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IgG Anti-GT1a antibodies which do not cross react with GQ1b ganglioside in a pharyngeal-cervical-brachial variant of Guillain-Barré syndrome

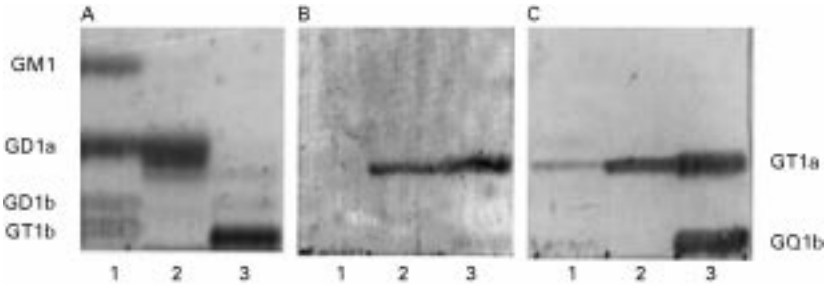
IgG anti-GT1a antibody which cross reacts with GQ1b ganglioside is associated with ophthalmoplegia in Guillain-Barré syndrome and in Fisher's syndrome, a variant of Guillain-Barré syndrome.[1] Pharyngeal-cervical-brachial weakness is another regional variant of Guillain-Barré syndrome originally described by Ropper.[2] Its symptoms resemble those of botulism and diphtheria and are characterised by marked oropharyngeal, neck, and shoulder weakness with areflexia only in the arms. We report on a patient with this variant who had high titres of serum IgG anti-GT1a antibody which did not cross react with GQ1b.

A 55 year-old woman was admitted due to sudden onset ptosis, trouble with swallowing, and nausea and vomiting. One week before admission she had had an upper respiratory infection with diarrhoea which lasted for several days. On admission, dilated pupils and ocular paralysis were noted. Fever of up to 40°C lasted for a week. Her neurological symptoms were at their worst on day 5. At that time, she was alert but could not open or move her eyes or swallow anything. Her pupils were dilated with sluggish light reflexes. The tip of her tongue did not protrude from her mouth. The Medical Research Council grades for the facial muscles ranged from 3 to 4; neck flexion and extension were graded 2; the deltoid and the biceps brachii and triceps muscles 4. Muscle strength in the lower limbs was normal. Deep tendon reflexes were absent in all the limbs, and the Babinski sign was negative. There was no sensory disturbance or cerebellar ataxia. She complained of headache, nausea, and vertigo on turning her head, but no meningeal signs were present. Her white blood cell count (15 600/μl) and serum C-reactive protein (9.0 mg/dl) were raised. Routine laboratory findings for liver and renal functions were normal. On day 5 her CSF had a raised protein concentration of 80 mg/dl, and cells were 7/μl. Motor conduction velocity in the right tibial nerve on Day 11 was slightly reduced (37 m/s), whereas the motor and sensory conduction velocities in the right medial nerve were normal. A stool culture was negative for *Campylobactor jejuni*. Botulism and diphtheria were excluded because her neurological symptoms occurred a week after the onset of diarrhoea. Results of the nerve conduction and CSF studies showed abnormal values.Guillain-Barré syndrome was diagnosed. A daily intramuscular injection (8 mg/day) of dexamethasone was given for 3 days then tapered off over the next 20 days. Two weeks after admission, her internal ophthalmoplegia and dysphagia had disappeared. One month later, ocular movements were almost normal, ptosis remained only in the right eye, the strength of the neck muscles had increased to 4, and deep reflexes became detectable. All these symptoms disappeared within 3 months of onset.

Serum antiganglioside antibody titres were determined using an enzyme linked immunosorbent assay (ELISA). On day 11, the titres of anti-GM1, GM2, GD1a, GD1b IgG, and IgM antibodies were <500; whereas the titre of the IgG anti-GT1a antibody had increased to 16 000 and that of the anti-GQ1b antibody was 500 (normal ranges were set at <500). No IgM antibodies to GT1a or GQ1b were detected. Thin layer chromatography with immunostaining confirmed that her serum IgG reacted with GT1a but not with GQ1b or GD1a (figure). An absorption study showed that her IgG anti-GT1a antibodies were not absorbed by GQ1b, GD1a, or GM1 (data not shown). Three months after onset, the respective serum titres of the anti-GT1a and anti-GQ1b IgG antibodies had decreased to 500 and <500.

The regional distribution of her symptoms was the same as that of the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome, except for the areflexia of the legs. Based on clinical observations of three patients, Ropper[2] described this variant as sparing the power and reflexes in the legs. One of his patients, however, had generalised areflexia. We therefore classified our patient as having the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome. Her headaches, nausea, and vertigo suggest the involvement of a central component.

Serum antiganglioside antibodies are present in patients with Guillain-Barré syndrome. Some of these antibodies are associated with certain clinical variants or signs of this syndrome. Chiba *et al*[1] detected IgG anti-GT1a and anti-GQ1b antibodies in most of a group of patients with Fisher's syndrome or Guillain-Barré syndrome with ophthalmoplegia and showed that the anti-GT1a antibodies in these patients cross reacted with GQ1b. Our patient with the pharyngeal-cervical-brachial variant of Guillain-Barré syndrome also had increases in IgG anti-GQ1b and anti-GT1a antibodies that paralleled the clinical course. The anti-GT1a antibody titre in our patient, however, was much higher than the anti-GQ1b antibody titre; moreover, the anti-GT1a antibody did not cross react with GQ1b. Mizoguchi *et al*[3] also detected IgG anti-GT1a antibodies which did not cross react with GQ1b, as well as anti-GD1a antibodies in the serum of a patient with this Guillain-Barré syndrome variant. Furthermore, O'Leary *et al*[4] reported IgG anti-GT1a and anti-GQ1b antibodies in three patients with acute oropharyngeal palsy. The presense of IgG anti-GT1a antibodies in these patients and in ours was associated with the emergence of acute polyneuropathy with marked lower cranial nerve involvement. IgG anti-GT1a antibodies which do not cross react with GQ1b may be closely related to the proclivity of Guillain-Barré syndrome to manifest oropharyngeal palsy or to the cause of its pharyngeal-cervical-brachial variant. The IgG anti-GT1a antibody present in Fisher's syndrome may recognise a structure common to GT1a and GQ1b gangliosides,[1] whereas in the pharyngeal-cervical-brachial variant it would react with another epitope specific to GT1a.



Thin layer chromatography with immunostaining. Thin layer chromatography plates stained with (A) orcinol/sulphuric acid for hexose; (B) IgG from our patient, subsequently stained by peroxidase conjugated antihuman IgG antibodies; (C) IgG from a patient with Bickerstaff's brain stem encephalitis. Lane 1: bovine brain ganglioside mixtures. Lane 2: the GD1a and GT1a fraction separated from bovine brain ganglioside mixtures by Q-sepharose column chromatography. Lane 3: fraction enriched with GT1a and GQ1b. The plates were developed with C-M-0.2% calcium chloride in water (5:4:1 by volume).

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Dexamethasone is not necessarily unsafe in primary supratentorial intracerebral haemorrhage

Controversy surrounds the role of steroids in the treatment of intracerebral haemorrhage.[1] Theoretically, the short term use of dexamethasone is justified because it lessens the damaging effects of cerebral oedema, decreases intracranial pressure, and strengthens the blood-brain barrier. However, the possible benefit has to be weighed against the risk of complications, especially infections and gastrointestinal haemorrhage.[2]