INTRODUCTION

When Stephen Hales first measured the arterial blood pressure of a horse in 1733, he was certainly not aware of the fact that blood pressure in humans can be elevated and that in the future this would become a major public health issue (1). Today, arterial hypertension (AH) is the main public health problem and leading factor in global morbidity. It is the main risk factor for heart, brain, and kidney disease. Chronic kidney disease is a complication arising from uncontrolled AH but is also frequently its cause. The interaction between AH and chronic kidney disease is complicated and increases the risk of adverse cardiovascular and cerebrovascular outcomes (2, 3). In 2015, standardized prevalence of hypertension in adults aged 18 and above was 22.1% (20.3% in women and 24.1% in men) (4). Among those, only 37% of men and 51% of women with AH knew they had AH. Among diagnosed hypertensives only 33% of women and 19% of men underwent antihypertensive treatment, and only 12% of women and 6% of men with AH had blood pressure values under control (5). The incidence of cardiovascular diseases is significantly higher in patients with chronic kidney disease as opposed to the general population, and cardiovascular diseases have become the leading cause of death in patients with chronic kidney disease (6-8). The likelihood of adverse cardiovascular events increases with the progression of renal insufficiency, resulting in a 40-50 times higher risk of cardiovascular diseases in patients on haemodialysis compared to the general population (9). Here we present a case with arterial hypertension and renal insufficiency of unexpected aetiology.

CASE REPORT

A 45-year-old man arrived at the emergency room (ER) due to nausea, stomach-ache lasting 2 days and dull pain in the lumbar spine region. He had no prior serious illness besides occasionally elevated blood pressure levels for the last 5 years, however without treatment. He had an increased body mass with a body mass index of 28.3 kg/m². Family history of both parents was positive for arterial hypertension (AH). Workup in the ER identified uncontrolled AH with blood pressure value of 250/150 mmHg and unremarkable status aside from mild pain in the epigastric region during stomach palpation. Furthermore, renal insufficiency of unknown duration with a creatinine level of 484 μmol/l (eGFR CKD EPI 12 ml/min/1.73 m²) and dipstick urine analysis positive for blood and 3+ proteins were detected. Additional findings included mild microalbuminuria.
Although rare, this condition should be a complication of AH which led to the most severe, and fortunately rarest examination, most likely due to adequate periprocedural hydration and temporary cessation of interfering medications such as diuretics. In our patient we hesitated to use intravenous contrast diagnostics due to risk of CIN and attempted non-contrast methods to evaluate primarily the renal arteries (Doppler ultrasound), however this method was limited by the patient’s constitution and methodism, and this is also the reason why abdominal aorta was not visualized. Additionally, the native MSCT examination is also not a diagnostic method for either aortic dissection or stenosis/dissection of the renal arteries, especially while the dissected aorta is still of normal calibre. When intravenous contrast examination was finally carried out, the definitive diagnosis and etiology of kidney damage and cause of the worsening of the arterial hypertension of the European Society of Cardiology (ESC) and the European Society of Hypertension (ESH), Eur Heart J. 2004; 25 (2): 119-33. doi: https://doi.org/10.1093/eurheartj/ehf077.


