necrotizing ulcer at the internal malleolus of the right foot, pharyngitis, and diffuse intraalveolar hemorrhage with a radiological pattern of bilateral alveolar infiltrate.

In the lungs, nodules, atelectasis, pleural effusion and infiltrates can be seen. Cavitation is frequent.[5]

At that moment, the patient had positive anti-neutrophilic cytoplasm autoantibodies (c-ANCA) with specificity for anti-proteinase 3.

The four diagnostic criteria are nasal or oral inflammation, abnormal radiological image, signs of activity in the urinary sediment, and demonstration of granulomatous inflammation.[6] The presence of ANCA has also been shown to be useful for making the diagnosis.[7,8]

The patient reinitiated treatment with oral cyclophosphamide (1 mg/kg/day) and prednisone (60 mg PO with progressive dose reduction). The treatment was maintained for more than a year.

It is known that cyclophosphamide is associated with bladder cancer, due to the action of a drug metabolite, called acrolein, which has also been related to a higher incidence of skin cancer and myeloproliferative syndromes.[9]

In May of 2002, he was diagnosed with a stage IV A non-Hodgkin lymphoma by means of a lymph node biopsy. He was treated with 6 cycles of CHOP and rituximab with partial remission. Since 2003, he has been on maintenance therapy with periodic cycles of rituximab.

In September of 2007, twenty-three years after the diagnosis of Wegener's disease, he presented an upper respiratory tract infection and a chest X-ray film was performed, which showed left pleural effusion not present on the control CT scan performed in January (fig. 1). Immunological investigations were negative. A new CT scan was made, which confirmed the presence of left pleural effusion and multiple pulmonary nodules. Diagnostic and therapeutic thoracocentesis was performed. The fluid was hematic in appearance and the biochemistry was compatible with an exudate. The pathological study disclosed an adenocarcinoma of the lung and palliative therapy was initiated.

It is well known that Wegener's disease has a fatal prognosis if left untreated, but the treatment is on the long term associated to an increased cancer risk, as clearly illustrated by this case.

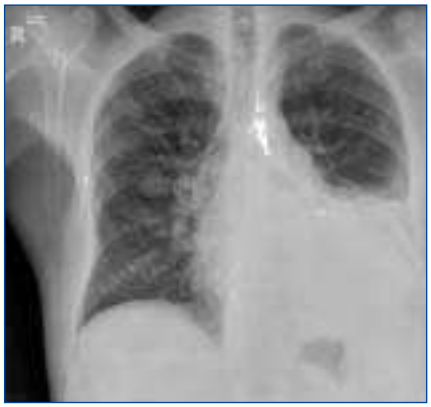

Figure 1. Left pleural effusion and multiple pulmonary nodes.

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