1. Pendse S, Singh A, Zawada E. Initiation of Dialysis. Handbook of Dialysis 4th edition, 2007, pp. 16-17.
2. Hulke MH, Mayer RJ. Carcinoid tumors. *N Engl J Med* 1999; 340 (11): 858-868.
3. Maroun J, Kocha W, Kvols L, Bjarnason G, Chen E, Germond C et al. Guidelines for the diagnosis and management of carcinoid tumors. Part 1. *Curr Oncol* 2006; 13 (2): 67-76.
4. Venizelos ID, Tamiolakis D, Lambropoulou M, Constantinidis T, Petrakis G, Alexiadis G et al. Primary combined carcinoid and adenocarcinoma of the ileum associated with transitional carcinoma of the bladder. Single case report. *Rev Esp Enferm Dig* 2007; 99 (3): 145-148.
5. Shurtleff BT, Shvarts O, Rajfer J. Carcinoid tumor of the kidney: case report and review of the literature. *Rev Urol* 2005; 7 (4): 229-233.
6. Levine RL, George DJ, Kulke MH. Incidental discovery of an appendiceal carcinoid tumor and a renal cell carcinoma. *Clin Adv Hematol Oncol* 2003; 1 (2): 99-100.

M. Picazo Sánchez, M. Cuxart Pérez and R. Sans Lorman
Servicio de Nefrología. Hospital de Figueres
Correspondence: *Montserrat Picazo Sánchez. montserratpicazo@ yahoo.es. Hospital de Figueres. Rda. Rector Arolas. 17600 Figueres. Girona. España.*

IgA nephropathy and lupus anticoagulant: an incidental association?

Nefrología 2008; 28 **(4)** 466-467

To the editor: IgA nephropathy is the most common chronic primary glomerulonephritis, leading to progressive renal failure in at least one third of patients.[1] No association of this disease with the lupus anticoagulant (LA) has been found in the literature. The occurrence of IgA nephropathy in two sisters, one of whom had LA, is reported.

CASE 1

A 26-year-old female patient with a history of acne and recurrent urinary tract infection. She was studied for gross hematuria after upper respiratory tract infections. Physical examination was normal. Laboratory test results included: creatinine 1.4 mg/dL, hematocrit 25%, prothrombin activity 87%, APTT 44.7 seconds , fibrinogen 434 mg/dL. Immunological test results included: IgG 1260 mg/dL (N: 751-1560), IgA 667 mg/dL (N:82-453), IgM 190 mg/dL (N: 46-304), C3 88 mg/dl (N: 79-152), C4 27.8 mg/dl (N: 16-38), negative ANA. Urine sediment showed 6-10 RBCs/field. Proteinuria in a 24-hour sample was 3 g/day. A renal pathology study confirmed the existence of IgA nephropathy. As the patient had a prolonged cephalin time, a hypercoagulability study was performed, showing a positive result for IgM anticardiolipin antibodies, 50.7 MPL/mL (N < 11).

CASE 2

A 29-year-old female patient with personal and clinical history similar to her sister (case 1). Physical examination was normal. Results of laboratory blood tests included: Creatinine 1 mg/dL. Normal complete blood count. Coagulation: Prothrombin activity 84%, APTT 39.5 sec. Negative LA and anticardiolipin antibodies. Immunological tests showed the following values: IgG 1470 mg/dL, IgA 388 mg/dL, IgM 417 mg/dL, C3 100 mg/dL, C4 28.6 mg/dL. ASLO, ANA, and anti-DNA antibodies were negative. Urine sediment showed more than 40 RBCs/field. No protein was found in a 24-hour urine sample. Renal biopsy confirmed the existence of nephropathy with IgA mesangial deposits.

DISCUSSION

IgA nephropathy is characterized by IgA mesangial deposits associated to a proliferative mesangial glomerulonephritis. Its pathogenesis is not fully known, but there is increasing evidence of the formation of immune complexes containing IgA that would be deposited in mesangium and would induce glomerular damage.[2]

The primary antiphospholipid syndrome (PAPS) is characterized by recurrent thrombosis, multiorgan damage, and abortion, as well as the presence of LA and/or anticardiolipin antibodies.

