In these two cases, intensified ultrafiltration caused a prompt decrease in BP. In three subjects, hypotension occurred after BN (BP <120/70 mmHg), and intradialytic ultrafiltration had to be decreased. Five patients required continuation of one antihypertensive drug. While in our centre, antihypertensive therapy was more intense during the last years of the study with 5.8 (5–6) daily antihypertensive agents compared with only 4.4 (4–5) in the years 1984–89 (NS).

The clinical condition and quality of life improved in all of our patients. In nine patients, antihypertensive treatment was no longer necessary, and five patients required continuation of only one antihypertensive agent. Previous studies showed similar significant improvements in hypertension after BN [2], although a case report described persistent hypotension after BN in a 4-month-old infant [3]. According to the literature, BN is infrequently used as treatment for intractable hypertension. The prevalence of BN varies between 0% and 7% in most countries [4]. In our centre, refractory hypertension has become a rare indication for BN, and the incidence of BN was continuously decreasing during our observation period. For example, the incidence rate (*n*/year) decreased from 1.2 during the first five years (1984–89), to 1.0 and 0.4 during the next two 5-year periods, and was only 0.2 during 2003–08 ($P < 0.05$). The reduced frequency of BN was explained by the use of more effective antihypertensive medication during the last years; however, this difference was not significant because of small patient group sizes. Reductions in BN were also due to more frequent treatments with ACE inhibitors/AII blockers (9% in the first five years versus 33% during the last five years).

In conclusion, we found that BN effectively lowered BP in cases that were extremely refractory to hypertension therapy. Despite this, the frequency of needed BN in haemodialysis patients appears to be decreasing. The lower frequency of BN may be associated with recent intensified antihypertensive therapy and a much greater use of ACE inhibitors during the last decade.

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Isolated *Aspergillus* thyroiditis in an immunocompromised patient

Sir,

We describe a 30-year-old woman who presented with a progressively enlarging anterior neck mass with pain, local warmth and skin erythema. The patient had been on haemodialysis because of a 3-month history of renal failure due to lupus nephritis. She was also on monthly cyclophosphamide pulse therapy and daily prednisolone (30 mg daily). Her vital signs were as follows: temperature, 37.6°C; blood pressure, 130/80 mmHg; pulse rate, 95–105/min; and respiratory rate, 20/min. Her laboratory results, including thyroid studies, are shown in Table 1. A thyroid scan revealed large defects bilaterally. Initially, subacute thyroiditis was considered the most probable diagnosis, and the daily dose of oral prednisolone was increased to 60 mg. The patient complained of nervousness, irritability, hyperactivity and palpitation, and a β-adrenergic blocker and propylthiouracil were prescribed. When the patient revisited our clinic 3 weeks later, she complained of a progressively enlarging thyroid and respiratory distress. Her chest X-ray revealed tracheal compression and deviation. Because of respiratory failure, she required endotracheal intubation and mechanical ventilation followed by emergency total thyroidectomy.

Microscopy of the thyroid revealed infectious thyroiditis with suppurative inflammation, abscess formation, and considerable tissue destruction throughout the gland. Septate hyphae, <5 μm in thickness, with branching at acute angles

Table 1. Results of the patient's laboratory tests

Test	Normal range (unit)	Result
White blood cell count	50 000–10 000 (/mm^3)	4440
Neutrophils	55–75 (%)	93.4
Lymphocytes	20–44 (%)	3.3
Monocytes	2–8 (%)	3.0
Haemoglobin	12–16.0 (g/dL)	10.0
Haematocrit	37.0–47.0 (%)	28.6
Platelet count	150 000–450 000 (/mm^3)	103 000
Erythrocyte sedimentation rate	0–20 (mm/h)	65
C-reactive protein	0.1–5.0 (mg/L)	265.04
Total triiodothyronine (T3)	0.8–2.0 (ng/mL)	0.94
Total thyroxine (T4)	4.5–12.0 (μg/dL)	16.24
Free T4	0.78–1.94 (ng/dL)	4.05
Thyroid-stimulating hormone (TSH)	0.3–4.0 (mIU/L)	0.02
TSH receptor antibody	0.0–9.0 (U/L)	0.73
Anti-thyroglobulin antibody	0.0–70.0 (IU/dL)	2.47
Anti-thyroid peroxidase antibody	0.0–100.0 (IU/dL)	6.67

