A 25 year old woman presented to her general practitioner complaining of impaired concentration, mood swings, disturbed sleep, and memory loss. One month later, she had noticed a fine tremor in both hands and occasional spasms affecting her right foot. Her concentration was worse and she mentioned word finding difficulties. She also had a tendency to stagger and fall. She was seen by a psychiatrist and somatisation was initially suspected. Later, she was referred to a neurologist. He noted that she had been acting oddly—for example, being found by her mother in a bath of cold water. Neurological examination was normal except for a mini mental test examination score of 20 of 30 with a child-like effect and stilted speech. A magnetic resonance imaging scan was unremarkable. She continued to deteriorate, developing right sided myoclonus. By this stage, she was unable to perform simple tasks, such as washing, and was aware of crawling sensations all over her body. Six months after her first presentation she had an EEG. This revealed repetitive complexes occurring every four to six seconds, often associated with a myoclonic jerk and consistent with SSPE. CSF analysis confirmed oligoclonal bands not present in the serum, which were positive for measles antibody antigen by immunoblotting. She had contracted measles at age 11 months. She was started on oral piracetam and inosiplex, along with subcutaneous interferon β 1a, but she became bed bound with quadriplegia, dysarthria, and diffuse hyperalgesia. Her treatment was changed to intraventricular interferon α 2a administered via an Ommaya reservoir. She required subcutaneous infusions of midazolam and diamorphine for symptom control, and nasogastric feeding was started. Her condition subsequently stabilised and she was discharged to a children's hospice.

Discussion

The estimated incidence of SSPE each year in developed nations is < 1/10 million of the population under the age of 20. The occurrence of two adult cases in the small region of West Yorkshire (population two million) within a few months of one another is remarkable. Although probably a chance finding, our experience emphasises the need for continued surveillance in populations where measles is no longer endemic. In theory more adult onset SSPE cases could present to neurologists in the future. This is because slow central nervous system spread of the virus over many years leaves open the potential for SSPE to present in later life, decades after population eradication of measles. In addition, routine immunisation has led to a shift in the incidence of measles towards unprotected children age < 1, who are at a higher relative risk of developing SSPE in later life after a longer incubation period.² Recently, there has been an increase in the number of measles outbreaks after a decline in the uptake of the combined MMR immunisation because of safety fears.³

In our patients, SSPE was not initially suspected, leading to diagnostic delay. Enquiring about measles in childhood may have been helpful in case 2, but not case 1, because infection in infancy was probably subclinical. In this patient the lack of typical findings on initial EEGs led to further delay, and instead we considered variant Creutzfeldt Jakob disease as a possibility. Patient 2 presented with early cognitive and

behavioural changes. Initially, patients may come to the attention of other specialties with non-specific or psychiatric symptoms.⁴ It was only subsequent worsening of the patient's mental state associated with the development of unilateral myoclonic jerks that prompted further investigation, including EEG.

There is no curative treatment for SSPE. Trials have been complicated by variable natural history and spontaneous long remissions of the disease, although it is eventually fatal (median survival, three years). One study demonstrated similar response rates among patients randomised to inosiplex, with or without intraventricular interferon α 2b treatment for six months (35% v 34%).⁵ However, these figures were substantially higher than historical remission rates of between 5% and 10%. In our hands, intraventricular interferon was associated with initial worsening of encephalopathy and pronounced hyperpyrexia, possibly the result of chemically induced meningitis, and reservoir infection was a further complication. In both patients, disease appeared eventually to stabilise after antiviral treatment, particularly in patient 1, in whom rapid progression at onset was suggestive of fulminant SSPE, usually fatal within three months.

Prevention seems to be the best approach, with mass immunisation leading to a drastic reduction in reported cases of measles and associated complications. However, our experience suggests that SSPE in adults should not be forgotten. Clinicians should remain vigilant for this devastating disease.

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References

- 1 Garg RK. Subacute sclerosing panencephalitis. *Postgrad Med J* 2002;**78**:63–70.
- 2 Miller C, Andrews N, Rush M, et al. The epidemiology of subacute sclerosing panencephalitis in England and Wales 1990–2002. *Arch Dis Child* 2004;**89**:1145–8.
- 3 Jansen VA, Stollenwerk N, Jensen HJ, et al. Measles outbreaks in a population with declining vaccine uptake. *Science* 2003;**301**:804.
- 4 Salib EA. Subacute sclerosing panencephalitis (SSPE) presenting at the age of 21 as a schizophrenia-like state with bizarre dysmorphic features. *Br J Psychiatry* 1988;**152**:709–10.
- 5 Gascon GG. Randomized treatment study of inosiplex versus combined inosiplex and intraventricular interferon-alpha in subacute sclerosing panencephalitis (SSPE): international multicenter study. *J Child Neurol* 2003;**18**:819–27.

