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Thyrotoxicosis factitia induces Takotsubo cardiomyopathy in end-stage renal disease: a pathogenetic hypothesis

To the Editor: We appreciate the report by Takemoto *et al.*[1] on Takotsubo cardiomyopathy without an identifiable cause in a patient undergoing long-term hemodialysis, which reflects the great diversity of cardiovascular events in end-stage renal disease patients. Although the etiology of Takotsubo cardiomyopathy remains unclear in most cases, we would like to bring to the readers' attention an unusual cause of this stress cardiomyopathy. A 31-year-old woman with Chinese herb nephropathy and on continuous ambulatory peritoneal dialysis for 16 months developed severe chest pain rapidly after taking slimming pills containing triiodothyronine (128 μg). Transient left ventricular apical ballooning without evidence of coronary artery abnormality was documented. In this patient, a burst of enhanced, excessive sympathetic activity arising from thyrotoxicosis factitia may have triggered a switch in intracellular signal trafficking in ventricular cardiomyocytes, from G_s protein to G_i protein signaling via the β_2-adrenergic receptors.[2] This strategy counteracts the proapoptotic effect of excessive β_1-adrenergic G_s protein pathway activation and seems to act as a physiological balance to prevent catecholamine-mediated myocardial damage.[3] In our patient, the ventricular function and apical wall motion returned to normal 2 weeks later.

1. Takemoto F, Chihara N, Sawa N *et al.* Takotsubo cardiomyopathy in a patient undergoing hemodialysis. *Kidney Int* 2009; **76**: 467.
2. Daaka Y, Luttrell LM, Lefkowitz RJ. Switching of the coupling of the β_2-adrenergic receptor to different G proteins by protein kinase A. *Nature* 1997; **390**: 88–91.
3. Chesley A, Lundberg MS, Asai T *et al.* The β_2-adrenergic receptor delivers an antiapoptotic signal to cardiac myocytes through G_i-dependent coupling to phosphatidylinositol 3′-kinase. *Circ Res* 2000; **87**: 1172–1179.

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Antineutrophil cytoplasmic autoantibody-associated vasculitis on chronic dialysis

To the Editor: In a recent issue of *Kidney International*, Lionaki *et al.*[1] suggested that in antineutrophil cytoplasmic autoantibody-associated vasculitis patients on dialysis, immunosuppressants should be restricted to those with active vasculitis. We quite agree with it, but some data are not convincing enough to make such a conclusion. First, the authors reported that in end-stage renal disease (ESRD) patients, the infection rate was twice as high in patients who were on immunosuppressants as in those who were not on immunosuppressants. It is not a pertinent reason for restricting immunosuppressants in ESRD patients, as immunosuppressants would definitely increase the chances of infection, regardless of ESRD or non-ESRD.[2] Instead, the authors should compare the frequency of infections between ESRD and non-ESRD patients, both on immunosuppressants, or demonstrate that in non-ESRD patients, the possibility of infections being increased by immunosuppressants is much less than twice. Moreover, other factors contributing to infections should be adjusted. Second, the authors reported that among ESRD patients, death rates were similar between those with and those without active vasculitis. It is improper to simply compare the 'percentages' without considering follow-up durations. Instead, Cox-regression or, at least, Kaplan–Meier analysis, should be used. Third, the authors reported that infections and active vasculitis were the first cause of death in ESRD and non-ESRD patients, respectively. Such comparison is an unbalance, as the former would mostly have already received immunosuppressants and would thus be in remission, while the latter are often in an active state. Instead, the authors should compare the cause of death between ESRD and non-ESRD patients, both in active and remission states. In addition, the unavailability of the causes of death in many patients makes such comparison unconvincing.

1. Lionaki S, Hogan SL, Jennette CE *et al.* The clinical course of ANCA small-vessel vasculitis on chronic dialysis. *Kidney Int* 2009; **76**: 644–651.
2. Chen M, Yu F, Zhang Y *et al.* Antineutrophil cytoplasmic autoantibody-associated vasculitis in older patients. *Medicine (Baltimore)* 2008; **87**: 203–209.

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The Authors Reply: We appreciate the opportunity to respond to Chen and Zhao[1] regarding our analysis of the clinical course of antineutrophil cytoplasmic autoantibody small vessel vasculitis[2] on chronic dialysis. Drs Chen and Zhao raise the concern that our results do not support the statement that immunosuppressive therapy should be geared only to patients with end-stage renal disease (ESRD) and