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Main Article

Unilateral associated laryngeal paralysis due to varicella-zoster virus: virus antibody testing and videofluoroscopic findings

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Abstract

The relationship between varicella-zoster virus and idiopathic associated laryngeal paralysis was examined in five patients, using complement fixation or enzyme immunoassay testing. In all cases, significant changes in serum levels of varicella-zoster virus antibody were observed. Videofluoroscopy was useful in assessing the severity of the dysphagia and in making an accurate diagnosis; both laryngeal elevation and weakness of pharyngeal wall contraction were also observed. In two cases in which antiviral therapy was delayed, the outcome was poor, with increased levels of varicella-zoster virus immunoglobulin M found on enzyme immunoassay. The outcome of the condition may thus depend both on the speed of antiviral therapy commencement following onset of symptoms, and on the levels of varicella-zoster virus immunoglobulin M antibody (measured by enzyme immunoassay). Our study suggests that varicella-zoster virus should be considered in the differential diagnosis of patients with idiopathic associated laryngeal paralysis, and rapid antiviral therapy should be initiated when necessary.

Key words: Herpes Virus 3, Human; Laryngeal Nerves; Paralysis; Glossopharyngeal Nerve Diseases; Photofluorography

Introduction

Varicella-zoster virus persists, in its latent phase, in the ganglia of the spinal cord and cranial nerves, and its reactivation may cause various types of cranial nerve palsy. Ramsay Hunt syndrome[1] is well known, presenting with facial palsy, with or without auditory or vestibular involvement. Sometimes, it may be followed by glossopharyngeal nerve (IX) and/or vagal nerve (X) palsies.[2–11] However, similar palsies due to varicella-zoster virus, but without facial nerve palsy, have so far been reported only rarely, and the diagnosis in such cases is not always easy to make.[12–16] Generally, varicella-zoster virus mediated IXth and Xth cranial nerve palsies are included in the aetiology of associated laryngeal paralysis. However, if no other abnormalities are observed except for mild dysphagia and vocal fold paralysis, then a correct diagnosis may be overlooked and the condition mistaken for idiopathic recurrent nerve paralysis.

In this study, the relationship between acute, unilateral associated laryngeal paralysis (i.e. cranial nerve IX and/or X palsies) and varicella-zoster virus reactivation was serologically examined, and unilateral associated laryngeal paralysis was evaluated by videofluoroscopy.

Patients and methods

We examined cases of patients with acute associated laryngeal paralysis, but without any organic or abnormal findings on head and neck imaging, who were treated at our otolaryngology clinic from 2002 to 2004. Among the 15 cases demonstrating unilateral associated laryngeal paralysis, five cases with a serologically positive response for varicella-zoster virus were identified and analysed.

Each case was examined, and the presence of herpetic eruptions on the skin, pharyngeal mucosa or laryngeal mucosa was identified. An inspection of the lower cranial nerve findings was performed, including assessment of tongue mobility and fasciculation, and the presence of the curtain sign (i.e. a right or left shift in the pharyngeal wall on phonation). Tactile sensation was examined using a cotton swab, and taste sensation was evaluated using a paper filter disc technique. The presence of any abnormal mobility in the sternocleidomastoid and trapezius muscles was also

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tested, and vocal fold paralysis was identified by laryngofibre-optic examination.

Virus antibody assay

Complement fixation and enzyme immunoassay testing were performed, and immunoglobulin (Ig) G and IgM antibody titres for varicella-zoster virus were serially measured.

In addition, complement fixation and enzyme immunoassay testing was also undertaken for herpes simplex virus IgG and IgM, because herpes simplex virus can also produce lower cranial nerve palsies.

In principle, changes in the complement fixation test and IgG and IgM enzyme immunoassay antibody titre results were divided into three stages: an early stage, one week after onset; a late stage, two to three weeks after onset; and a recovery stage, more than four weeks after onset.

The complement fixation test titre was expressed at the largest dilution magnification which showed a positive finding, and a greater than four-fold increase (two tube differences) in the measurements at two different stages was considered to be a significantly positive result.

Enzyme immunoassay measurements were made using a commercially available analyser (Denka Seiken Co., Tokyo, Japan). An IgG value twice the normal level and an IgM value increase of over 0.8 were considered to be significantly positive.

The cerebrospinal fluid (CSF) cell count and protein, glucose and IgG levels were examined only for case one. In this case, varicella-zoster virus deoxyribonucleic acid (DNA) in the CSF was also examined by polymerase chain reaction.

Evaluation of swallowing

Using videofluoroscopy, pharyngeal wall contraction, laryngeal elevation and aspiration were evaluated by means of a 3 ml barium swallow.