Laterality of symptoms in patients admitted to a stroke unit who had a discharge diagnosis of a psychiatric condition

Some psychiatric conditions produce symptoms that can mimic an acute neurological disease, including stroke.^{1–3} In several studies, such symptoms seemed to be more common on the left side of the body.^{1–3} The

predominant processing of emotional information by the right hemisphere offers a hypothetical explanation for this finding.

We reviewed the discharge summaries of patients who were admitted to a stroke unit during the period May 1996 to December 2003 with a diagnosis of acute stroke and who had a discharge diagnosis of somatoform disorder and/or anxiety disorder according to the DSM-IV, revised⁴ criteria, and no recent stroke, according to the World Health Organisation definition.⁵

Two investigators (CS and LC) reviewed the discharge summaries independently and collected the following data in a standardised form: (a) age; (b) sex; (c) discharge psychiatric diagnosis (somatoform disorders: somatisation disorder and conversion disorder; anxiety disorders: generalised anxiety disorder and panic disorder); (d) type (motor paresis and/or involuntary movements, sensory, visual, other) and side (right, left, bilateral) of the symptoms; (e) vascular risk factors; (f) neuroimaging data; and (g) length of stay. Disagreements were solved by consensus.

RESULTS

From 2279 consecutively admitted patients to our stroke unit, we included 35 (1.5%) discharge summaries for review. Of these, 25 patients had received a diagnosis of somatoform disorder (14 with somatisation disorder and 11 with conversion disorder), and 7 patients had a diagnosis of anxiety disorder (4 with generalised anxiety disorder and 3 with panic disorder). Three patients had other psychiatric diagnoses. Symptoms were presented on the left side of the body in 11 patients, on the right side of the body in 14, and 10 presented bilateral symptoms (table 1). There were 21 patients (60%) with vascular risk factors, of whom 11 (31.4%) had more than one risk factor. Median hospitalisation stay was 3 days.

There were no statistical differences in demographic variables, discharge psychiatric diagnosis, type of the symptoms, vascular risk factors, neuroimaging data, or length of stay between patients with left sided symptoms and those with right sided symptoms concerning (table 1). Patients with somatoform disorders had motor symptoms more frequently than did patients with anxiety disorders ($\chi^2 = 6.84$; $p = 0.02$).

DISCUSSION

In this study, 1.5% of the patients admitted to a stroke unit presented symptoms unexplained by stroke or other neurological disease and fulfilling the criteria for psychiatric diagnosis. Contrary to most of published studies, our results did not show a preponderance of left sided symptoms. In a meta-analysis of all studies describing patients with medically unexplained symptoms, Stone *et al* concluded that is not certain that the functional symptoms are more common on the left side than on the right side of the body. They found a preponderance of the left symptoms only in the studies in which the investigators were aware of the laterality hypothesis before performing the study.¹ As in that meta-analysis, we did not find a preponderance of left sided symptoms.

The younger median age and the female preponderance of patients with psychiatric conditions mimicking a stroke reflects the demographic characteristics of somatoform

Table 1 Differences between patients with a psychiatric diagnosis and with left and right sided symptoms

Variables	n (%)	Left group	Right group	p value
Demographics				
Age (median)	41.0	41.0	38.0	0.94*
Sex (F/M)	28 (80.0)/ 7 (20.0)	9 (81.8)/ 2 (18.2)	10 (71.4)/ 4 (28.6)	0.66†
Diagnosis				
Somatoform disorders	25 (71.4)	9 (81.8)	10 (90.9)	–
Somatisation disorder	14 (40.0)	4 (36.4)	7 (63.6)	1.00†
Conversion disorder	11 (31.4)	5 (45.5)	3 (27.3)	–
Anxiety disorders	7 (20.0)	2 (18.2)	1 (9.1)	–
GAD	4 (11.4)	1 (9.1)	1 (9.1)	–
Panic disorder	3 (8.6)	1 (9.1)	0	–
Other	3 (8.6)	–	3	–
Symptoms				
Paresis				
Total	26 (74.3)	9 (81.9)	12 (85.7)	1.00†
Left	10 (38.5)	–	–	–
Right	12 (46.2)	–	–	–
Bilateral	4 (15.4)	–	–	–
Involuntary movements				
Total	1 (2.9)	0	1 (7.1)	1.00†
Right	1	–	–	–
Sensory				
Total	17 (48.6)	4 (36.4)	7 (50.0)	0.69†
Left	4 (23.5)	–	–	–
Right	7 (41.2)	–	–	–
Bilateral	6 (35.3)	–	–	–
Visual				
Total	5 (14.3)	1 (9.1)	2 (14.3)	1.00†
Bilateral	5	–	–	–
Other				
Total	19 (54.3)	3 (27.3)	9 (64.3)	0.15†
Total				
Left	11 (31.4)	11	–	–
Right	14 (40.0)	–	14	–
Bilateral	10 (28.6)	–	–	–

*p value for Mann-Whitney U test (left group/right group); †p value for χ^2 test (left group/right group).
GAD, generalised anxiety disorder.

and anxiety disorders. One interesting finding was the high frequency of vascular risk factors, which increases diagnostic uncertainty and could explain the admission to the stroke unit. A major limitation of our study is the lack of use diffusion magnetic resonance imaging to exclude definitively the unlikely possibility of a concomitant ischaemic lesion.

