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Secondary exotropia following H_1N_1 viral infection ('swine' influenza)

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Abstract

Aim: **To present a case of secondary exotropia following H_1N_1 viral infection ('swine' influenza).**
Method: **The case of a 27-year-old woman with optic atrophy and secondary exotropia with previous history of H_1N_1 viral infection is presented.**
Result: **H_1N_1 infection led to optic atrophy and secondary exotropia.**
Conclusion: **Optic atrophy is a common cause for secondary exotropia; however, the H_1N_1 virus is a very rare cause for such optic neuropathy. Recent worldwide pandemics and immunisation programmes may increase the chance of clinical presentation.**

Key words: H_1N_1, 'Swine' influenza, Optic atrophy, Optic neuropathy, Secondary exotropia, Vaccination

Introduction

Recent pandemic threats from viruses H_1N_1 and H_5N_1 ('swine' and 'avian' influenza, respectively) provoked sizeable international media and public attention.[1] There are well-known, albeit very rare, potential neurological complications that may follow such infection or vaccination, including optic neuropathy, Guillain-Barré syndrome, meningoencephalitis, encephalomyelitis, polyneuropathy and peripheral neuritis.[2–10]

Secondary exotropia is a consequence of diminished unilateral or bilateral visual function where attenuated and disparate binocular simultaneous perceptions are severe enough to thwart their ultimate fusion. Secondary exotropia may result from a congenital or early-onset visual pathology; however, more commonly it is the result of an acquired defect of visual function in later life. A case is reported of a secondary exotropia in an adult resulting from unilateral optic atrophy following infection with the H_1N_1 virus.

Case report

In November 2009, a 27-year-old woman presented to the Accident & Emergency Department at Doncaster Royal Infirmary with a 1-week history of feeling unwell with 'flu-type symptoms that included a cough and chest/back pain. She had previously seen her General Practitioner who prescribed Tamiflu (Roche) and sent her home to rest. No recent foreign travel or relevant medical history was reported, specifically no previous 'swine' influenza vaccination.

Baseline investigations demonstrated a body temperature of 37.2 °C (normal), a resting pulse rate of 142 bpm (tachycardia) and brachial artery blood pressure fluctuating between a hypotensive 62/32 and a normal 96/60 mmHg. Glasgow Coma Score (GCS) was assessed at 14/14, respiration rate was a vigorous but shallow 40 per minute and blood-oxygen saturation was just 69% on air. Additional blood gases revealed severe hypoxia. An initial diagnosis of type 2 respiratory failure (respiratory failure caused by increased airway resistance) secondary to 'swine' influenza was given and the patient was admitted.

Over the next weeks the patient's condition deteriorated and she was transferred to the Extra-Corporeal-Membrane-Oxygenation (ECMO) centre in Leicester. After making sufficient progress within the ECMO, she was returned to Doncaster Royal Infirmary for continued in-patient care where she reported an acute loss of the vision in her left eye. The on-call ophthalmologist recorded ambiguous unaided visual acuities of Right: 'normal' and Left: 'NPL' (no perception of light). Pupil responses demonstrated a distinct left 'Marcus Gunn' relative afferent pupil defect (RAPD). Fundal examination of the right eye was unremarkable but the left was noted to have a 'pale optic disc'. No 'cherry red spot', retinal abnormality or retinal vascular issues were noted. A bedside diagnosis of anterior ischaemic optic neuropathy was made and the patient given an appointment to be reviewed in the out-patient eye clinic. Additional investigations such as CT/MRI scans (of the brain and visual pathway) and carotid artery competence assessments were requested in addition to other tests to aid in ruling out hyper-viscosity syndrome (in view of the patient's recent smoking cessation).