Treatment and outcome

Acyclovir (4000 mg/day) and prednisolone (30 mg/day) were administered for 12 days in all cases except for case three, as soon as a herpetic infection was suspected. We clearly recorded the date of the first examination following onset of associated laryngeal paralysis, and the date of antiviral therapy commencement.

The associated laryngeal paralysis was assessed, based on the laryngofibre-optic and videofluoroscopic findings, from the initial hospital visit to six months after presentation. The outcome of each case was evaluated as follows: 'excellent' for cases showing rapid recovery without therapy; 'good' for recovery within several weeks of onset; 'fair' for delayed recovery within six months of onset; and 'poor' for palsy remaining after six months.

Results

Five cases of unilateral associated laryngeal paralysis showing significant results for varicella-zoster virus testing were identified (Table I). Patients' mean age was 61 years (range: 53–72); they comprised four men and one woman. The chief symptom in all cases was either hoarseness or dysphagia.

No case showed skin eruptions (i.e. redness or blisters) in the auricle, external auditory canal or oral cavity, or on the limbs or trunk (either side). However, in case two, diffuse redness with white spots on the palsied side was recognised on the left mucous membrane from the epiglottis to the arytenoids, for one week. In all other cases, no abnormality was observed in the pharyngeal or laryngeal regions.

On examining lower cranial nerve function, all cases showed good tongue mobility without fasciculation. The curtain sign in all cases was positive, but the shift was slight in cases three and four. All cases except for case four showed a reduction in both pharyngeal tactile sensation and taste sensation in the posterior one-third on the palsy side. Bilateral mobility of the sternocleidomastoid and trapezius muscles was good in all cases. All cases demonstrated paramedian fixation of the unilateral vocal fold. As a result, cases one to three and five were determined to have unilateral palsy of the IXth and Xth cranial

TABLE I

CLINICAL CHARACTERISTICS OF FIVE UNILATERAL LARYNGEAL PALSY CASES

Pt no	Age (yrs)/sex	Symptoms	Herpetic eruption	Nerve palsies	Curtain sign	Pharyngeal sensation	Vocal fold fixation
1	53/F	Hoarseness Severe dysphagia	−	L IX, X	+	↓	+
2	72/M	Sore throat Hoarseness Dysphagia	+	L IX, X	+	↓	+
3	61/M	Hoarseness Mild dysphagia	−	L IX, X	±	↓	+
4	64/M	Hoarseness Mild dysphagia	−	R X	±	Normal	+
5	54/M	Sore throat Hoarseness Severe dysphagia	−	L IX, X	+	↓	+

Pt no = patient number; yrs = years; F = female; M = male; L = left; R = right; IX = glossopharyngeal nerve; X = vagal nerve; ± = unremarkable

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nerves, while case four demonstrated only palsy of the Xth cranial nerve.

Varicella-zoster virus serology

In case one, the serum complement fixation result for varicella-zoster virus decreased from 1:64 to 1:16, comparing the acute to the recovery stage, and the enzyme immunoassay result for varicella-zoster virus IgG increased from 39.2 to 81.9.

In case two, the serum complement fixation result for varicella-zoster virus decreased from 1:128 to 1:32, and the enzyme immunoassay result for varicella-zoster virus IgG increased from 36.1 to $\geq$128.

In case three, the serum complement fixation result for varicella-zoster virus also decreased from 1:128 to 1:16, but the enzyme immunoassay result for varicella-zoster virus IgG showed high values of 128 and 101 at both the late acute and the recovery stages, respectively (Figure 1a and 1b).

In the late acute stage in cases four and five, the enzyme immunoassay result for varicella-zoster virus IgM showed high values of 1.35 and 2.76, respectively (Figure 1c). In all cases positive for varicella-zoster virus infection, a significant change in complement fixation result and enzyme immunoassay IgG result, or the presence of IgM on enzyme immunoassay, were found. In all cases, enzyme immunoassay for herpes simplex virus IgG was positive. However, no significant increases were observed. These responses seemed to indicate a cross-reaction for varicella-zoster virus, and enzyme immunoassay for herpes simplex virus IgM was negative in all cases.

In case one, CSF analysis revealed: 24 cells per mm^3 (normal finding: less than five cells); protein 41 mg/dl; glucose 70 mg/dl; and IgG 4.4 mg/dl. In addition, varicella-zoster virus DNA was not detected in the CSF, using polymerase chain reaction.

Videofluoroscopic findings

Videofluoroscopy findings are shown in Table II. In all cases, no abnormality of the oral preparatory and oral propulsive phases was found, and the tongue was pushed backwards and downwards into the pharynx.

Cases one and five showed absence of the normal pharyngeal wall contraction on the palsy side and delayed laryngeal elevation of a half vertebra width during swallowing. As a result, not all 3 ml of the barium passed through the oesophagus aditus but instead remained in the bilateral piriform sinus, and some of the barium was thus aspirated.

