ENCEPHALOMYELITIS AND BILATERAL OPTIC PERINEURITIS AFTER INFLUENZA VACCINATION

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SUMMARY
We report the case of one patient suffering from headache, urinary retention, bilateral optic disc swelling and a mild bilateral visual defect after influenza vaccination. The presumptive diagnosis was encephalomyelitis with bilateral optic perineuritis caused by influenza vaccination. We stress on the interest to search for this aetiology in unexplained optic neuropathy.

KEY WORDS
Influenza vaccination, optic neuropathy, optic perineuritis, encephalomyelitis.

RÉSUMÉ
Nous rapportons le cas d’un patient qui a présenté des céphalées, une rétention urinaire, un gonflement bilatéral des papilles accompagné d’un léger déficit visuel dans le décours d’un vaccin anti-influenza. Le diagnostic retenu est une encéphalomyélite avec une périnévrite optique bilatérale causées par le vaccin anti-influenza. Nous voulons attirer l’attention sur la recherche de cette étiologie en cas de neuropathie optique inexpliquée.

KEY WORDS
Influenza vaccination, optic neuropathy, optic perineuritis, encephalomyelitis.

MOTS CLÉS
Vaccin anti-influenza, neuropathie optique, périnévrite optique, encéphalomyélite.

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INTRODUCTION
Although well-known, the neurological complications after influenza vaccination are uncommon: they include Guillain-Barré syndrome, meningoencephalitis, encephalomyelitis, polyneuropathy and peripheral neuritis. Optic neuritis with visual loss after influenza vaccination has been infrequently reported in the literature: few cases of optic neuritis and two cases of anterior ischemic optic neuritis have been reported.

CASE REPORT
This 64 year old male patient received an influenza vaccination on October 1, 1996. Five days thereafter, he complained of headache and arthralgies, for which he was prescribed cefaclor. On October 20, he presented urinary retention, constipation and unsteadiness. Since the headache persisted, a fundus examination was performed on December 3. It revealed a bilateral swelling of the optic discs. The medical history included: hiatal hernia, hypercholesterolemia and arterial hypertension; the patient was on ranitidine and bisoprolol. He did not smoke nor consume alcohol. He had no record of allergies or exposure to toxic substances. Ophthalmological examination was performed by us on December 17, 1996. The visual acuity after correction was RE: 8/10 and LE: 9/10. The pupils were reactive to light and there was no relative afferent pupillary defect. Color vision was mildly abnormal: Ishihara plates were RE: 18/20 and LE: 19/20. A mild defect of contrast vision was elicited in both eyes with Pelli-Robson contrast sensitivity Chart-4L. Goldmann perimetry showed a bilateral enlarged blind spot. Anterior segment examination and intraocular pressure were normal in each eye. Visual evoked potentials were normal for both eyes. Fundus examination revealed a bilateral swelling and elevation of both optic discs with flame-shaped hemorrhages. Neurological examination revealed humerostyloradial and ankle hyporeflexia. Vibration was not perceived adequately at the malleolus bilaterally. Laboratory tests including erythrocyte sedimentation rate, C-reactive protein, complete blood cell count, plasma level of fibrinogen, protein C, protein S, glucose, hepatic function tests (serum chemistry/enzymes), VDRL-FTA ABs, Lyme titer, antinuclear antibodies, rheumatoid factor, angiotensin converting enzyme, vitamin B12 and folate levels were normal. Cholesterol was 225 mg/100ml. Chest X-rays were normal. Cerebral magnetic resonance imaging and angiographic resonance imaging were normal. A lumbar puncture was performed on December 26, 1996: cerebrospinal fluid (CSF) was clear with an opening pressure of 170 mmHg, the total protein level was 30 mg/dl, bacterial and viral cultures were negative, viral serology work-up was normal; in the CSF, IgG oligoclonal bands were detected that were not detected in the blood. Median and posterior tibial nerve somatosensory evoked potentials were normal. He was treated with intravenous methylprednisolone 1 g/day for 3 days. During the next months, ophthalmoscopic examination showed regression of the swelling of the optic discs. In July 1997, visual acuity was RE: 9/10 and LE: 10/10. Funduscopy revealed a bilateral moderate temporal pallor of the optic discs.

DISCUSSION
The presumptive diagnosis in our patient is post-vaccinal encephalomyelitis with bilateral optic perineuritis. The persistent headache, the urinary retention and constipation as well as the local synthesis of IgG were consistent with encephalomyelitis. The absence of inflammatory cells in the CSF may be explained by the delay between the onset of the symptoms and the lumbar puncture. The ophthalmological involvement was attributed to bilateral optic perineuritis. Optic perineuritis is an involvement of the optic nerve which, in a majority of cases, entails no other ocular symptoms or signs than disc swelling, that is usually bilateral (7). Optic neuropathy after influenza vaccination has been infrequently reported. A few cases of optic neuritis have been described (1, 2, 3, 8, 9). Although the number of reported cases is too small to draw any definitive conclusions, it would seem that only those patients who were treated by means of steroids experienced visual improvement. A case of bilateral optic nerve atrophy and blindness has been reported (6). Two patients were reported with bilateral anterior ischemic optic neuropathy following influenza vaccination (5).
The pathogenesis of postinfectious and post-vaccination neuropathy is not clearly understood. Hypotheses on the aetiology were suggested (4, 10). An autoimmune response caused by the distinct sequence homologies of myelin basic protein and the proteins of several viruses has been suggested. However, postviral and postvaccination neuropathy is relatively infrequent and sequence homology alone does not seem sufficient to cause neuropathy. Studies on experimental animal models prompted some authors to suggest that another antigenic factor interacts with the influenza viral antigens before immunoprocessing. The presence of that second antigen would be very rare at the time of vaccination, which could explain the low frequency of postvaccination neuropathy.

The cases of optic neuritis or ischemic injury to the optic nerve after influenza vaccination reported in the medical literature and our case of presumed optic perineuritis should remind us to investigate this possible cause in every patient who develops optic neuropathy.

REFERENCES


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