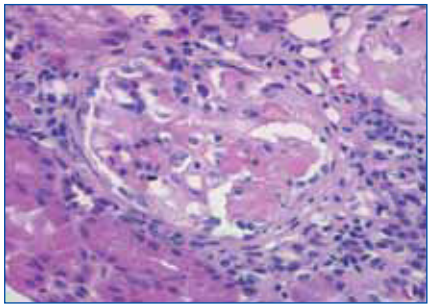

Figure 1. Optical microscope. Glomerule with fibrinoid necrosis. HE stain, x20.

was followed by mycophenolate mofetil at 500mg/8hr and levothyroxine at 75μg/day. At 6 months from diagnosis, the haemoglobin (Hb) was 12.6g/dl; creatinine: 1.4mg/dl; sediment: 10-20 red blood cells/field; ANCA were negative and thoracic CT did not show infiltrates.

Pulmonary bleeding symptoms include haemoptysis, coughing, chest pain, dyspnoea, anaemia and acute respiratory failure. Chronic evolution of pulmonary bleeding in MPA with non-specific symptoms and iron deficiency, as occurred with our patient, is less documented.[2,5] However, in Lauque et al's study, 28% of patients had symptoms for more than a year before the pulmonary bleeding was diagnosed.[2]

Autoimmune thyroiditis is associated with glomerulopathies, especially with membranous,[4] membranoproliferative[6] and IgA glomerulonephritis.[7] A common pathogenic link is suggested given that the two autoimmune diseases are present at the same time.[4] It is thought that thyroglobulin and thyroid peroxidase released by destroying the thyroid follicles would be deposited in the kidney and *in situ* immunocomplexes would be formed; depositing of circulating immunocomplexes seems less likely.[8] In the case of autoimmune thyroiditis with ANCA vasculitis, other mechanisms could be involved, such as polyreactive antibody development and regulator and effector T cell alterations.[8] With regards the antibodies, it is worth highlighting that myeloperoxidase shares a certain structural homology with thyroid peroxidase and that it could produce cross-over reactions,[9] but other authors do not support this mechanism.[10] Although the association between autoimmune thyroiditis and ANCA-positive vasculitis does not seem to be common, Tanaka et al[9] found that 4 cases out of a series of 10 with ANCA-positive nephropathy had hypothyroidism, 2 of which were subclinical.

To conclude, it is important to consider that pulmonary bleeding in the MPA may evolve over a long period, with no marked haemoptysis or changes visible in the chest X-ray, being the cause of iron deficiency anaemia. For patients with ANCA vasculitis it is convenient to determine thyroid function and anti-TPO antibodies, and for those with autoimmune thyroiditis, the possibility that patients have an underlying nephropathy must be assessed.

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Mushroom poisoning: Orellanus syndrome

Nefrologia 2011;31(4):495-6

doi:10.3265/Nefrologia.pre2011.Apr.10824

To the Editor,

Around 42 million kilograms of wild mushrooms are eaten in Spain each year. Mushroom poisoning is an increasingly common medical emergency. Annually, 400 people are hospitalised with this diagnosis. Depending on the latent period (around six hours), clinical symptoms can be classified as short- or long-incubation syndrome. The latter are the most severe,

and include gyromitra, orellanus and phalloides syndrome.[1]

The orellanus syndrome is produced by several species from the genus *Cortinarius* that contains toxins, orellanines, with a marked renal tropism. After a long period without any symptoms (3-17 days), the patient presents with polyuria and severe renal failure, which is often irreversible.[2-4] We present a case of mushroom poisoning with signs of orrellanus syndrome.

We present the case of an 83-year-old male patient with history of arterial hypertension (AHT), dyslipidaemia and a haemorrhagic stroke in 2006. He underwent surgery for rectal neoplasia in 2000, and received chemotherapy and adjuvant radiotherapy. The patient came to the emergency department due to vomiting and liquid bowel movements, with no other symptoms. The only event that the patient referred was having eaten wild mushrooms that he had picked 4 days before. The biochemistry found: glucose: 130 mg/dl; urea: 240 mg/dl; creatinine: 4.62 mg/dl; glutamate-pyruvate transaminase (GPT): 3903IU/l; glutamate-oxaloacetate transaminase (GOT): 868IU/l; bilirubin: 0.80mg/dl; amylase: 86IU/l, CK: 86IU/l; sodium: 134mEq/l; potassium: 5.1mEq/l; ionic calcium: 1.12mmol/l; and lactate: 1.3mmol/l. The blood gases showed a pH of 7392 and HCO_3 of 15.1mEq/l. The haemogram showed thrombocytopenia with $69x10^3/\mu l$ of platelets; the rest of the haemogram and coagulation were normal. The abdominal ultrasound did not show any changes. Given that it was suspected that the patient had mushroom poisoning, he was admitted to the intensive care unit (ICU). Intensive fluid therapy was started, with sibilin and penicillin G. The patient was haemodynamically stable throughout his hospital stay, with good diuresis forced with mannitol during the first few hours, and then spontaneously. After 48 hours in the ICU he was transferred to the medical ward, where his hepatic function continued to improve, but he had polyuria and gradual deterioration of kidney function (reaching creatinine levels of 10.6mg/dl 13 days after admission). He was therefore indicated renal replacement therapy. Kidney biopsy was not performed given that he was considered a high-risk patient. Complementary examinations were also performed, with the following results: negative antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA), anti-DNA and anti-glomerular basement membrane antibodies. The serology tests for hepatitis B virus (HBV), hepatitis C (HCV), and human immunodeficiency virus (HIV) were negative. The C3 and C4 proteinogram and Ig assessment were within normal limits. Basic urine test: normal. Microalbuminuria: $36\mu g$/min. Negative Bence-Jones proteinuria.

Although the orellanus toxin is not common in our area,[5] the clinical symptoms described for delayed polyuric renal failure after wild mushroom consumption, with interstitial failure data, match with orellanus syndrome.[2,4] The patient was indicated continuous treatment with regular haemodialysis every 48 hours. No improvement in renal function was observed in the long term.

In summary, when a patient presents with clinical symptoms of liver and kidney involvement, mushroom poisoning must be considered, including orellanus syndrome, especially in regional areas with a tradition of wild mushroom picking.[3]

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Hepatotoxicity following cyclophosphamide treatment in a patient with MPO-ANCA vasculitis

Nefrologia 2011;31(4):496-8
doi:10.3265/Nefrologia.pre2011.May.10917

To the Editor,

Cyclophosphamide is a synthetic alkylating agent used in chemotherapy and as an immunosuppressive agent. Among its adverse effects are infections, myelosuppression, haemorrhagic cystitis, hypersensitivity reactions, digestive/hepatic, pulmonary, cardiac and neurological toxicity, sterility and syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH).[1-3]

We describe a patient with abdominal pain and an increase in hepatic and pancreatic enzymes after cyclophosphamide administration.

Male, 57-year-old patient, admitted for renal failure. Patient history: arthralgia and arthritis during the past 10 years,