The advantages of the technique are obvious: it is non-invasive, reproducible, economical, and quick and simple to perform by staff with the appropriate training. Its disadvantages include the fact that 10% of patients with intracranial hypertension do not present these changes in the optic nerve, probably due to individual anatomical variability. It is also unable to detect cases of intracranial hypertension with acute progression. This should be considered a complementary technique, and the results must be confirmed with definitive diagnostic studies, such as neuroimaging or CSF pressure testing, in cases of strong diagnostic suspicion.[5]

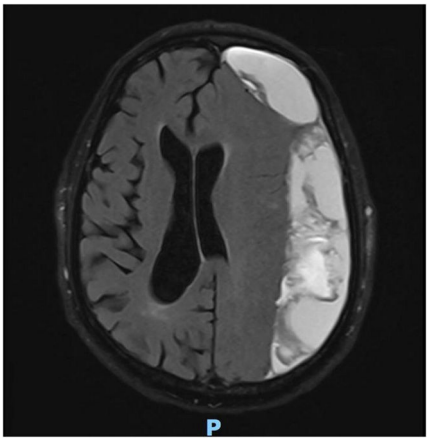

Figure 2 FLAIR MRI study, axial plane.

This case demonstrates how orbital ultrasound may be useful in clinical practice in patients with rapidly progressive dementia and history of trauma. Proper training and experience, as well as the availability of an ultrasound system at the consultation, were essential in performing the study. Used by experienced professionals, it can be highly effective for diagnosing suspected intracranial hypertension and for indicating more efficient diagnostic or therapeutic measures in various areas of neurology.

This ultrasound technique may provide highly valuable information in the initial assessment of the following conditions: *a)* headache with signs or symptoms of intracranial hypertension; *b)* headache associated with obesity; *c)* differential diagnosis of bilateral papilloedema/pseudopapilloedema; *d)* non-invasive monitoring of intracranial pressure in critical patients with extensive stroke, meningitis, or trauma; and *e)* rapidly progressive dementia associated with focal neurological signs or history of falls or trauma, as in the case of our patient. Scientific data are currently limited, and further clinical research may be beneficial.

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F. Delgado López[a,b]

[a] *Neurólogo, Centro de Neurología Avanzada, Jerez de la Frontera, Cádiz, Spain*
[b] *Hospital Puerta del Sur, Jerez de la Frontera, Cádiz, Spain*

E-mail address: fdelgadolopez@gmail.com

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Rowland Payne syndrome☆

Síndrome de Rowland Payne

Dear Editor:

We present the case of a 71-year-old woman with history of arterial hypertension and type 2 diabetes mellitus who visited the emergency department due to left-sided ptosis of 2 weeks' progression. She also reported constant dull pain in the left shoulder, located on the belly of the trapezius muscle, which was not altered with postural changes or Valsalva manoeuvres. The patient reported no other focal neurological signs or other symptoms. Three weeks earlier, she had undergone laparoscopic cholecystectomy and was discharged with no neurological symptoms. The neurological examination revealed narrowing of the left palpebral fissure and anisocoria with relative miosis in the left eye, observable only in semi-darkness; these findings are compatible with Horner syndrome (Fig. 1A). Examination of the cranial nerves, motor system, sensitivity, reflexes, coordination, and balance revealed no other alterations.

Upon arrival at the hospital, a blood analysis ruled out relevant alterations in the blood count, coagulation,

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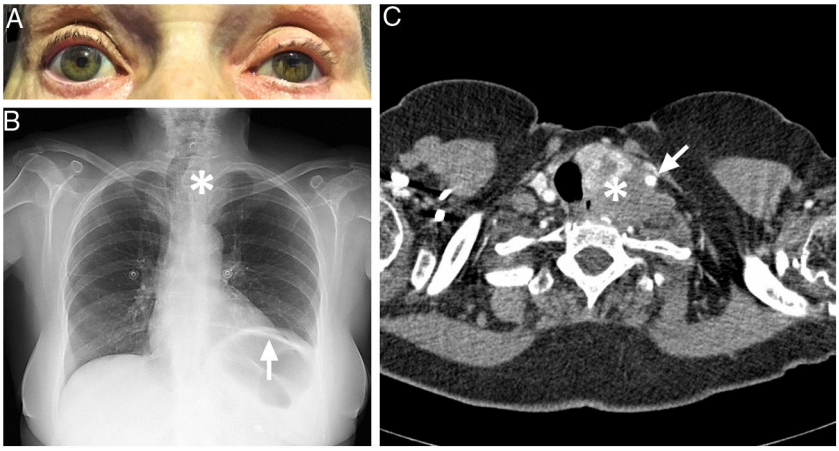

Figure 1 (A) Left ptosis and miosis in semi-darkness. (B) Anteroposterior chest radiography showing elevation of the left hemidiaphragm (arrow) and right-sided tracheal deviation in the superior mediastinum (asterisk). (C) Neck CT scan (axial plane) with intravenous contrast, revealing a mass originating from the left thyroid lobule (asterisk), with irregular density and involving the left common carotid artery (arrow).

Table 1 Cases of Rowland Payne syndrome reported in the literature.

