Dihydroergocristine-Induced Retroperitoneal Fibrosis with an Episode of Reversible Obstructive Acute Renal Failure

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Dear Sir,

Retroperitoneal fibrosis (RF) can be caused by various pathogenetic mechanisms: radiation, trauma, retroperitoneal malignancies, inflammatory bowel diseases, atherosclerosis of abdominal vessels, and drugs [1, 2]. The majority of cases are, however, still classified as idiopathic forms of RF [3].

Since the report by Graham et al. [4] in 1966, several drugs have been proposed to cause RF: beta blockers [5], amphetamines [6], and ergot alkaloids and related compounds such as methysergide, a diethylamide of lysergic acid [7]. Up to now no report has been presented on dihydroergocristine (DHE), the ergopeptidic group of which is characteristic of ergot alkaloids. We describe the clinical course of RF in a DHE-treated patient with special regard to the reversal of acute renal failure and regression of anatomical lesions.

A 61-year-old man was admitted with acute-onset anuria without any accompanying symptoms. During the last 2 years he had been treated daily with 6 mg of DHE for ageing-related brain disease. The plasma creatinine level was 11 mg/dl (972 µmol/l). Renal ultrasonography showed bilateral hydroureteronephrosis without abdominal or urological masses. A single dose of methylprednisolone (80 mg i.v.) was given, resulting in immediate relief from the anuria with post obstructive polyuria (> 5 liters

Fig. 1. Computerized tomogram at presentation (a) and 1 year later (b): The fibrotic plaque extension is significantly reduced after drug withdrawal.

during the following 24 h). The plasma creatinine concentration returned to normal within 3 days.

Computerized tomography (fig. 1a) revealed the presence of RF with plaque of

both ureters, extending downward from the renal vessels to the inferior renal pole and involving the aorta to a length of 6-7 cm, while urography disclosed the characteristic medialization of both ureters (fig. 2a).

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Fig. 2. Urogram showing the characteristic medialization of both ureters (a), followed by partial regression 1 year later (b).

The patient was discharged 11 days after admission with a plasma creatinine concentration of 1.2 mg/dl (106 μ mol/l), normal diuresis, and with the advice to suspend DHE based on the hypothesis of drug-related RE

One year later, computerized tomography revealed a marked reduction in the extension of the fibrotic plaque (fig. 1b), and the urogram disclosed an almost normal appearance of the ureters (fig. 2b). The reduction in fibrotic plaque can be ascribed only to DHE withdrawal.

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