repeated and showed no leak. During the following two months, the patient continued on APD until receiving a transplant.

The presence of dyspnoea compels us to rule out the most common pathology in our patients (hydrosaline retention, heart failure, etc.) but we must not forget about mechanical complications.[4]

The diagnosis will primarily be clinical, and on analysing the effusion we can test the properties of the dialysis liquid.

We stress the Tc-99 scintigraphy as a simple and safe technique for confirming a diagnosis.

It is carried out by means of a manual exchange with 2mCi Tc-99. The first reading is taken after 10 to 15 minutes, and after 3-4 hours delayed images are taken in different positions. Finally, all liquid is drained and destroyed according to nuclear medicine department protocol.


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## Treatment with thymoglobulin as the cause of acute demyelinating polyneuropathy in a renal transplant patient

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**Dear Editor:**
Guillain-Barré syndrome (GBS), or acute inflammatory demyelinating polyneuropathy (AIDP) is a severe pathology, often fulminating, which in more than two thirds of cases presents as an antecedent infection usually caused by a virus (cytomegalovirus [CMV] or Epstein-Barr virus). We present the case of a 48 year old male, recipient of a renal transplant in January 2007; D/R CMV serology positive; immunosuppression with steroids, mycophenolate and tacrolimus; delayed graft function during initial progress but good subsequent progress (creatinine on discharge, 1.5mg/dL).

During evolution, the patient presented with an acute Banff grade IIB cellular rejection, treated with thymoglobulin, which caused an allergic reaction, and bicytopenia. Treatment was withdrawn, but renal function improved. Four days after receiving thymoglobulin the patient presented with arthromyalgias and febricula, rapidly progressing to weakness of the lower extremities and 4/5 paresis in all four extremities, and severe dysphonia and dysphagia. He remained afebrile and without respiratory compromise. Laboratory tests showed a deterioration of renal function with 2.5mg/dL creatinine. Supplementary tests: cranial CT showed no alterations. Negative cultures. CRP for CMV negative. EMG: alterations compatible with acute motor demyelinating polyneuropathy. On assessment by Neurology, the patient was diagnosed as having symptoms compatible with GBS, starting treatment with polyclonal immunoglobulin IV at a dose of 2g/kg and high doses of steroids. The symptomatology disappeared 24 hours after treatment, although a slight motor deficit persisted for several weeks. Renal function on discharge: Cr: 1.7mg/dL

### Discussion

In an immunodepressed population, it is logical that the most frequently reported issue has been CMV2-4. Immunosuppression in itself constitutes an alteration of immunological equilibrium. This alteration, in keeping T suppressor lymphocytes inhibited, allows lymphocyte clones which are capable of generating an autoaggressive response to remain free. No habitual risk factor associated with the development of this entity was found in our patient. Circumstances exist in which abnormal circulating proteins have been associated with neuropathy (Waldenström's disease, multiple myeloma, POEMS syndrome). It is possible that the administration of thymoglobulin causes a condition similar to dysproteinemia, through the formation of immune complexes (serum sickness). T cells and neurons possess similar glycolipids in the membrane, with the associated chalcogens against GBS GM1. We have found only one reference[5] in the literature to a possible relationship with antilymphocyte polyclonal antibodies. In this instance, the temporal ratio and the symptoms displayed by the patient on administering the ATG, as well as an absence of other causes, cause us to think that there is a possible association between thymoglobulin treatment and subsequent AIDP, possibly related to serum sickness.


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## C) BRIEF CASE REPORTS

### Hypertensive urgency and stunting. What is the diagnosis?

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**Dear Editor:**
Tuberous sclerosis (TS) is a congenital anomaly of embryonic development, autosomal dominant inherited disorder, which has different forms of clinical expression. It is classified among the so-called phacomatoses: developmental anomalies susceptible to generating tumours or hamartomas of the nervous system. We present the case of a patient who was diagnosed by chance after a consultation in the Emergency Unit for another reason.

