Letters to the Editor


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Higher level gait disorders

SIR—Liston et al. propose a simple and practical classification for higher level gait disorders (HLGD) supported by objective measurements [1]. Until now the classification of gait apraxia is confusing and has largely been descriptive based on clinical observation. We present our data based on patients with gait disorders referred to our movement disorder clinic. We retrospectively analysed the case notes of 34 patients with a diagnosis of gait apraxia between 1993 and 1999. We classified the gait disorders according to Liston and Tallis [2] based on the description from the medical notes. We looked at the mode of presentation, co-morbidity, neuroimaging and the natural course.

The onset of gait disorder was gradual and most patients presented within 3 years. Twenty-four patients had vascular risk factors (Table 1). Eight patients had cognitive impairment at presentation. Twenty-three patients had mixed gait apraxia, 5 had ignition apraxia, 2 had equilibrium apraxia and in 4 patients we were unable to make a distinction based on the description available in the notes. Seven patients had a trial of levodopa without any clinical benefit. Neuroimaging by CT scan showed periventricular deep white matter ischaemia in 15 patients, cerebral atrophy in 14, basal ganglia infarcts in 4 and in one patient the scan was reported as normal. Nine patients needed institutional care within 18 months of diagnosis. Our study suggests that HLG D is not benign, as 25% of the patients needed institutional care in 18 months. Universally acceptable classification as suggested by Liston et al. could be a very useful tool for further research.

Table 1. Vascular risk factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>19</td>
</tr>
<tr>
<td>CVA/TIA</td>
<td>3</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>2</td>
</tr>
</tbody>
</table>

ECF impairment is common

SIR—We appreciated the recent article by Allen et al. [1] describing the ability of an executive measure [i.e. the Executive Interview (EXIT25)] [2] to identify patients with chronic obstructive pulmonary disease who may be non-adherent with their medication. Executive control functions (ECF) are robust determinants of such ‘real world’ functional capacities [3]. The EXIT25 has previously been shown to be a significant independent predictor of instrumental activities of daily living (IADL) [4], and level of care [4, 5] among elderly retirees, as well as treatment adherence in Human Immune Deficiency viral (HIV) infection [6]. Dymek et al. recently found that the EXIT25 independently accounted for 56% of the variance in the capacity of patients with Parkinson’s disease to understand the circumstances and choices associated with their treatment, and 45% of the variance in a ‘rational reasons’ standard of the capacity to give informed consent [7]. Allen’s finding, i.e. that the EXIT25 perfectly distinguished between elderly patients who can, and cannot, be taught to competently use inhalers, is consistent with these studies.

However, we wish to point out that the EXIT25’s threshold for impairment (i.e. 15/50) is not age adjusted. Instead, it was set to detect functional incapacity. The mean EXIT25 score for non-institutionalised, affluent, well-educated elderly retirees is about 12.5/50 [4]. Thus, although statistically ‘normal’ for their age, 38% will fail the EXIT25 at 15/50; compared with 25% of community dwelling young adults with schizophrenia [8], 19.9% of cancer patients presenting for radiotherapy [9], 42% of consecutively admitted medical inpatients [10], 59% of medical outpatients with Type 2 diabetes mellitus [11], and 62% of medical inpatients referred for psychiatric consultation [12]. Thus, ECF impairment sufficient to interfere with many aspects of health care delivery is likely to be quite common in medical settings. Moreover, in each of the studies cited above, Mini-Mental State Examination (MMSE) scores [13] were within the normal range. Thus, the MMSE should not be used as the sole determinant of cognitive impairment, especially if one wishes to detect potentially disabling ECF impairment in medical patients.

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References


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Letters to the Editor

Amiodarone and cirrhosis

SIR—In a recently published case report of pseudo-alcoholic hepatotoxicity secondary to amiodarone, Singhal et al. claimed that ‘severe hepatic toxicity and cirrhosis with low dose amiodarone has not been reported in the English language literature’ and mentioned it as an ‘exceedingly rare’ complication [1].

We would like to make a few comments. First and foremost, the entity of amiodarone hepatotoxicity mimicking alcoholic liver disease had been well characterised in the seminal paper almost two decades ago [2]. Thereafter, numerous cases of amiodarone-associated cirrhosis had been reported from English literature alone [3–13] although the exact incidence (obviously subject to ascertainment bias with varying liver biopsy rates) remained unknown. Similar to the case under discussion, the majority of amiodarone-associated cirrhosis complications were fatal. Among them, cumulative dose of amiodarone varied widely; in particular, several of the cases [6, 8, 9] had received similar or even lower doses of amiodarone (Table 1) than the patient described by Singhal et al.

While the evidence stands at odds with the authors’ statement that their case represented the first report of severe cirrhosis after low dose amiodarone, it does not preclude the scientific value of the work. That said, there is another important unanswered question. Readers would be puzzled as the authors alluded to the ultrastructural hallmark of lysosomal myelin figures (phospholipidosis) in the Discussion section, and yet failed to mention any electron microscopy finding in the case history. The latter would undoubtedly give weight to the diagnosis (albeit not pathognomonic) of amiodarone hepatotoxicity, as well as distinguishing from alcoholic liver injury.

Table 1. Case summary of cirrhosis after low dose amiodarone

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age (years)</th>
<th>Length of treatment (months)</th>
<th>Cumulative dose (g)</th>
<th>Comment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>79</td>
<td>33</td>
<td>200</td>
<td>Encephalopathy</td>
<td>Death</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>13.5</td>
<td>165</td>
<td>Encephalopathy, hepatorenal syndrome</td>
<td>Death</td>
</tr>
<tr>
<td>8</td>
<td>83</td>
<td>35</td>
<td>213</td>
<td>Peripheral neuropathy, corneal deposits</td>
<td>Non-fatal</td>
</tr>
<tr>
<td>9</td>
<td>77</td>
<td>12</td>
<td>202</td>
<td>Portal hypertension, inactive hepatitis B</td>
<td>Death</td>
</tr>
</tbody>
</table>

*Case under discussion.

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