

glucose in the dialysate (1–1.5 g/l) may have contributed to the presentation of hypoglycaemia intradialysis and not at home.

Conclusion

In the differential diagnosis of hypoglycaemia during hemodialysis in a non-diabetic patient, the use of toxic agents such as cannabinoids and the use of pharmacological agents have to be ruled out.

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Adoración Martín-Gómez*, Maria Eugenia Palacios-Gómez, Sergio Antonio García-Marcos

Unidad de Hemodiálisis, Hospital de Poniente, El Ejido, Almería, Spain

*Corresponding author.

E-mail addresses: doritamg@gmail.com, doritamg@mixmail.com (A. Martín-Gómez).

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Hypertensive crisis in a patient with a medullary lesion[☆]

Crisis hipertensiva en paciente con lesión medular

Dear Editor,

The patient is a 22-year-old quadriplegic male with trauma was referred to us due to hypertensive emergency with blood pressure values of 250/150 mmHg associated with headache, blushing, perspiration, bradycardia and somnolence. Two years earlier patient had trauma that caused tetraplegia due to craneoencephalic and vertebro-medullary injury resulting in C5 fracture and anterior cervical arthrodesis with complete medullary motor C5 and sensitive C4 lesions. The patient was dependent on others to perform everyday activities and required intermittent urinary catheterization. At medical examination, the patient was conscious and oriented, eupneic in resting position with normal skin and mucosae colouration. Heart sounds were regular at 60 heartbeats per minute without murmurs or audible extra sounds; normal respiratory sounds. Abdomen was soft, nontender, without masses and palpable organomegaly, with no abdominal murmurs. Palpable and symmetric pedal pulse in both lower extremities was noticed. No oedemas were found.

A summary of blood tests: haemogram; haemoglobin 13.2 g/dl, haematocrit 37.1%, leukocytes 7.200/mm³, platelet count 307.000/mm³; biochemistry: glucose 65 mg/dl, urea 37 mg/dl, creatinine 0.63 mg/dl, GF >60 ml/min/MDRD4, cholesterol 140 mg/dl, LDL 73 mg/dl, Triglycerides 60 mg/dl, AST 28 U/l, ALT36 U/l, GGT 29 U/l, sodium 140 meq/l, potassium 4.7 meq/l, calcium 9.4 mg/dl, phosphate 4.3 mg/dl urine screening without alterations or sediments; 24-h urine sample collection: sodium 262 meq/24 h, ClCr 187.9 ml/min, proteins 198 mg/24 h, albumin 15.6 mg/24 h; endocrine screening: TSH 3.3 mIU/ml, cortisol 13.2 mcg/dl, 24 h urine free cortisol 41.8 mcg/24 h. Catecholamine and metanephrine in urine were normal. Echocardiogram showed no dilation or hypertrophy of left ventricle with preserved systolic function and normal wall motion. No dilation in right cavities; no valve abnormalities; no pericardial effusion. Normal Eye fundi; chest X-ray: no abnormal findings; normal kidneys by ultrasound. A 24-hour outpatient blood pressure (BP) monitoring was performed with the following results: average BP while awake 125/86 mmHg with average heart rate (HR) 75 b/m. Average BP during sleep

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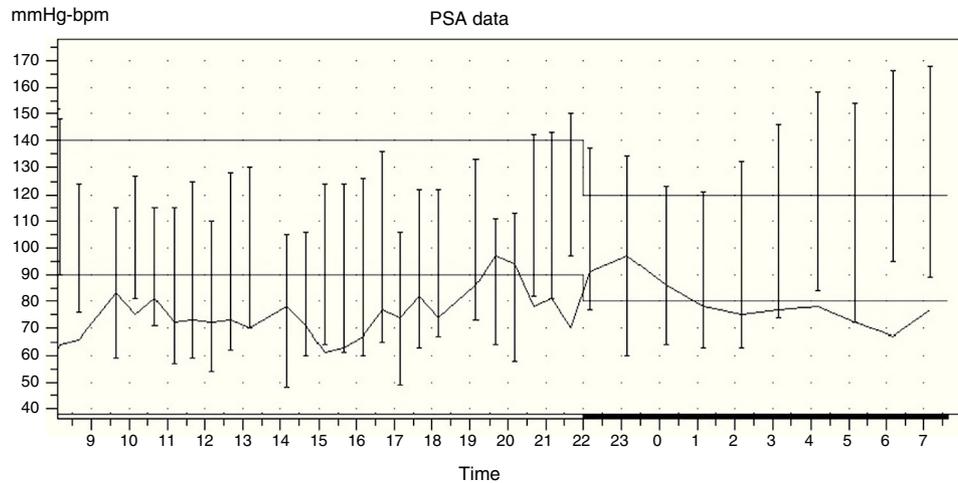


Fig. 1 – Blood pressure outpatient monitoring (BPOM).

