Dear Sir,

We report a uremic patient with simultaneous and acute-onset bilateral external ophthalmoplegia. Our purpose is to document this unusual case and to call attention if there is any other similar observations. This 21-year-old woman was admitted with the complaints of double vision and droopy eyelids for 2 days. She has been followed with the diagnosis of chronic pyelonephritis for the past 6 years, and she had never required a dialysis. Her physical and neurological examination was normal. On follow-up she became drowsy but was easily arousable and gave reasonable answers to questions. Her examination revealed bilateral ptosis and nearly complete ophthalmoplegia (fig.1). Vision and pupils were normal. Her laboratory findings were unrevealing except high levels of blood urea nitrogen (BUN) and creatinine. Fasting blood sugar 4.0 mmol/l (72 mg/dl), hemoglobin 62 g/l (6.2 g/dl), BUN 59.3 mmol/l (166 mg/dl), creatinine 1,706.1 µmol/l (19.3 mg/dl), Na 136 mmol/l (136 mEq/l), K 3.8 mmol/l (3.8 mEq/l), VDRL negative. Cerebrospinal fluid (CSF) findings were: protein 0.32 g/l (32 mg/dl), glucose 3.1 mmol/l (57 mg/dl, Cl 119 mmol/l (119 mEq/l), VDRL negative. There were no cells in the cytologic examination of the CSF. Intravenous edrophonium test was negative. Cranial computerized tomography with intravenous contrast enhancement was normal. On discharge her BUN and creatinine levels were decreased: BUN 24.6 mmol/l (69 mg/dl), creatinine 636 µmol/l (7.2 mg/dl), and hemoglobin level was increased: 101 g/l (10.1 g/dl). Following admission peritoneal dialysis was initiated. The edema in the lungs and external ophthalmoplegia began to resolve, and on the third day of peritoneal dialysis her ophthalmoplegia was completely resolved. Hemodialysis therapy was then started. Two weeks later her examination was normal.

The exact cause and specific localization of the lesion was not determined in our patient. It is possible that either brainstem oculomotor nuclei or peripheric parts of oculomotor cranial nerves are affected simultaneously. Ophthalmoplegia and Wernicke’s encephalopathy has been reported in uremic patients due to thiamine deficiency induced by a genetic defect in transketolase activity, anorexia, vomiting, intravenous alimentations, and glucose load or infections, only during chronic dialysis treatment [1, 2]. Our patient’s mental status was not disturbed, her nutritional status was good and she did not experience vomiting or diarrhea. Improvement in her neuro-ophthalmological findings after the dialysis is not compatible with the other reported cases of Wernicke’s encephalopathy in the literature.
Fisher’s syndrome occurring after dialysis has also been reported with no clear explanation of its occurrence [3]. We exclude this diagnosis because of normoactive deep tendon reflexes, normal CSF protein and improvement with the dialysis. The nervous system complications in patients with uremia are thought to be caused by many factors [4,5]. In the pathogenesis of uremic neuropathies urea, creatinine, parathyroid hormone, myoinositol, transketolase, guanidine derivatives and middle molecules are the possible uremic toxins [1]. The fact that her ophthalmoplegia responded quickly to institution of peritoneal dialysis and that there was no other cause to explain her ophthalmoplegia, we think that her bilateral simultaneous external ophthalmoplegia was due to uremic toxins.

References
Fig. 1. Limited eye movements to all directions and bilateral ptosis.

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