LAs are antibodies directed against plasma proteins such as b2-glycoprotein I, prothrombin, or annexin V.[3,4] In case 1, a basic coagulation study detected a prolonged cephalin time, and a subsequent hypercoagulability study was positive for LA anticardiolipin antibodies, but there were no associated clinical signs consistent with PAPS.

Association of LA and anticardiolipin antibodies with various autoimmune and rheumatic diseases (SLE, scleroderma, psoriatic arthritis…) has been reported in the literature,[5] but there are no reports of an association with IgA nephropathy, except in cases having, in addition to LA, the clinical signs of PAPS.[6] It is also possible that this patient initially had or may have subsequently developed a Schönlein-Henoch glomerulonephritis or a lupus nephropathy. These are both conditions with mesangial IgA deposits that pathologists are sometimes unable to differentiate from pure mesangial glomerulonephritis.

In the reported cases, the finding of LA alone, without other signs of PAPS, in only one of the sisters (with the same nephropathy and the same clinical sign of hematuria following upper respiratory tract infections) raises the question whether this association was incidental or its occurrence was related to a common immune pathogenetic mechanism.

1. Galla JH. Ig A nephropathy. *Kidney Int* 1995; 47 (2): 377-387.
2. Barratt J, Smith AC, Molyneux K, Feehally J. Immunopathogenesis of Ig AN. *Semin Immunopathol* 2007; 29 (4): 427-443,.
3. Roubey RA. Autoantibodies to phospholipid-binding plasma proteins: a new wiew of lupus anticoagulants and other «antiphospholipid» autoantibodies. *Blood* 1994; 84 (9): 2854-2867.
4. Passam F, Krilis S. Laboratory tests for the antiphospholipid syndrome: current concepts. *Pathology* 2004; 36 (2): 129-138.
5. Cervera R, Asherson RA. Clinical and epidemiological aspects in the antiphospholipid syndrome. *Immunobiology* 2003; 207 (1): 5-11.
6. Sinniah R, Gan HC, Yoon KH. Primary antiphospholipid antibody syndrome and mesangial IgA glomerulonephritis. *Am J Nephrol* 2001; 21 (2): 134-140.

M. Heras Benito, R. Fisac Herrero*, M.ª J. Fernández-Reyes Luis and R. Sánchez Hernández
*Servicio de Nefrología y *Hematología. Hospital General de Segovia.*
Correspondence: *Manuel Heras Benito. mheras@hgse.sacyl.es. Hospital General de Segovia. Ctra. de Ávila, s/n. 40002 Segovia. España.*

Iatrogenic thyroid dysfunction in peritoneal dialysis

Nefrología 2008; 28 **(4)** 467

To the editor: The list of drugs that may cause changes in thyroid hormone levels would be endless (amiodarone, metformin, dopamine, dobutamine, propranolol, carbamazepine, lithium, glucocorticoids….).[1,2] However, radiographic contrast agents[3] and iodine-containing solutions used as general antiseptics and broad-spectrum disinfectants, such as povidone iodine, may also cause thyroid dysfunction.[4] Thus, it is known that povidone iodine contained in the disconnect caps of peritoneal dialysis may be a factor contributing to changes in thyroid function.. The patient population with a higher risk is however limited to infants and children on peritoneal dialysis with small filling volumes, where iodine concentration in the dialysis fluid is higher, while thyroid function changes are considered uncommon in the adult population.[5]

The case of an elderly female patient who showed changes in TSH levels probably induced by the iodine contained in the peritoneal dialysis cap is reported.

This 70-year-old patient had been diagnosed CKD secondary to renal amyloidosis in the setting of a familial amyloidotic polyneuropathy. She also had cardiac amyloid infiltration, and had been implanted a pacemaker in 2005. In addition, the patient had chronic diarrhea due to intestinal infiltration.