Reference	Author	Year	Patient	Aetiology
1	Rowland Payne	1981	1	Adenopathy, breast cancer
			2	Adenopathy, breast cancer
			3	Adenopathy, breast cancer
7	Vaghadia	1983	4	Adenopathy, breast cancer
8	Amin	1984	5	Adenopathy, breast cancer
			6	Lung epidermoid carcinoma
9	Ismail	1988	7	Tuberculous lymphadenitis
10	Kapoor	2005	8	Neuroblastoma

biochemical parameters, and C-reactive protein. A non-contrast head CT scan revealed normal results. A chest radiography showed a loss of volume of the left lung secondary to elevation of the ipsilateral hemidiaphragm (absent in the preanaesthetic assessment conducted 2 months earlier) and right-sided tracheal deviation in the superior mediastinum (Fig. 1B).

The combination of Horner syndrome, left phrenic nerve palsy, and tracheal deviation led us to suspect a cervical lesion in the left superior mediastinum. Neck CT scans performed before and after contrast administration confirmed the presence of a left paratracheal mass arising from the thyroid gland, showing irregular contrast uptake, partially involving the ipsilateral common carotid artery (Fig. 1C). An MRI study of the cervical spine revealed that the mass, located in the left prevertebral region between C6 and C7, did not infiltrate the vertebral bodies or intervertebral foramina. The anatomical pathology study of samples obtained by fine needle aspiration biopsy and subsequent core needle biopsy was not sufficient for diagnosis; therefore, we opted to perform open surgery for diagnostic and therapeutic purposes. The procedure revealed a left anterior cervical mass with poorly-defined edges, infiltrating the surrounding tissues; the mass was partially resected. The anatomical pathology diagnosis was anaplastic thyroid carcinoma. Treatment was started with radiotherapy, despite which we observed rapid progression of local pain and thrombosis of the left internal jugular vein. The patient was enrolled in a palliative care programme and was lost to follow-up 5 months after symptom onset.

The ipsilateral presence of Horner syndrome, shoulder pain, phrenic nerve palsy, and vocal cord paralysis (absent in our patient at the time of diagnosis) was described by Rowland Payne[1—3] as a manifestation of expansive lesions in the anterior and inferior region of the neck or in the thoracic inlet. These alterations have been described as complications of interscalene nerve blocks.[4—6] Rowland Payne syndrome in the absence of this kind of anaesthetic block has been reported almost exclusively in association with malignant tumours, as in the case of our patient (Table 1).[1,7—10] The lesion causing the syndrome is usually located at the level of the sixth cervical vertebra, although it may also be caused by expansive lesions with greater volume and more caudally located (in the thoracic inlet).[1—3,7] This anatomical location is the only one where the cervical sympathetic plexus, the phrenic nerve, and the recurrent laryngeal nerve run in close proximity.

It is therefore important to be aware of and to recognise this rare syndrome, given its anatomical and clinical correlation and its strong association with neoplastic lesions.

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F. Sierra-Hidalgo*, E. Aragón Revilla

Sección de Neurología, Hospital Universitario Infanta Leonor, Madrid, Spain

*Corresponding author.
E-mail address: fernando.sierragh@salud.madrid.org
(F. Sierra-Hidalgo).

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Hereditary cerebellar ataxias and hereditary spastic paraplegias: experience of disease from the patient's perspective☆

Las Ataxias y Paraparesias Espásticas Hereditarias: experiencia en torno a la enfermedad desde la perspectiva del paciente

Dear Editor:

Hereditary cerebellar ataxias (HCA) and hereditary spastic paraplegias (HSP) are a very heterogeneous group of genetic neurodegenerative diseases that may affect populations of any sex and at any age. The main symptoms consist of slowly progressive cerebellar ataxia and/or spastic paraparesis, which may be associated with other neurological or systemic manifestations.[1] Their diagnosis is complex, and no disease-modifying treatment is available in most cases.[2]

Patients with this type of condition face several issues: insidious symptom onset that may lead the patient to consult even years after disease onset; occasionally, difficulty in identifying the nature of symptoms, which may lead to the patient being assessed by other specialists (traumatologists, otorhinolaryngologists, etc), which in turn delays diagnosis; the need for extensive and time-consuming batteries of diagnostic tests, which do not always lead to a definitive diagnosis (ie, a specific molecular diagnosis)[3,4]; and finally, after diagnosis, a progressive and incapacitating disease that, due to its genetic nature, may have implications not only for patients but also for their direct family members.

In this context, the aim of this study was to identify the main experiences and concerns related to the disease from the patient perspective. To that end, we recruited 12 consecutive patients with HSA/HSP from our hospital's specialised unit between April and June 2018. Participants gave written informed consent to participate in a group interview (discussion group) addressing their main experiences and concerns regarding their condition. The discussion or focus group, which included 12 patients and 8 family members, met at a meeting room where a 2-hour session was held; 3 expert moderators led the session, and participants explained the concerns and needs arising over the course of the disease. The semi-structured questionnaire focused on the following settings: the patient's environment (what does he/she feel/think/do?), the external resources available to the patient (physician, family environment, and community), and the progression stage (first symptoms, diagnosis, and progression). The moderators noted the participants' answers as patients recounted their journey with the disease.

Table 1 includes the characteristics of the sample of participating patients.

With respect to the data collected, we observed that patients' main questions at disease onset were related to recognising the first symptoms and undergoing specialised assessment. Later, at diagnosis, patients' emotional experi-

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