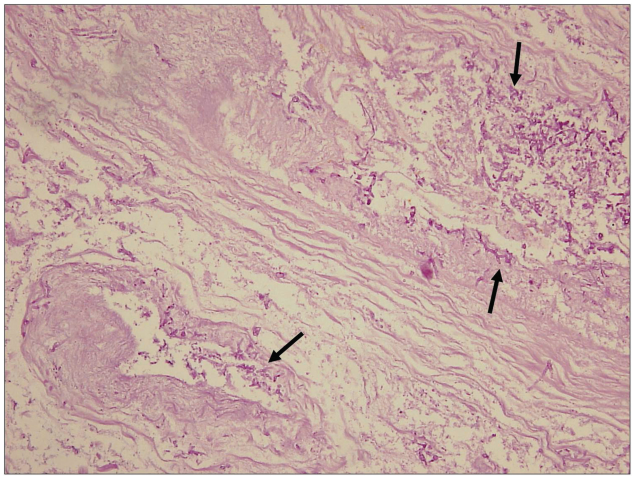

Fig. 1. Periodic acid–Schiff stain demonstrates numerous fungal profiles (arrows) in the thyroid stroma with angio-invasion (original magnification, ×200).

were identified. These findings were consistent with a fungal thyroiditis caused by *Aspergillus*. Blood vessels were invaded by the hyphae of the fungus (Figure 1). A culture of the aspirated fluid showed no growth. The results of repeated tests with an *Aspergillus* antigen (galactomannan) enzyme-linked immunosorbent assay (ELISA) were negative. There was no evidence of aspergillosis in the other organs. Blood and sputum cultures were negative. Intravenous liposomal amphotericin B (5 mg/kg daily) was initiated, and later switched to oral voriconazole (200 mg twice a day for 2 days, and then 100 mg twice a day) on the day of discharge, 2 weeks after the initiation of amphotericin B, and was continued for the following 12 weeks. Because tests showed a decreasing thyroid function, hormone replacement was begun 10 days after surgery.

Discussion

Isolated fungal infections of the thyroid are uncommon because of its rich vascular and lymphatic supply, well-developed capsule, and high iodine content [1]. Although involvement of the thyroid gland has been detected at autopsy in 9–15% of patients with disseminated fungal disease, there are few reports of isolated infections of the gland without signs of disseminated disease in a living patient [1]. Biopsy, direct microscopy and culture of fine-needle aspirate are still essential for obtaining a diagnosis because systemic antigenaemia (as measured by galactomannan screening) may not develop in patients with localized cases [2].

It is clear that the introduction of the serum *Aspergillus* galactomannan antigen detection test has made earlier diagnosis possible in high-risk patients. Nonetheless, a major problem with the galactomannan test is that its sensitivity varies greatly, reportedly ranging from 30% to 100%, and its specificity 38% to 98% [3]. Such wide variation in the levels of circulating galactomannan may be attributable to the administration of antifungal drugs, a low frequency of sampling, or a low fungal burden [3,4]. One clinical study suggests that the lesser sensitivity of galactomannan in some patients, especially patients with airway-invasive aspergillosis, might be a result of the minor intravascular fungal burden in these patients [4]. Hence, the cut-off value in the galactomannan assay may need to be set differently for different risk populations [5]. Another possibility is that *Aspergillus* could be secreting a galactomannan antigen with only one galactofuranose epitope not detected by some ELISA methods. Finally, already formed antibody to *Aspergillus* might influence the result of the galactomannan test [6].

In conclusion, we report a rare case describing the successful treatment of serum galactomannan-negative isolated *Aspergillus* thyroiditis in a haemodialysis patient with end-stage renal disease due to lupus nephritis.

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A rare presentation of pulmonary hemorrhage with hepatitis C-associated cryoglobulinemia and membranoproliferative glomerulonephritis

Cryoglobulinemia (CG)-associated pulmonary hemorrhage is an unusual entity. An even rarer occurrence is that of membranoproliferative glomerulonephritis (MPGN) in a patient with CG and alveolar hemorrhage. As far as we can tell, there has been no more than a total of four such