On attending the Eye Clinic, the patient reported a slight return of vision in her left eye and described this as 'a horizontal strip of vision with blur around'. Accurate unaided visual acuities of Right: 6/4-1 and Left: 6/38 (ph6/18) were attained along with a left RAPD but with no abnormality of the anterior segment or ocular media of either eye. A left pale and atrophic optic disc was documented and the previous scans and carotid artery investigations were clear of defects. After failing to keep a number of following appointments the patient returned

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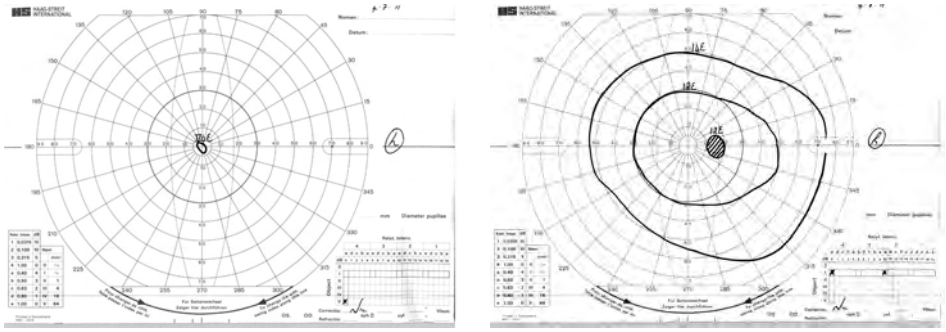

Fig. 1. Goldmann visual fields of a 27-year-old with left optic atrophy and secondary exotropia follow H_1N_1 infection ('swine' influenza).

5 months later with unaided visual acuities of Right: 6/5 and Left: 6/18. The left RAPD remained and Ishihara colour vision assessment reported 10/10 with the right eye but 0/10 with the left. The ocular diagnosis was amended to that of optic atrophy. Further appointments detailed no change in results or diagnosis until June 2011 when the left visual acuity was noted to have deteriorated to 6/60 alongside an acute left exotropia. Goldmann visual fields were produced showing an intact right visual field but a diminutive left field, extending just a few degrees in any single direction, even with the V4E target (Fig. 1).

Orthoptic examination in October 2011 recorded unchanged unaided visual acuities of Right: 6/5 and Left: 6/60. Cover test revealed a diplopia-free 30^{Δ} BI left exotropia at 1/3 m and 6 m and ocular motility was full. No evidence of binocular functions was demonstrable in either free space with prisms or using the synoptophore, even with larger slides. The absence of reported diplopia and binocular functions were considered the consequence of a severely restricted left visual field in addition to the compromised visual acuity.

Discussion

The rare association between H_1N_1 infection or its vaccination and ensuing neurological disorders such as optic neuropathy or Guillain-Barré syndrome has been previously scrutinised but the precise pathogenesis remains unknown.[2–10] With reference to optic neuropathy, the integrity of the nerve axon myelin sheaths has been hypothesised to be compromised by autoimmune systems.[11,12] It has been suggested that coexisting antigenic factors that interact with the H_1N_1 viral structure at the time of initial infection are also required. The requisite presence of two rare antigens perhaps offers a reason for the scarcity of post-infection or post-vaccination disorders. Some authors have speculated that a 'molecular mimicry' may be the cause, with antigens on infected pathogens resulting in the development of anti-ganglioside antibodies. This summation is based on the knowledge that these antibodies have an already proven pathogenesis within the neuropathy of Guillain-Barré syndrome.[3]

Conclusion

Secondary exotropia is a consequence of diminished unilateral or bilateral visual function where attenuated and disparate binocular simultaneous perceptions are severe enough to thwart their ultimate fusion. Secondary exotropia may result from a congenital or early-onset visual pathology, although more commonly, as demonstrated within the case presented, is the result of an acquired deficiency of visual function in later life. Optic atrophy is a common cause for secondary exotropia; however, the H_1N_1 virus is an extremely rare cause of optic neuropathy. Recent worldwide 'swine' influenza and 'avian' influenza pandemics and immunisation programmes thereof may increase the chance of clinical presentation.

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