In case two, weak pharyngeal wall contraction and delayed laryngeal elevation caused aspiration during swallowing. Such aspiration was observed during laryngeal descent (Figure 2).

Cases three and four demonstrated weakness of unilateral pharyngeal wall contraction during swallowing (Figure 3), and a small amount of barium remained in the piriform sinus on the paralysed side after swallowing.

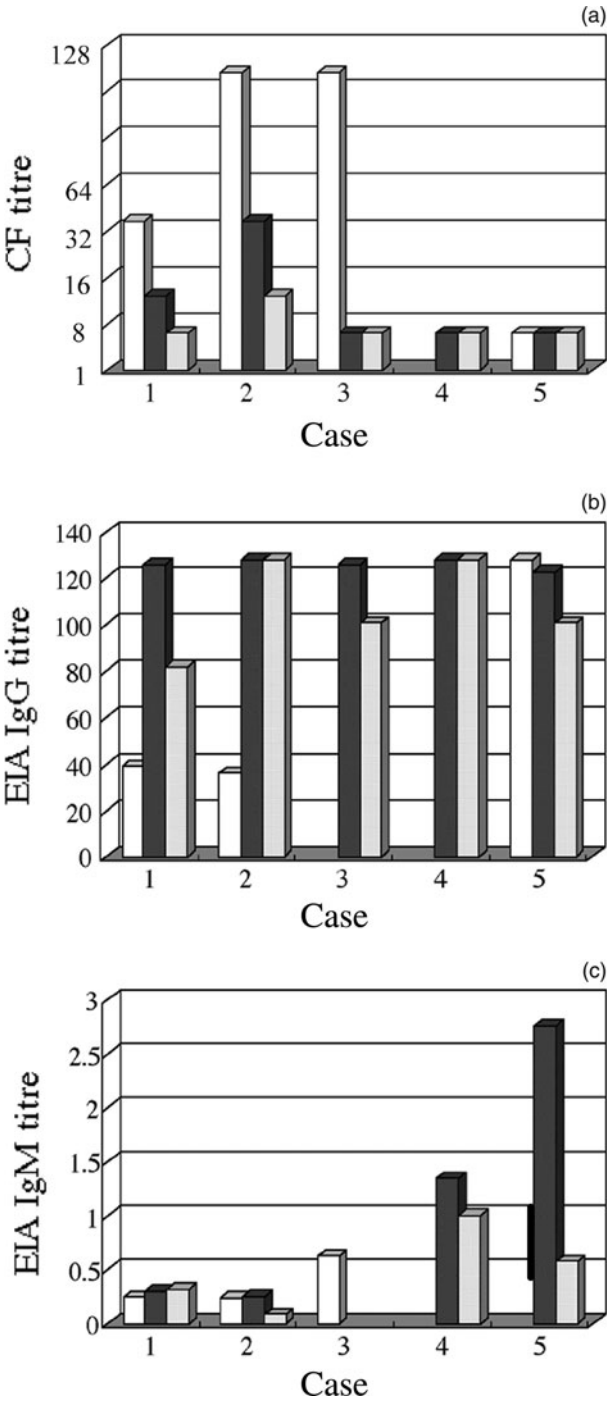

FIG. 1

Changes in varicella-zoster virus serology results. White bar = early acute stage (<1 week after onset); dark grey bar = late acute stage (<2–3 weeks after onset); light grey bar = recovery stage (>4 weeks after onset). (a) Varicella-zoster virus complement fixation (CF) test (cases 1, 2 and 3 show significant decreases over time, comparing the acute and recovery stages); (b) enzyme immunoassay varicella-zoster virus immunoglobulin (Ig) G test (cases 1 and 2 show significant increases over time); (c) enzyme immunoassay varicella-zoster virus IgM test (cases 4 and 5 show high values in the late acute stage, of 1.35 and 2.76, respectively).

Outcomes

For each case except for case three, antiviral therapy was administered. In cases one to five, following onset of unilateral associated laryngeal paralysis,

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examination occurred after: one day, three days, one week, two weeks, and two weeks, respectively. Following onset, antiviral therapy was commenced in cases one, two, four and five after: seven days, 10 days, three weeks and four weeks, respectively. In case five, antiviral therapy was delayed due to initial suspicion of a brainstem infarction.

