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Iatrogenic thyroid dysfunction in peritoneal dialysis

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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.

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Primary antiphospholipid syndrome: dormant, not cured

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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