This study shows that oesophageal varices (46.5%) were the commonest source of upper gastrointestinal haemorrhage, and that alcoholic cirrhosis was the leading cause of portal hypertension. Our findings are comparable to a previous report from Sri Lanka on this acute medical emergency [1].

Medication was the main cause (82.9%) of peptic ulcer related bleeding and only 21.1% of peptic ulcers were positive histologically for Helicobacter pylori infection. More than 50% of patients with peptic ulcer bleeding had been taking aspirin with or without clopidogrel. The number of NSAIDs users was small.

References

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To the Editors:
A case of possible familial Guillain-Barre syndrome

Familial Guillain-Barre syndrome (GBS) is rare. Occurrence in families and the association with HLA types have resulted in a genetic susceptibility being postulated [1,2,3]. In the absence of large scale epidemiological studies, isolated case reports of GBS within families support this hypothesis. We report a Sri Lankan father and daughter, who shared HLA types DR12 and DQ 6, 7 and developed GBS eleven months apart.

A 7-year old girl developed sudden onset lower limb weakness progressing to involve upper limbs. Within three days, she was unable to walk even with support. There was no ataxia, bladder or bowel involvement. Ten days prior to this illness she had bloody diarrhoea and stool culture grew Campylobacter jejuni. On examination she was conscious and oriented. Cranial nerves were normal. Cranial nerves were normal. There was generalised hypotonia, lower limbs were areflexic with grade 2 muscle power; upper limbs were hyporeflexic with grade 4 power. There was no sensory or autonomic involvement. On day 12 of the illness cerebrospinal fluid analysis was normal. Nerve conduction was compatible with acute motor axonal neuropathy (AMAN) type GBS. HLA typing found DR 12, 15, 51 and 52 and DQ 6 and 7. Over the next six months she made a complete recovery.

Eleven months previously, her father, aged 51 years, had developed back pain, numbness and weakness in lower limbs, progressing rapidly to loss of ambulation. Medical records showed that muscle power was grade 0 in lower limbs and grade 3 in upper limbs. Unilateral lower motor neuron type 7th nerve palsy was noted. Nerve conduction studies showed an acute inflammatory demyelinating polyneuropathy (AIDP) and cerebrospinal fluid showed a cytoprotein dissociation. HLA typing showed DR 12, 13, 52 and DQ 6 and 7. He received intravenous immunoglobulins and recovered completely by the time his daughter fell ill. There was no family history to support hereditary neuropathy with liability to pressure palsy.

Most reported cases of familial GBS are among Caucasians. The only previous case report from Asia we found was of two siblings of Japanese parentage who developed GBS within a few days suggesting an infective rather than a familial aetiology [4]. The largest reported survey for a familial incidence in Netherlands described twelve affected families. Common HLA types have been studied without a definitive association being established. We found HLA types DR12 and DQ 6, 7 to be identical in this father and daughter pair.

References


Horner's syndrome occurs due to damage to the ipsilateral cervical sympathetic chain. It is a rare complication of thyroidectomy. A 35-year old female underwent left hemithyroidectomy for a solitary thyroid nodule. She was euthyroid and had no history of previous neck surgery. The patient went home on the second postoperative day after uneventful recovery. One week later, the patient complained of inability to fully retract her left upper eyelid. On examination, she had left sided partial ptosis and meiosis. She had no features suggestive of impaired sweating or flushing on that side of the face. After 3 months, her symptoms had reduced, but she still had noticeable partial ptosis and meiosis. Histology of the removed specimen indicated a degenerating colloid nodule.

Horner's syndrome, originally described in 1869 [1], classically includes meiosis, partial ptosis, enophthalmos and anhidrosis. It follows the disruption of the fibres of the cervical sympathetic chain, which supply the radial muscle of the iris, the superior tarsal muscle (muscle of Mueller) and the sweat glands of the face.

Causes of Horner's syndrome include direct trauma to the neck, ischaemia of the brain stem, dissection of the carotid artery, thyroid and lung malignancy and iatrogenic causes. Among the last, Horner's syndrome has been noted following epidural analgesia, central venous cannulation, coronary artery bypass grafting, carotid endarterectomy, chest tube thoracostomy and tonsillectomy.

Horner's syndrome after thyroidectomy, is a rare complication, noted in less than 0.2-0.3% of cases [2]. In most cases the syndrome is incomplete, usually with the absence of vasomotor symptoms, as in our case. In one study 70% of cases reported permanent symptoms or incomplete recovery, although the follow up period was only 15 months [3].

The pathophysiology of Horner's syndrome following thyroidectomy remains unexplained. Theories include postoperative haematoma compressing the cervical sympathetic chain, ischaemia induced neural damage, stretching of the chain during retraction and direct damage especially in patients with anatomical variations [3, 4].

References