Dear Sir,

We read with interest the paper from Stratta et al. [1] reporting a case of proliferative glomerulonephritis with linear glomerular IgG deposits after inhalation of paraquat. The authors state that this is the first case report of such an association. They further suggest that glomerulonephritis has been mediated by antiglomerular basement membrane (anti-GBM) antibodies, despite the absence of circulating anti-GBM antibodies 5 months after the exposure.

We actually reported, 5 years ago, a strikingly similar case, in whom the existence of anti-GBM antibodies was clearly demonstrated [2]. The renal damage was far more severe in our patient than in that of Stratta et al. Of additional interest is the course of our patient during the subsequent 5 years after our report.

Our patient, also a farmer, had been intensely and repeatedly exposed to various insecticides and herbicides including paraquat while spraying his fields during the month preceding admission. He presented with a 2-week history of malaise, headache, diarrhea and epistaxis and a 2-day history of fever and dark urine. Oligoanuric rapidly progressive glomerulonephritis developed, requiring institution of hemodialysis 9 days after admission. Percutaneous renal biopsy disclosed severe necrotizing crescentic glomerulonephritis; linear deposits of IgG and C3 were present along the GBM, and circulating anti-GBM antibodies were detected by radioimmunoassay (Dr. P. Mahieu, Liège). Renal failure was irreversible. Anti-GBM antibody was still present in the serum 13 months after onset of the disease but was no longer detected 6 months later and repeatedly thereafter up to now, 7.5 years after exposure. Six and a half years after the onset of the disease, the patient received a cadaver kidney. In the nephrectomy sample obtained at that time, linear glomerular IgG deposits were still visible. No rejection occurred. Currently, 1 year after transplantation, this 57-year-old man is fully rehabilitated and has a well-functioning graft (serum creatinine 1.2 mg/dl; normal urinalysis).

The new case reported by Stratta et al. [1] supports our suggestion that the inhalation of some chemical compounds used as insecticides and herbicides, such as paraquat, may induce glomerulonephritis through anti-GBM immunization. Such an association had previously been observed after inhalation of hydrocarbon solvents [3]. Just as in other forms of oligoanuric anti-GBM nephritis, anti-GBM antibodies spontaneously disappear from the circulation usually within 2 years [4], allowing the patient with irreversible terminal renal failure to be safely transplanted.
Factors able to induce the anti-GBM antibody response are increasingly recognized: exposure to toxic compounds such as paraquat and hydrocarbon solvents, or to drugs such as penicillamine [5] should be looked for systematically. Recognition of inciting agents may have therapeutic as well as physiopathologic implications.

References