Optic neuritis (ON) is an acute disease of the optic nerve with demyelination, infectious disease or autoimmunity. ON presents with subacute monocular visual loss associated with pain during eye movement. Visual loss usually develops during hours or days. ON is mostly idiopathic and infrequently occurs on the background of autoimmune diseases, infectious diseases or vaccination. We report a lady with unilateral ON following influenza vaccination and successfully treated with intravenous methylprednisolone.
38-year-old woman presented with left retrobulbar pain and development of unilateral visual disturbance three days after receiving the administration of trivalent inactivated influenza vaccination. The patient had no history of autoimmune disease. Past medical history was unremarkable otherwise. The neurological examination of the patient was normal except visual disturbance. Fundus examination showed normal optic disc appearance. Best corrected visual acuities (BCVA) were 20/40 in the right eye (OD) and 20/120 in the left eye (OS). The anterior segment and media of each eye were normal. There was a relative afferent pupillary defect (RAPD) of grade 2 OS and color vision defect in both eyes (OU). Visual field testing showed an inferotemporal field defect OD and a superior hemifield defect accompanied with visual field constriction OS. Laboratory examinations were unremarkable for erythrocyte sedimentation rate, complete blood cell count, C-reactive protein, ANA (antinuclear-antibody), anti-double stranded DNA antibodies, anti-citrullinated protein antibodies, rheumatoid factor, complement components, antiacardiolipin antibodies, immunoglobulins (Ig) G, A and M, angiotensin-converting enzyme, anti-neutrophil cytoplasmic antibodies and aquaporin-4 antibodies. Microbiological tests were negative for hepatitis B and C viruses, human immunodeficiency virus, syphilis, latent tuberculosis, toxoplasmosis, toxocariasis, Bartonella henselae, Lyme disease, Herpes simplex virus and Varicella zoster virus. Cranial magnetic resonance imaging (MRI) revealed a high T2 signal intensity in the left optic nerve, but there were no pathological lesions in the cerebrum or spinal cord (Figure 1).

Examination of the cerebrospinal fluid (CSF) were normal without oligoclonal bands. The patient received intravenous methylprednisolone (1.0 g/day for 3 days), followed by oral prednisolone on day 4 of treatment, at 1 mg/kg/day tapered according to remission. The patient completely recovered after treatment with high dose intravenous methylprednisolone followed by a tapered dose of oral prednisolone. After 1 week, her best corrected visual acuities (BCVA) had improved to 20/25 OD and 20/30 OS. RAPD was absent, color vision was normal and visual field defects had almost completely resolved OU.

**DISCUSSION**

ON is a focal inflammation of the optic nerve. ON is mostly idiopathic and infrequently occurs on the background of autoimmune disease, infectious disease or inoculation with adjuvanted vaccines. Presumed post-immunization ON is rarely follows inoculations with Bacillus Calmette-Guerin and tetanus toxoid, pneumococcal and meningococcal group C polysaccharides, anthrax and various antiviral vaccines.1-6 Post-vaccination ON occurred most commonly after influenza vaccination (13 cases), followed by rabies (seven cases) and combined rubella-measles (five cases) vaccination.5,6

ON presents with subacute monocular visual loss associated with pain during eye movement. Visual loss usually develops during hours or days. All cases of post-vaccination ON occurred within 3 weeks after inoculation, even within several hours in two cases.5 Most patients report diffuse blurring or forgiving of vision. Recovery from typical ON usually begins within the first few weeks of symptom onset. An initial rapid recovery is followed by a slow improvement that can continue for up to a year after onset, with more than 90% of patients making a good visual recovery (20/40 acuity or better). Our case is consistent with the literature with regard to the interval between vaccination and disease onset and visual outcome. In our case, the di-
agnosis of ON was definite and there was no apparent evidence for other causes of visual loss. The symptoms developed after the vaccination and the temporal relationship is consistent with the literature.5,6

There are still many points that remain unclear in terms of the mechanism underlying the development of ON following influenza vaccination. Some authors suggested that anti-phosphatidylcholine (anti-PC) antibodies may be one of the causes of autoimmune vasculitis such as influenza vaccination-associated ON.8 Phospholipids are major structural components of vascular endothelial cells, the myelin sheath and the optic nerve. High serum titer of anti-phosphatidylcholine antibody levels were defined during acute phase in patients with ON. The two previously cited retrospective case-control studies evaluated the association between influenza vaccines and ON.9,10 Anti-phosphatidylcholine antibodies may be one of the causes of ON.

REFERENCES