was 144/74 mmHg with average HR of 80 b/m. The variation between the average BP between wakefulness and sleep state was of 19/6 mmHg (15%/9%). The variation of the average HR between wakefulness and sleep was of 5 b/m (7%). The following values of BP were recorded: 150/97 at 9:40 p.m.; 158/84, 154/72, 166/95 mmHg at 4:00–5:00 a.m. It could not be demonstrated that blood pressure elevation occurred prior to the procedure of bladder catheterization (when bladder was replete). The patient mentioned that around 5:00–6:00 a.m., he usually undergoes perspiration and headache that wakes him up, which disappear after the catheterization (Fig. 1). The first suspicion was autonomic dysreflexia (AD); other possible causes assessed were primary hypertension (AMPA and Holter results did not show continuous high BP), pheochromocytoma (catecholamine and methanephrine within normal boundaries), migraine headache (he only mentioned headache coinciding with episodes of high BP) and the presence of brain tumours (though ophthalmoscopic examination was normal, but no imaging tests were performed to rule out tumors). Therefore we believe this is a case of AD in a patient with medullary injury, probably caused by bladder stimulation and, as a consequence, by blood pressure rising. AD is an acute syndrome due to excessive and uncontrolled sympathetic response produced in patients with spinal cord injuries.¹⁻³ It usually appears after the medullary injury and affects between 48% and 90% of patients with medullary lesions affecting T6 or higher. As far as the pathophysiology, after spinal cord lesion, the sympathetic modulation of impulses travelling from the bladder zone to the brain through the spine is lost. In patients with injuries above T6, medulla afferent reflexes stimulate a sympathetic response, which originates in the mid-lateral cells column which are still operational despite the medullary injury; this is associated to an inadequate supraspinal control because the parasympathetic stimulus cannot travel through the injured medulla. The results include: hypertension as a sympathetic response, and bradycardia, perspiration, piloerection and headache a parasympathetic response.^{4,5} As far as treatment, first, avoid pharmacological treatment, and apply postural changes to lay the patient in supine position, removal of tight-fitting garments which perpetuate bladder

stimulus. The search and elimination of the sympathetic stimulus is crucial for the case to be controlled; it must be initially addressed to discard its origin in bladder and rectum, which are responsible for triggering AD in more than 80% of the crises.⁶ It must be checked that the bladder catheterization is permeable, that it is not causing injuries, that it is not painful when removed, etc. A second most frequent cause of hypertensive crises must be taken into account, i.e., gastrointestinal tract stimuli, such as constipation due to faecal impaction.^{7,8} Other causes, though less frequent, are cutaneous stimuli, menstruation, trauma, etc. (Table 1). In such a case that the triggering cause cannot be eliminated, a rapid-acting oral pharmacological treatment should be initiated. The most commonly used drugs are nifedipine and nitrites. Alpha-blockers (phenoxybenzamine) and alpha-agonists (clonidine) are also effective drugs in an AD crisis; other drugs used are hydralazine and, if necessary and under monitoring, IV sodium nitroprusside. Some studies advise the use of angiotensin converting enzyme inhibitors (ACEi) as a second option. Finally, alpha-blockers such as doxazosin can be used as prophylaxis and, therefore, as baseline treatment in such crisis in patients with recurrent episodes that

Table 1 – Possible AD causes.

Stimuli that can trigger AD:

- Bladder distension, vesical lithiasis, urinary sepsis, traumatic catheterization, cystoscopy, urodynamic testing
- Rectal distension, faecal impaction, complicated haemorrhoids, rectoscopy/colonoscopy, gastroduodenitis, gastroduodenal ulcer, "Silent" acute abdomen, gastroscopy, gallstones
- Tight garments, footwear or orthosis
- Menstruation, pregnancy, particularly during labour, vaginitis
- Epididymitis, ejaculation
- Pressure ulcers, burns
- Traumas: fractures, dislocations and sprains, heterotopic ossification
- Lymphangitis, deep vein thrombosis
- Surgical procedures

cannot be prevented.^{9,10} AD is a specific complication in patients with medullary injury that can involve risk of death, therefore a hypertensive crisis must be known and suspected of in this group of patients. Its management will be focused on its detection and avoiding the triggering cause of hypertensive crisis.

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Katia Toledo-Perdomo^{a,*}, Yareli Viña-Cabrera^b, Basilio Martín-Urcuyo^a, Adelaida Morales-Umpiérrez^a

^a Servicio de Nefrología, Hospital José Molina Orosa, Arrecife, Las Palmas, Spain

^b Atención Primaria, Centro de Salud Valterra, Arrecife, Las Palmas, Spain

*Corresponding author.

E-mail address: katia.toledo.perdomo@gmail.com (K. Toledo-Perdomo).

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Grover's disease in chronic kidney failure[☆]

Enfermedad de Grover en fracaso renal crónico

Dear Editor,

Patients with chronic renal failure may present multiple cutaneous manifestations such as pruritus, xerosis, cutaneous pigmentation, metastatic calcinosis cutis, calciphylaxis, pseudoporphyria and late cutaneous porphyria. Grover's disease (GD) must also be included in the differential diagnosis of cutaneous lesions in these patients.

We are presenting a case of a 69-year-old woman who presented with one and a half moth of cutaneous lesions in the back with moderate itching. She had no personal or family history of skin pathology. She had been receiving haemodialysis for five months due to Good pasture syndrome induced rapidly progressive renal failure. During the previous months, she had been treated with plasmapheresis, IV cyclophosphamide and IV methylprednisolone. At the time of consultation, her

treatment included prednisone 5 mg/day, calcitriol 0.25 mg/day, omeprazol 20 mg/day, enoxaparin sodium 20 mg 3 days/week and darbepoetin alfa 30 mcg weekly. Physical examination shows non-confluent papular erythematous lesions with keratotic surface, located in the back (Fig. 1a). She did not have lesions on palms or soles, back of the hands or the oral mucosa. She did not have a facial or scalp lesions either. Microscopic evaluation showed areas of hyperkeratosis with parakeratosis, acanthosis and focal acantholysis with presence of round and granular lesions (Fig. 1b). Taking into account the almost asymptomatic character of the lesions, the patient preferred to adopt an expectant attitude. Three months later, the lesions disappeared spontaneously. The absence of a previous family history, the onset at an adult age, and the spontaneous resolution of the symptoms led to the diagnosis of GD.

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