Neuro-ophthalmic complications are common in GCA and are seen in approximately 70% of patients. Peri-orbital manifestations (Table I) are uncommon and can lead to a delay in diagnosis. Clinicians should be aware of typical and atypical features of GCA in order to intervene in time before permanent damage occurs.

Table 1. Orbital/peri-orbital features of GCA1-6

<table>
<thead>
<tr>
<th>Feature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbital infarction syndrome – acute blindness, ophthalmoplegia, orbital pain</td>
</tr>
<tr>
<td>Orbital pseudotumour – proptosis</td>
</tr>
<tr>
<td>Orbital apex syndrome – pain, proptosis, chemosis, restricted eye movements</td>
</tr>
<tr>
<td>Oedema around the orbit</td>
</tr>
<tr>
<td>Orbital inflammation – haemorrhagic chemosis, ophthalmoplegia, fever</td>
</tr>
<tr>
<td>Peri-orbital ecchymosis</td>
</tr>
</tbody>
</table>

References


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Orthostatic hypotension following acute intracerebral haemorrhage

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Abstract

Background: blood pressure regulation may be impaired following acute stroke. Typically, there is overactivity of the sympathetic nervous system and underactivity of the parasympathetic system resulting in transient hypertension. Orthostatic hypotensive responses in acute stroke are less well documented.

Case report: we present a case of severe persistent orthostatic hypotension (OH) following acute intracerebral haemorrhage in a previously fit and well man. Symptomatic OH persisted for 60 days post-stroke. No known causes of OH could be identified.

Conclusions: such profound and persistent orthostatic hypotension following an acute intracerebral haemorrhage has not previously been documented. The precise cause of this finding in the case described is unknown, but may have been due to impaired higher-level regulation of the autonomic nervous system. A conservative approach with prolonged physical methods proved successful in rehabilitating this patient without the need for pharmacological intervention.

Keywords: hypotension, orthostatic, stroke, physiotherapy, elderly

Case report

A 71-year-old right-handed male was admitted with an acute-onset right hemiplegia. His only past medical history was of glaucoma for which he was using carteolol eyedrops. He was an ex-smoker and consumed 28 units of alcohol weekly. He was fully alert with normal speech and no cranial nerve or visual field defects. Power was 0/5 in the right arm...
and 4/5 in the right leg. There was no sensory deficit. The supine blood pressure (BP) was 144/77 mmHg and all other routine examinations were unremarkable. All routine blood tests, inclusive of full blood count, renal function, liver function, glucose, clotting profile, erythrocyte sedimentation rate and lipid profile, were normal. The resting ECG and chest X-ray were also normal. A computed tomography brain scan performed on the day of admission revealed a large (5 cm diameter) intracerebral haemorrhage in the left parietal lobe with surrounding oedema, but no midline shift.

The patient was managed conservatively and remained in bed until day 10 post-stroke when physiotherapy was started. However, within minutes of standing he became pale, clammy and hypotensive. On day 12 passive tilt-testing was performed. In the supine position (0°) the BP was 130/70 mmHg and the heart rate (HR) was 60 bpm. Within 1 minute of tilting to 70° he became pale and dizzy, the systolic BP was unrecordable and he lost consciousness. Within seconds of lying flat he regained consciousness, all vital signs returning to normal including a supine BP of 130/70 mmHg.

Possible causes of orthostatic hypotension (OH) were sought. Repeat blood tests including cortisol and thyroid function tests were normal, a 24-hour ECG recording did not show any arrhythmia or heart block and the carteolol eye drops were changed to a preparation not containing a β-blocker. Thromboembolic disease stockings were precluded because of evidence of peripheral vascular disease demonstrated and prolonged bed-rest and immobility. It is also possible that the haemorrhage affected higher-level regulation of the normal autonomic response.

