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CLINICAL CASE

Resistant hypertension and Morgagni hernia: The importance of nocturnal hypoxaemia

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Abstract A 51-year-old woman consulted for resistant arterial hypertension despite adequate antihypertensive treatment. Physical examination and analytical study showed no relevant abnormalities, with pulse oximeter saturation of 95%. The study highlighted nocturnal respiratory polygraphy with data of mild intensity sleep apnoea syndrome, and severe nocturnal hypoxaemia (apnoea hypopnoea index per hours of sleep [AHI] 7.8; desaturation index per hour [ODI]: 12.6. Oxygen-medium saturation: 89%, minimum saturation: 72%. CT90: 34.2%).

The chest X-ray showed elevation of the right hemidiaphragm, and the chest computed tomography (CT) revealed a Morgagni hernia with a maximum diameter of 20 cm. After adjusting the antihypertensive treatment, the patient was referred to General Surgery for intervention. The onset of resistant hypertension secondary to severe nocturnal hypoxemia from a large Morgagni hernia has not been previously described in the literature.

PALABRAS CLAVE
Hipertensión;
Hipertensión resistente;
Hernia de Morgagni;
Hipoxemia nocturna;
Síndrome de apnea hipopnea nocturna

Hipertensión resistente y hernia de Morgagni: la importancia de la hipoxemia nocturna

Resumen Mujer de 51 años de edad que consultó por hipertensión arterial resistente, pese a tratamiento antihipertensivo adecuado. Presentaba una exploración y estudio analítico sin alteraciones relevantes, con saturación del 95% por pulsioximetría. En el estudio destacaba una poligrafía respiratoria nocturna con datos de síndrome de apnea del sueño de intensidad leve, e hipoxemia nocturna grave (índice de apneas hipopnea por horas de sueño [IAHH] 7,8; índice de desaturaciones por hora [IDH]: 12,6. Oxígeno-saturación media: 89%, saturación mínima: 72%. CT90: 34,2%).

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La radiografía de tórax mostró una elevación de hemidiafragma derecho, comprobándose en la tomografía computarizada (TC) torácica una hernia de Morgagni de 20 cm de diámetro máximo. Tras ajustar el tratamiento antihipertensivo, la paciente se derivó a cirugía general para su intervención. La aparición de hipertensión resistente secundaria a hipoxemia nocturna grave por una gran hernia de Morgagni no ha sido descrita previamente en la literatura.

Introduction

Resistant hypertension (HTN) is a relatively common problem in clinical practice and may affect 20%–30% of the hypertensive population.[1] According to the current guidelines, it is defined as resistant when adequate blood pressure control is not achieved by antihypertensive treatment in appropriate doses with three or more drugs, including a diuretic.[2]

Multiple studies in recent years have identified sleep apnoea/hypopnoea syndrome (SAHS) as one of the most significant secondary causes of resistant HTN, with a prevalence of 30%–40% of hypertensive patients, reaching rates of 70%–90% in subjects with resistant HTN,[3] and it is a frequent reason for referral to our clinics.

In SAHS patients, the sequence of episodes of intermittent cyclic hypoxia leads to endothelial dysfunction, along with activation of the renin–angiotensin system, the sympathetic system, and the release of inflammatory mediators, which ultimately results in the development of HTN and, eventually, cardiovascular disease.[3]

In fact, low oxygen saturation levels at night double the risk of death and increase the risk of cardiac or cerebrovascular events by 50%.[4]

However, although nocturnal hypoxia may be a marker of SAHS, it may also be a symptom of other diseases. We present an exceptional case in which nocturnal hypoxaemia was key in the development of resistant HTN.

Clinical case

A 51-year-old woman, smoker of 30 packs/year, and BMI of 33. Her personal history included an operation in 2016 for a Morgagni hernia. She was studied for a non-functioning adrenal incidentaloma in the endocrinology department. She was referred from this department due to refractory HTN, despite treatment with amlodipine 10 mg, doxazosin 8 mg, and hydrochlorothiazide 25 mg.

A secondary HTN study was completed in the clinic. Examination and laboratory tests showed no relevant alterations. Pulse oximetry in consultation showed oxygen saturation of 95%. Ambulatory blood pressure monitoring (ABPM) was requested, showing sustained systolic–diastolic hypertension with mean blood pressure during wakefulness of 154/97 mmHg and 149/88 mmHg at rest, with a nocturnal non-dipper pattern (−3%). Renal artery echo-Doppler was normal. The study of secondary causes was extended, and nocturnal respiratory polygraphy revealed mild sleep apnoea syndrome and severe nocturnal hypoxaemia (apnoea hypopnea index per hour of sleep [AHI] 7.8; hourly oxygen desaturation index [ODI]: 12.6. Mean oxygen saturation: 89%, minimum saturation: 72%. CT90: 34.2%).

Chest X-ray showed an elevated right hemidiaphragm, and chest CT scan showed a Morgagni hernia with a maximum diameter of 20 cm. This hernia was causing compression of the right lower pulmonary lobe, as well as downward displacement of the liver parenchyma (Fig. 1).

After adjusting her antihypertensive treatment, the patient was referred to general surgery for reoperation of her hernia.

Clinical judgement

Resistant hypertension secondary to nocturnal hypoxaemia due to Morgagni–Larrey diaphragmatic hernia.

