Figure 2 CT of the sacrum. The arrow points to a small hypodense area representing air.

zones corresponding to soft mass density. Sporadically, inside the spinal canal some small hypodense zones were visible, the absorptivity of which corresponded to air (fig 2). Abdominal and pelvic CT scans were normal. Findings of lung and heart radiography and ECG were normal. The EEG indicated slow activity. Lumbar puncture was not carried out. Because of the condition of the patient neurosurgical intervention did not take place. The patient's condition gradually deteriorated and she died three days after admission to hospital. The basic pathophysiological mechanism of pneumocephalus is the development of a dural fistula which permits the flowing out of the CSF and the entry of air into the subarachnoid space, which, because it cannot flow back, successively accumulates intracranially. In head injuries, pneumocephalus is recorded in 0.5% to 1.0% of all cases.¹ It may also develop as a complication of neurosurgical treatment of chronic subdural haematoma or after posterior cranial fossa surgery.² It is described in the craniofacial approach to tumour removal from the base of the anterior cranial fossa as well as in nasal and otological surgery.³ Intracranial air appears as the consequence of eroding otological infections, of eroding tumours of the brain, nose, and nasopharynx in combination with radiotherapy.⁴ It is mostly described as a complication which follows insertion of a nasogastric tube and a case has been described also after insertion of a nasopharyngeal tube.⁵ Subarachnoid-pleural fistula and intracranial air may occur after severe chest trauma. Cases after thoracotomy and surgery of the tumour of the lung apex have also been described.⁶ Pneumocephalus may also develop when administering epidural anaesthesia and in continuous external lumbar subarachnoid drainage in the treatment of dural fistula.⁷ The first symptom of pneumocephalus is a headache which in anterior cranial fossa fractures or eroding processes may also be accompanied by rhinorrhea. Should the headache intensify and be coupled with other signs of increased intracranial pressure, an urgent brain CT is required to diagnose pneumocephalus. In

most cases it is a small quantity of air and the developed fistula may heal spontaneously. Should the fistula persist, urgent operative repair is needed to prevent brain herniation and death.

We consider the case described above to be exceptionally rare given the way pneumocephalus develops. In our opinion, the destruction of the sacrum, of skin, and of subcutaneous tissue of the sacral region, assigned to a malignant process which was hastened by eroding infection, caused a communication between the subarachnoid space and the surface of the skin. We think that the developed sacrodural fistula is the only possible explanation for the development of pneumocephalus.

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- Orebaugh SL, Margolis JH. Post-traumatic intracerebral pneumatocele: case report. *J Trauma* 1990;30:1577-80.
- Tripathy P, Chowdhury BK, Bhattacharya A, Munshi AK, Bhattacharya MK. Tension pneumocephalus following posterior cranial fossa surgery in sitting position. *J Indian Med Assoc* 1995;93:109-10.
- Wanamaker JR, Mehle ME, Wood BG, Lavertu P. Tension pneumocephalus following craniofacial resection. *Head Neck* 1995;17:152-6.
- Ng WF, Fung KH, Sham JS. Tension pneumocephalus a rare complication of radiotherapy in nasopharyngeal carcinoma. *Pathology* 1995;27:204-8.
- Glasser SA, Garfinkle W, Scanlon M. Intracranial complication during insertion of a nasogastric tube. *AJNR Am J Neuroradiol* 1990;11:1170.
- Malca SA, Roche PH, Touta A, Pellet W. Pneumocephalus after thoracotomy. *Surg Neurol* 1995;43:398-401.
- Gonzales Carrasco FJ, Aguilar JL, Llubia C, Nogues S, Vidal Lopez F. Pneumocephalus after accidental dural puncture during epidural anesthesia. *Reg Anesth* 1993;18:193-5.