Proteinuria was initially found in January 2004, and progressed to levels in the nephrotic range. The patient showed a progressive renal function impairment since April 2005, and was implanted a peritoneal dialysis catheter in February 2006.

Continuous ambulatory peritoneal dialysis was started on 12-04-06, but a catheter leak occurred and a switch to intermittent nocturnal peritoneal dialysis with cycler and low volume (1200 mL per cycle) was made on 28-04-06. The leak subsequently resolved.

The patient had not previously shown any change in thyroid function, and normal TSH levels were found before the start of dialysis. Low, sometimes undetectable TSH levels were seen after the low volume dialysis technique was started. T3 and T4 levels were within the normal range, and anti-thyroid antibodies were normal. The endocrinology department was consulted, and a thyroid ultrasound was performed, showing a diffuse thyroid enlargement that was related to the underlying disease. Fine needle puncture allowed for ruling out malignancy or an amyloid infiltrate. While uncommonly, infiltrative diseases such as amyloidosis may also cause thyroid dysfunction.[6]

The patient was asymptomatic at all times and did not require additional treatment. Once the catheter leak was resolved, filling volume could be increased, but continues to be low (1500 mL), now because of the discomfort experienced by the patient with higher volumes. Hormonal changes persist.

Similar to when treatment is started with drugs altering thyroid function, thyroid hormone monitoring is also recommended in patient on peritoneal dialysis with small filling volumes, because the iodine contained in the disconnect cap may reach high concentrations in peritoneal fluid and pass into the blood, inducing iatrogenic changes such as those occurring in the rare case reported.

1. Vigersky RA, Filmore-Nassar A, Glass AR. Thyrotropin suppression by metformin. *J Clin Endocrinol Metab* 2006; 91: 225; 2006.
2. Figge HL, Figge J. The effects of amiodarone on thyroid hormone function: a review of the physiology and clinical manifestations. *J Clin Pharmacol* 1990; 30: 588.
3. Martin FI, Tress BW, Colman PG, Deam DR. Iodine-induced hyperthyroidism due to nonionic contrast radiography in the elderly. *Am J Med* 1993; 95: 78.
4. Lim VS, Thyroid function in patients with chronic renal failure. *Am J Kidney Dis* 2001; 38 (4): 80-4.
5. Brough R, Jones C. Iatrogenic iodine as a cause of hypothyroididm in infants with end-stage renal failure. *Pediatr Nephrol* 2006; 21 (3): 400-2.
6. Kimura H, Yamashita S, Ashizawa K et al. Thyroid dysfunction in patients with amyloid goiter. *Clin Endocrinol* 1997; 46: 769.

A. Sastre López and M.ª V. Mascarós Ferrer
Servicio de Nefrología. Hospital Son Llàtzer. Palma de Mallorca.
Correspondence: *Aránzazu Sastre López. aranchasastre@hotmail.com. HUCA. Avda. Fernández Ladreda, 30. 24005 León. España.*

Primary antiphospholipid syndrome: dormant, not cured

Nefrología 2008; 28 **(4)** 467-469

To the editor: A case that may be of clear practical help for healthcare professionals treating similar patients because of some characteristics of its clinical course, response to treatment, and potential confusion for responsible physicians is reported below.

Case report: A 24-year-old female patient who experienced generalized tonic-clonic seizures when she was 20. Physical examination revealed postictal disorientation, mild Raynaud's phenomenon, and malar erythema. She had severe autoimmune hemolytic anemia due to complement-fixing hot IgG antibodies and thrombocytopenia; renal failure with serum Cr levels of 2.2 mg/dL and dysmorphic RBCs; hypocomplementemia (C3 80 mg/dL, C4 8 mg/dL), ANA 1/640, IgG anticardiolipin 57.7 UGP/mL (negative < 10), IgM anticardiolipin 58.2 UGP/mL, lupus anticoagulant DVV test 131 (negative < 45). MRI of the brain showed cortical-subcortical vasculitic-ischemic