Discussion

Morgagni–Larrey hernia is a very rare congenital anterior or retrosternal diaphragmatic defect that constitutes 2%–3% of congenital diaphragmatic hernias.[5] It is generally detected during childhood, and is rarely diagnosed in adulthood, as in our patient. In these cases, it is usually asymptomatic and discovered by chance, often on a chest X-ray.[6]

A diagnosis of Morgagni's hernia is usually suspected with a chest X-ray. However, it should usually be confirmed by CT or MRI, which help to better delimit and assess the condition.[5]

Treatment is always surgical. Repair is recommended even in asymptomatic patients to reduce the risk of gastrointestinal complications (volvulus, intestinal malrotation, incarceration, etc.), and respiratory compromise.[6]

In relation to the development of resistant hypertension, as mentioned above, nocturnal hypoxia episodes will be the key, as evidenced in our patient.

Episodes of hypoxia stimulate chemoreceptors that increase sympathetic activity, increasing blood pressure readings and variability compared to healthy subjects.[7]

In addition, these episodes of hypoxia trigger the release of inflammatory mediators: hypoxia-inducible factor 1, nuclear factor kappa, vasoconstrictor endothelin-1, interleukins (IL) 1, 2 and 6, tumour necrosis factor alpha (TNFα) and interferon gamma (IFNγ), which both directly and indirectly damage the endothelial lining of blood vessels, in addition to causing increased platelet release and

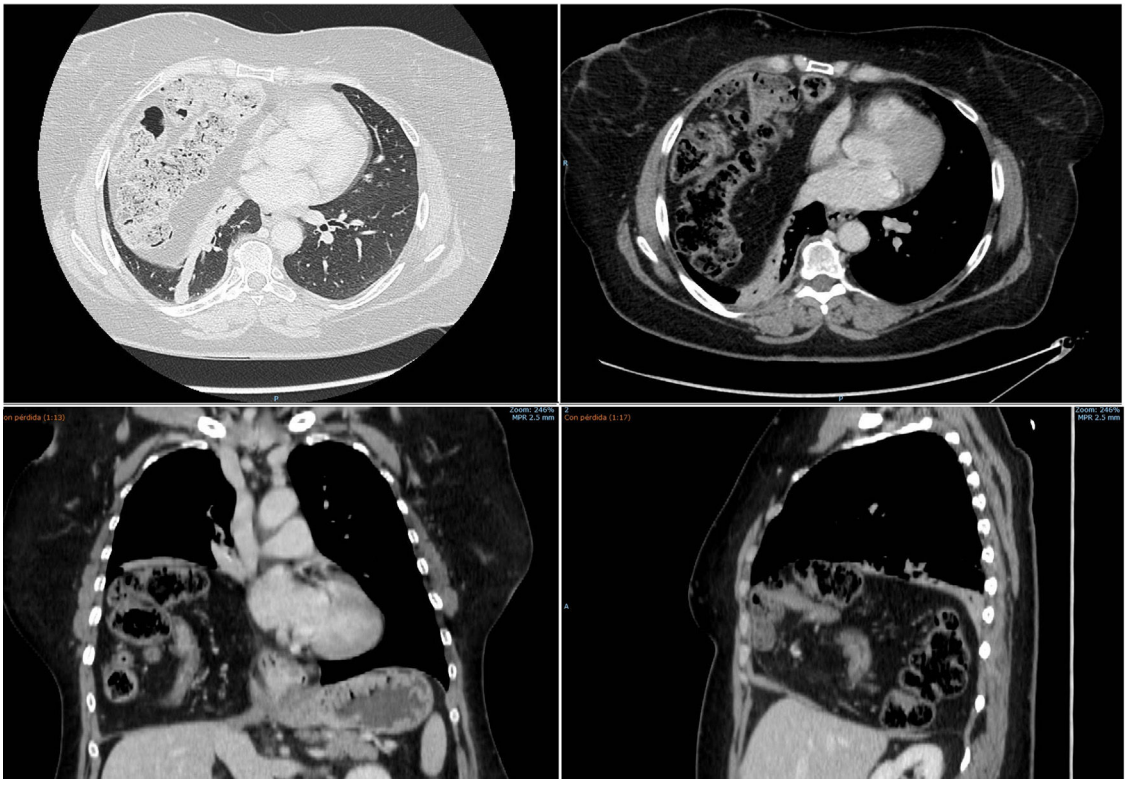

Figure 1 Chest CT scan confirming the presence of a large right-sided Morgagni hernia with compression of the lower pulmonary lobe and displacement of the liver.

aggregation, which ultimately leads to increased oxidative stress and vascular endothelial damage, causing further increases in arterial stiffness and tone.[3]

Thus, increases in C-reactive protein (CRP), IL-6 and IL-8 released during hypoxaemia episodes have been associated with an increase in carotid intima-media thickness (IMT), which is a precursor to HTN.[8]

Hypoxaemia also increases oxidative stress in the body, which would alter the biological activity of endogenous vasoactive factors (e.g., with decreased nitric oxide [NO] synthesis), which in turn would raise blood pressure.[9]

In fact, it is now known that both the development of hypertension and the onset of cardiovascular events in patients with sleep-disordered breathing (SDB), like SAHS, are more closely related to hypoxic events than to indices representing the frequency of apnoea or hypopnoea episodes.[10]

Therefore, it is important to consider that there are other respiratory causes of resistant HTS secondary to nocturnal hypoxaemia. However, occurrence of the condition in the context of a large Morgagni hernia is an exceptional case that has not been previously described in the medical literature.

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Conflict of interests

The authors have no conflict of interests to declare.

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