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Post-micturition syndrome: a neglected syndrome dangerous for the bladder and the heart

Ilaria Jane Romano, Francesco Gentile, Antonio Lippolis

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**Post-micturition syndrome: a neglected syndrome dangerous for the bladder and the heart.**Ilaria Jane Romano <sup>a</sup>, Francesco Gentile <sup>a</sup>, Antonio Lippolis <sup>a</sup><sup>a</sup> *Ospedale E. Bassini Cinisello Balsamo (MI)*

**Abstract.** Post-micturition syndrome refers to symptoms caused by over distension of the bladder or micturition. Bladder paraganglioma (BP) is a rare neuroendocrine neoplasm, which arises from the chromaffin tissue of the sympathetic nervous system embedded in the muscle layer of the bladder wall. Clinical presentation of catecholamine-secreting paragangliomas may mimic that of hyperfunctioning adrenal pheochromocytoma. Typical symptoms such as sweating, palpitations, headache, nausea, hypertension or flushing are due to catecholamine release and are related to micturition or bladder over-distension. We herein report the case of a 22-year-old woman admitted to the Emergency Department (ED) because of cranial trauma secondary to car accident. She referred history of micturition-related headache, nausea, sweating and increase in blood pressure since she was 13 years old. Neurological investigation was normal. No urogenital tract investigation was performed and, on admission, blood pressure was 190/125 mmHg. During hospitalization, abdominal ultrasonography, performed in order to rule out secondary hypertension, unexpectedly showed a large vascular soft tissue mass in the bladder wall, compatible with a paraganglioma. Twenty-four h urinalysis of catecholamines revealed high values of urine metanephrines. Abdominal magnetic resonance imaging (MRI) and histopathological evaluation of the surgical specimen, following resection of the bladder lesion, confirmed the diagnosis. Our case underlines the importance not to underestimate symptoms compatible with post-micturition syndrome, especially in young patients, in order to make early diagnosis of BP.

**1.Introduction.** Pheochromocytomas are neuroendocrine tumors usually arising from the chromaffin cells of the adrenal medulla. However, almost 10-15% of these tumors are found in extra-adrenal sites along the sympathetic or parasympathetic nervous system, and they are referred to as paragangliomas. Depending upon their localization, we classify two different type of paragangliomas: 1) parasympathetic paraganglioma, usually benign and not metastatic, not catecholamine-secreting whose symptoms derive from the compression of the adjacent anatomical structures (i.e., veins, nerves); this type represents the rarest variant of the disease (3%); 2) sympathetic paraganglioma, catecholamine-secreting, which tends to be malignant and metastatic, whose symptoms are both related to mass growth and catecholamine hyperproduction. In young patients (i.e., <20 years of age), the predominant type of pheochromocytoma is extra-adrenal. The majority of extra-adrenal tumors are intra-abdominal along the sympathetic chain (85%). However, they may also occur in the urinary tract: the urinary bladder is the most common site (79.2%) of genitourinary tract paragangliomas (1-2). They are extremely rare and represent less than 0.06% of all bladder tumors and less than 1% of all pheochromocytomas (3-4). Their rarity and often insidious clinical presentation makes early diagnosis extremely difficult.

**2.Case report.** A 22-year-old woman was admitted to the ED because of cranial trauma secondary to car accident. Brain computed tomography (CT) scan did not show any acute hemorrhagic lesion. However, the patient was hypertensive with systemic blood pressure of 190/125 mmHg. Patient had a familial history of arterial hypertension and was formerly a smoker. She also referred history of headache, nausea, sweating and increase in blood pressure occurring at the end of urination since she was 13 years old. Neurological investigation was normal. No urogenital tract investigation was performed. Electrocardiogram (ECG) upon arrival to the ED showed deep negative T waves in D1 and precordial leads with prolonged QTc. Antihypertensive therapy with nifedipine was prescribed and the patient was discharged and referred to Cardiology Department for further investigation. During hospitalization, despite therapy with nifedipine, patient tended to be hypertensive with systolic blood pressure close to 140 mmHg; laboratory tests were normal as well physical examination without arterial pressure gradient between upper limbs. Echocardiogram showed slight increase of left ventricle wall thickness that did not reach pathological criteria. Holter ECG did not show arrhythmias and basal ECG was unchanged with persistence of deep negative T waves in precordial leads, probably related to arterial hypertension

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