We conclude that left sided laterality of symptoms cannot be used as a tool to establish a psychiatric diagnosis in patients with acute lateralised neurological symptoms.

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REFERENCES

- 1 Stone J, Sharpe M, Carson A, *et al.* Are functional motor and sensory symptoms really more frequent

- on the left? A systematic review. *J Neurol Neurosurg Psychiatry* 2002;**73**:578–81.
- 2 Rothwell P. Investigation of unilateral sensory or motor symptoms: frequency of neurological pathology depends on side of symptoms. *J Neurol Neurosurg Psychiatry* 1994;**57**:1401–2.
- 3 Ron MA. Somatisation in neurological practice. *J Neurol Neurosurg Psychiatry* 1994;**57**:1161–4.
- 4 American Psychiatric Association. Mood disorders. *Diagnostic and statistical manual of mental disorders*, 4th ed. Washington DC: American Psychiatric Press, 2002.
- 5 WHO MONICA Project Principal Investigators. The World Health Organization MONICA Project (monitoring trends and determinants in cardiovascular disease): a major international collaboration. WHO MONICA Project Principal Investigators. *J Clin Epidemiol* 1988;**41**:105–14.

BOOK REVIEW

Atlas of neuromuscular diseases

Edited by Eva L Feldmand, Wolfgang Grisold, James W Russell, Udo Zifko. Published by Springer Verlag, 2005, €198 (hardcover), pp 474. ISBN 3-211-83819-8

This book is titled an “atlas” and subtitled “a practical guideline”. I had the expectation of a heavily illustrated book of neuromuscular conditions with annotations. The book has been designed as a comprehensive coverage

of disorders of peripheral nerve (including cranial nerves), muscle, neuromuscular junction, and motor neurone. Each condition is outlined in note form, with particular emphasis on causation—which can be lengthy and repetitive. The style of the text is abbreviated and can be difficult to read. The précis can be misleading or inaccurate at times. A useful list of references is given.

It is not clear to me to whom this book would appeal. The text is probably too comprehensive and potentially misleading or confusing for students, and does not add much to those already informed. The number and quantity of the illustrations is disappointing. I had expected more clinical illustrations of the conditions described. As a practical guideline it is not helpful and in particular the therapeutic suggestions are too abbreviated, incomplete, and uncritical for clinical application. Problems to be addressed in the next edition include the use of the same nerve biopsy illustration for patients with chronic inflammatory demyelinating polyneuropathy and Charcot-Marie-Tooth disease, despite one image being rotated by 90 degrees; the illustration of text relating to genetically defined young onset spinal muscular atrophy with an elderly man who appears to have progressive muscular atrophy; and the spelling of fascioscapulohumeral (sic) dystrophy.

The authors point out that “no other book provides a complete overview in a structured and easily comprehensible pattern supported by figures and pictures”. There are, however, a number of excellent books related to diseases of muscle and nerve, some providing a wider range of clinical and pathological illustrations and a more critical review of clinical presentation, diagnosis, and treatment. At the present time the role claimed by this book remains unfilled.

R W Orrell

CORRECTION

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M S Welgampola, S M Rosengren, G M Halmagyi, *et al.* Vestibular activation by bone conducted sound (*J Neurol Neurosurg Psychiatry* 2003;**74**:771–8). This relates to calibration of the output of the bone conductor that was used. The audiometric calibrator was not fully aware of the specific units in which bone conductors are generally calibrated but rather was influenced by the units of the sound level meter (suitable for air conducted sound). The calibration procedure is normally performed purely for testing hearing, in which case the precise units used are not strictly relevant (and under conditions which are quite different from those that the authors used). The authors have now purchased the required equipment to allow calibration of the B71, and have provided a guide to the force levels obtained:

Bone conducted stimuli should be measured in units of dB FL (force level) as a ratio to a reference force of 1 μ N. These levels are 10–20 dB higher, depending upon frequency, than the values given as “SPL”. The standard parameter 500 Hz 7ms “112 dB SPL” tone burst stimulus, obtained with a 10 V peak to peak input, corresponds to an intensity of 127 dB FL (RMS).