Renal amyloidosis, which has been observed in 14% of patients with TRAPS (3), is the most important complication of this disease. To our knowledge, glomerulonephritis in TRAPS has not been reported so far. We hypothesize that there was a causal relationship between TRAPS and the crescentic glomerulonephritis in our patient, because it occurred during an acute episode, and no other cause of nephritis was found. Our observation is consistent with previous works that demonstrated an increased production of TNFα in human glomerulonephritis by activated infiltrating mononuclear cells and intrinsic renal cells and effectiveness of TNF blockade in experimental models of crescentic glomerulonephritis (4,5). Moreover, Lamprecht et al reported the case of a patient with small-vessel vasculitis and TRAPS (6). Because impaired TNFα regulation has been implicated in the pathogenesis of systemic vasculitis (7) as well as in glomerular inflammation, we speculate that proliferative glomerulonephritis can be a feature of TRAPS. Our report suggests that clinicians should consider glomerulonephritis as part of the growing range of TRAPS manifestations.