Paraneoplastic opsoclonus associated with papillary renal cell carcinoma

Opsoclonus is an ocular dyskinesia consisting of involuntary, repetitive, rapid conjugate ocular saccades that are irregular in amplitude and frequency and occur in all directions without an intersaccadic interval.¹ Opsoclonus appears more commonly in children,² and in half of such cases in association with neuroblastoma.¹ In adults, the most frequent causes are idiopathic (50%) and paraneoplastic (20%).¹ Among the second group, different types of tumours involving a wide variety of organs have been reported, although no case of renal tumour has been published.¹

A previously healthy 64 year old man complained of progressive vertigo and unsteady gait, together with mild headache, since a month before admission. At examination he showed severe opsoclonus (increased by saccadic movements and visual fixation), slight cephalic myoclonic movements, and he could not stand or walk because of truncal ataxia. The rest of the neurological evaluation was unremarkable.

Blood tests included white cell count, haemoglobin, erythrocyte sedimentation rate, glucose, urea, creatinine, bilirubin, immunoglobulins, and angiotensin converting enzyme. These were all normal. Serological tests for syphilis and HIV were negative, as was the screening for several tumour markers. Anti-Hu and anti-Ri serum antineuronal antibodies were not detected. On admission, CSF contained 19 mononuclear white blood cells (WBC)/mm³, 73 mg/dl protein, with normal glucose concentration and negative cytological examination. Two months later, a new CSF examination showed 40 mononuclear WBC/mm³, 47 mg/dl protein, and normal glucose concentration. Serological tests, as well as bacterial and fungal cultures, were negative in both CSF studies. Brain MRI and thoracoabdominal CT were normal.

Soon after the beginning of treatment with 60 mg/day oral prednisone the patient's neurological symptoms gradually improved. Seven weeks later, despite a slow decrease in the dose of prednisone, the patient became asymptomatic. Six months later, he complained of lumbar pain and radiography suggested the existence of lumbar osteoblastic lesions. Both ultrasound and CT studies disclosed a mass in the right kidney, without evidence of any bone metastasis. After a radical nephrectomy, the pathological study gave the diagnosis of a papillary type of renal cell carcinoma. At present, three years after the initial presentation, the patient remains asymptomatic and without any treatment.

Paraneoplastic opsoclonus usually antedates the detection of the underlying neoplasm by a period that may last up to one year.^{1,3} This sequence of events was also found in our patient, as the first abdominal CT examination did not detect the tumour. This could be explained by the fact that a substantial proportion of small renal masses can be missed by CT and ultrasound studies.⁴ Therefore, we consider that the development of opsoclonus in our patient was probably linked to his renal tumour. The pathogenesis of paraneoplastic opsoclonus is unknown, although there is increasing evidence that it may be immune mediated.¹ It has been proposed that antigenic substances may be shared between certain tumours and portions of the nervous system.⁵ This would elicit an efficient immune response against the

tumour, but also against the neural tissue.⁵ This hypothesis would be supported by the complete recovery of our patient during treatment with prednisone and before surgery. However, the possibility of spontaneous remission in paraneoplastic opsoclonus cast doubts on the role of steroids on this improvement.³ In any case, if the mechanism is autoimmune, it would have been mediated by an unknown antineuronal antibody as those known at present were not found in the serum of our patient.

The diagnosis of opsoclonus has to be followed by an extensive search for malignancy which, after the present case, should include renal carcinomas.

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- 1 Caviness JN, Forsyth PA, Layton DD, McPhee TJ. The movement disorder of adult opsoclonus. *Mov Disord* 1995;10:22-7.
- 2 Digre KB. Opsoclonus in adults. Report of three cases and review of the literature. *Arch Neurol* 1986;43:1165-75.
- 3 Anderson NE, Budde-Steffen C, Rosenblum MK, Graus F, Ford D, Synek BJL, et al. Opsoclonus, myoclonus, ataxia, and encephalopathy in adults with cancer: a distinct paraneoplastic syndrome. *Medicine Baltimore* 1988;67:100-9.
- 4 Jamis-Dow CA, Choyke PL, Jennings SB, Linehan WM, Thakore KN, Walther MM. Small (<3-cm) renal masses: detection with CT versus US and pathologic correlation. *Radiology* 1996;198:785-8.
- 5 Posner JB. Pathogenesis of central nervous system paraneoplastic syndromes. *Rev Neurol (Paris)* 1992;148:502-12.