### Table 1. Tilt-testing day 25 post-stroke

<table>
<thead>
<tr>
<th>Time</th>
<th>0°</th>
<th>30°</th>
<th>40°</th>
<th>50°</th>
<th>60°</th>
<th>70°</th>
<th>90°</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP</td>
<td>155/78</td>
<td>164/81</td>
<td>151/82</td>
<td>156/85</td>
<td>158/84</td>
<td>149/87</td>
<td>137/81</td>
</tr>
<tr>
<td>HR</td>
<td>80</td>
<td>85</td>
<td>87</td>
<td>90</td>
<td>100</td>
<td>110</td>
<td>73</td>
</tr>
</tbody>
</table>

Patient comfort precluded BP recording at all angles of tilt.

BP, blood pressure; HR, heart rate.

Hypotension following acute stroke is uncommon. In a study involving 40 stroke patients who were subjected to 90° passive tilting, within 10 days of stroke onset no cases of OH were found [1]. In another study comparing 40 stroke patients with 40 non-stroke control in-patients at 2 days and 1 week post-admission, persistent OH occurred in <10% of either group on both assessments [2]. In contrast, Carlsson et al. found that OH occurred in 20% of stroke patients at 1-month follow-up [3]. In a more recent cohort study of 71 stroke patients, within 1 month of stroke onset 37 patients had OH at 3 days post-stroke, of whom two experienced near syncope [4]. This study included patients with pre-morbid medical conditions such as congestive cardiac failure and diabetes that may have contributed to OH. In only five of the 37 patients with OH could no obvious pre-morbid contributing factor be identified. There was no association between the occurrence of OH and the nature or site of the stroke. On discharge, of those patients with initial OH, 23 had fully recovered with no residual OH and the remainder had mild persistent OH.

Determining the precise cause or mechanism of OH following acute stroke is difficult. Premorbid conditions such as diabetes, ischaemic heart disease and hypertension may predispose to OH post-stroke [4]. Stroke patients also experience prolonged immobility and bed-rest leading to muscle weakness and cardiovascular de-conditioning. The consequent venous pooling and reduced cardiac output could also cause OH [5]. Factors leading to OH in our patient included possible subclinical ischaemic heart disease (evidence of peripheral vascular disease demonstrated) and prolonged bed-rest and immobility. It is also possible that the haemorrhage affected higher-level regulation of the normal autonomic response.
This case illustrates a severe and debilitating consequence of acute intracerebral haemorrhage that has not previously been documented. It highlights that a successful resolution of OH can occur using a conservative approach supported by physiotherapy with the emphasis on graduated postural methods of improving cardiovascular conditioning.

Key points
- OH following acute intracerebral haemorrhage is rarely documented.
- Screen for OH in all stroke patients before starting physiotherapy in the upright position.
- Exclude remedial causes of OH.
- Conservative management and physical methods can effect resolution of OH in this setting.

References

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Spindle cell sarcoma: a rare cause of a large abdominal mass

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Abstract

A 73-year-old man presented with a huge intra-abdominal mass. Initially a gastrointestinal stromal tumour (GIST) was diagnosed, but his tumour was subsequently classified as a spindle cell sarcoma. Difficulties in the classification of rare intra-abdominal tumours are discussed.

Keywords: spindle cell sarcoma, gastrointestinal stromal tumours (GIST), elderly

Case Report

A previously fit 73-year-old man was referred complaining of backache, abdominal discomfort and possible weight loss. A huge, solid, non-tender and non-pulsatile intra-abdominal mass, the size of a 28-week uterus, was found. Ultrasound examination showed an inhomogeneous midline mass containing small cysts. Abdominal CT confirmed the mass, with solid and cystic spaces within it (see Figure 1). No origin was identified but the liver, spleen, pancreas and adrenals appeared normal. Ultrasound-guided biopsy revealed necrotic tumour tissue, which stained positively with antibodies to CD34, vimentin and smooth muscle actin, but was negative for melanoma, mesothelioma, epithelial markers, and with antibodies to CD117 (also called KIT). A provisional diagnosis of gastrointestinal stromal tumour (GIST) was made and it was thought likely to be malignant owing to its large size and central necrosis. The tumour was judged to be surgically resectable. At laparotomy, the tumour was found to arise from within two leaves of the small bowel mesentery and involved a segment of the