The case concerns a schoolgirl of 6 years and 6/12 months who attended the Emergency Unit due to an evanescent, occasionally petequial, rash which had been developing for several hours. Associated fever. Examination: weight 15.300kg (SD 6.3kg). Height 100cm (SD 16cm). Blood pressure: MID-160/120, MII-177/107, MSI- 169/118, MSD-169/110.

On physical examination the child showed no indication of severe illness. Afebrile. Dry skin, mucous membranes hydrated and somewhat pale. Dermal lesions associated with acute virosis. Rhythmic heart tones and strong II/VI systolic murmur heard in all positions. No findings in the abdomen: soft, depressible without pain, no organomegaly, sparse adipose tissue. Femoral pulse palpated. DTRs present. No meningeal signs. Hyperemic oropharynx.

Additional explorations: CBC: normochromic, normocytic anaemia. Leukocytosis, slight netrophilia, eosinophilia. Biochemistry: glucose 121mg/dL, urea: 135mg/dL, creatinine: 1.6mg/dL. Control: glucose: 81mg/dL, urea: 113mg/dL, creatinine: 1.1mg/dL, magnesium: 2.2, CRP: 215.8mg/l (N: 2-5). Control: 11.4, ABB: normal; serum iron: 64mg/dL (N: 45-156); lipid profile: normal; ESR: 4mm (N: 0-20); PTH: 75U/mcrl; infectious mononucleosis serology negative; pharyngeal culture: *Streptococcus pyogenes*; aldosterone: 50.10NG/dl, renin: 0.50ngml/h aldosterone/renin: 100.2 (<30). Coagulation test: normal. Urine: haematuria, mild proteinuria. Renal function test: glomerular filtration: 34ml/min/1.73m$^2$; calciuria: 3mg/kg/day; phosphaturia: 29mg/kg/day (N = 15-20); natriuria: 3.44mEq/kg/day (3.87 ± 1.3mEq/kg/day); kaliuria: 2.24 mEq/kg/day (1.73 ± 0.7mEq/kg/day). RTP: 64%; urine culture: negative; microalbunin/creatinine index: 3. 222mg/g; folic acid, vitamin B12 and eye depth: papillary pallor. No oedema of the papilla, clear edges. No other abnormalities were found; ECG: left ventricular hypertrophy. Echocardiogram: normal; left hand and wrist x-ray: osseous age of five years; immunoglobulins, C3 and C4 ANA and AMA normal; catecholamine: normal.

Renal abdominal ultrasound (Doppler): kidneys were within normal size range but with structural alterations. Focal lesions of bilateral renal parenchyma were present.

MRI of the abdomen, cranium and pelvis: angiomyolipomas with a sparse or almost undetectable quantity of intratumoral fatty tissue. Cerebral images compatible with small hamartomata in the white matter. Both suggestive of tuberous sclerosis. Genetic tests on TSC1 and TSC2 were both negative.

Evolution: improvement in blood pressure after starting treatment with ACEI and ARA II, being normal for the patient's age and size after use and with negative proteinuria.

Angiomyolipoma (AML) is the most common renal lesion in TS (34-80%), followed by renal cysts and polycystic disease. This is due to the fact that the TSC2 locus is adjacent to one of the polycystic kidney disease genes (PKD1) and adjacent deletions can produce both phenotypes.

Angiomyolipoma is a benign tumour of the renal cortex, characterised by the presence of mature or immature fatty tissue, vascular wall and smooth muscle though with the capacity to provoke severe haemorrhage, replacement of renal parenchyma and mass effect, which can induce pain and may compromise renal function.[1] Renal failure is less frequent, and is generally associated with glomerulosclerosis secondary to hyperfiltration as a result of surgery or tumoral invasion, particularly by cysts.[2] Some patient groups have indicated that there are variants of angiomyolipoma which have the capacity for metastatic growth.[3]

Despite being benign there is a possibility of malignant transformation.[4] Furthermore, the tumour can occasionally relapse in