In vivo visualisation of the longitudinal callosal fascicle (Probst's bundle) and other abnormalities in an acallosal brain

A 45 year old office clerk with a two year history of intermittent tension like headache presented with difficulties of walking heel to toe and of exchanging sensorimotor information between both hemispheres (alternating bilateral tapping, contralateral mirroring of finger position, cross localisation of tactile stimuli, finger sequence repetition, and bilateral coordinated arm rotations). There were no complaints about limitations in activities of daily life. The family history was unremarkable regarding motor disabilities, mental retardation, or epilepsy. MRI (A, C-F: TR 25 ms, TE 6.9 ms; B: TR 4000 ms, TE 90 ms) disclosed an absence of the corpus callosum, massa intermedia, posterior commissure (figure A) and septum pellucidum (figure C-F) and a hypoplastic anterior commissure (figure A). On the basis of the history and the anatomic findings, the patient can be classified as having an "asymptomatic" acallosal brain or a complete agenesis of the corpus callosum.¹

The convolutional pattern on the medial surface of the hemisphere was abnormal with gyri radiating in a fan-like fashion and without a visible callosomarginal sulcus (figure A). On the axial and coronal slices an anteroposteriorly running fibre tract (arrow head, figure B-F) was visible which could clearly be distinguished from the medially situated cingulate gyrus (arrow in figure D). This fibre tract has been named after Probst (Balkenlängsbündel of Probst, callosal longitudinal bundle) who saw it as a typical sign of agenesis of the corpus callosum.² Pathoanatomical investigations of the origin and course of the fibre tract led to the conclusion

that it consists of heterotopic myelinated callosal fibres and results from a migration disorder of callosal fibres.^{2,3} During embryogenesis the fibres are thought to arrive at the midplane where they are hindered in their further migration across the midline and then change their direction of growth into an anteroposterior direction and by this form the Probst's bundles in each hemisphere.³ This hypothesis about the development of the Probst's bundles is supported by recent investigations in acallosal mice.⁴

The absence of the corpus callosum and the volume of the Probst's bundles influence the shape of the ventricles. The posterotemporal parts of the lateral ventricles and the third and fourth ventricle are enlarged. Frontally, the Probst's bundles are thickest and become smaller on their frontooccipital course (figure B). Frontally they are comma shaped, bulge into the medial wall of the lateral ventricles, and cause a narrowing and dorsolateral transposition of them (bull horn formation of the ventricles in figure D, E).^{2,3} Posteriorly, the Probst's bundle forms a thin layer on the upper medial wall of the lateral ventricles which are consecutively dilated

(figure F). Additionally, a completely separated fornix could vaguely be identified at the lower medial wall of the lateral ventricles directly ventral to the longitudinal callosal bundle (figure D, short arrow in E).

This case illustrates that MRI allows a precise in vivo examination of the anatomical situation in patients with accidentally detected acallosal brains. Especially, the detailed characteristics of Probst's bundle were to our knowledge previously only defined pathoanatomically, which is somewhat limited by shrinkage artifacts. The detection of Probst's bundle during life might be useful to further elucidate the function of this fibre tract by evoked potential studies using transcranial magnetic or electric brain stimulation similar to the approach described for an activation of interhemispheric fibres in humans.⁵

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In vivo visualisation of the longitudinal callosal fascicle (Probst's bundle) (arrow head) and other abnormalities in an acallosal brain. Long arrow in D=cingulate gyrus; short arrow in E=fornix.