In case one, vocal fold paralysis resolved following rapid recovery of pharyngeal swallowing over a four-week period; the outcome was thus judged to be good. Case two showed an improvement in vocal fold paralysis symptoms over an eight-week period, following rapid recovery from aspiration; this outcome was thus also classified as good. In case three, vocal fold paralysis (comprising weak pharyngeal wall contraction) rapidly recovered over a three-week period before varicella-zoster virus was diagnosed, and thus the outcome was considered to be excellent. In case four, weak pharyngeal wall contraction gradually recovered, but vocal fold paralysis persisted for longer than six months; this outcome was thus classified as poor. In case five, vocal fold paralysis recovered, following rapid recovery of pharyngeal swallowing, over a six-month period; the outcome was thus judged to be fair.

Discussion

Our study analysed five cases of associated laryngeal paralysis due to varicella-zoster virus. Associated laryngeal paralysis is noted to involve various combinations of palsy of the IXth and Xth cranial nerves, and also other types of palsy of the lower cranial nerves. However, our five cases showed unilateral palsy of the IXth and Xth cranial nerves. In our first, second and fifth cases, either a brainstem infarction or Guillain–Barre syndrome was initially suspected. Serological analysis indicated a varicella-zoster virus infection, and dysphagia was evaluated by either laryngeal fibrescope or videofluoroscopy.

To our knowledge, unilateral palsy of the IXth and Xth cranial nerves due to varicella-zoster virus has only been reported in a few cases.[12–16] Engström and Wohlfart[11] have reported two cases of unilateral palsy in the IXth and Xth cranial nerves, together with Ramsay Hunt syndrome. Since then, several cases of associated laryngeal paralysis due to varicella-zoster virus, with or without Ramsay Hunt syndrome, have been reported by other authors.

In Ramsay Hunt syndrome, palsy of the VIIIth cranial nerve is often accompanied by spread of inflammation due to varicella-zoster virus reactivated in the geniculate ganglia,[9,12] as well as in the cochlea or vestibular ganglia.[2] Since, anatomically, the geniculate ganglia lies far from the IXth and Xth cranial nerves, our cases of varicella-zoster virus induced IXth and Xth cranial nerve palsy are thought to have been caused by the vagal varicella-zoster virus focus of the IXth and Xth cranial nerves, rather than by a geniculate focus, as in Ramsay Hunt syndrome. In addition, cranial magnetic resonance imaging revealed gadolinium enhancement of the jugular foramen[17] or the IXth and Xth ganglia.[18] On the other hand, Parker[19] reported that bilateral cranial nerve palsy due to varicella-zoster virus sometimes occurs. He considered such palsy might be caused by local meningitis, being followed by infection of the nerve root or more extensive neural involvement.

Five cases of IXth and Xth cranial nerve palsy due to varicella-zoster virus have been reported to involve pharyngolaryngeal herpetic eruptions.[12–14,16] In case two of our series, since the region of white spots was considered to be related to the dominant region of the internal branch of the superior laryngeal nerve, such lesions would seem to represent herpetic eruptions. Aitken and Brain[20] have reported Ramsay Hunt syndrome without any herpetic eruptions. Muroi *et al.*[21] have reported that herpetic eruptions in the pharyngolarynx disappear quickly, compared with those occurring on the skin. This would indicate that, in case 1, 3, 4, 5 without herpetic eruptions of Table 1, such lesions could not have appeared from the onset, or they might have already disappeared before the first examination. It therefore seems difficult to diagnose reactivation of varicella-zoster virus and to identify resultant cases of cranial nerve palsy, in the absence of active eruptions.

The significant changes in the serum complement fixation test and enzyme immunoassay IgG test for varicella-zoster virus in cases one, two and three, and the high value of the enzyme immunoassay result for varicella-zoster virus IgM in cases four and five, led us to diagnose a varicella-zoster virus infection. Aizawa *et al.*[22] reported variable patterns of varicella-zoster virus antibody in patients with Ramsay Hunt syndrome. In one case, the varicella-zoster virus antibody level was increased at the initial hospital visit. In a case of paralysis, the

TABLE II

VIDEOFLUOROSCOPIC FINDINGS AND OUTCOME IN FIVE UNILATERAL LARYNGEAL PALSY CASES

Pt no	Videofluoroscopic findings			1st examination*	Therapy begun*	Outcome (palsy duration)
	Contraction	Elevation	Aspiration			
1	Absent	Half	+	1 day	7 days	Good (4 wks)
2	Weak	Delayed	+	3 days	10 days	Good (8 wks)
3	Weak	Normal	−	1 wk	No therapy	Excellent (3 wks)
4	Weak	Normal	−	2 wks	3 wks	Poor (>6 mths)
5	Absent	Half	+	2 wks	4 wks	Fair (6 mths)

*Time delay from onset of laryngeal palsy. Pt no = patient number; contraction = pharyngeal wall contraction on palsy side; elevation = laryngeal elevation; half = laryngeal elevation of a half vertebra width; wk = week; mth = month

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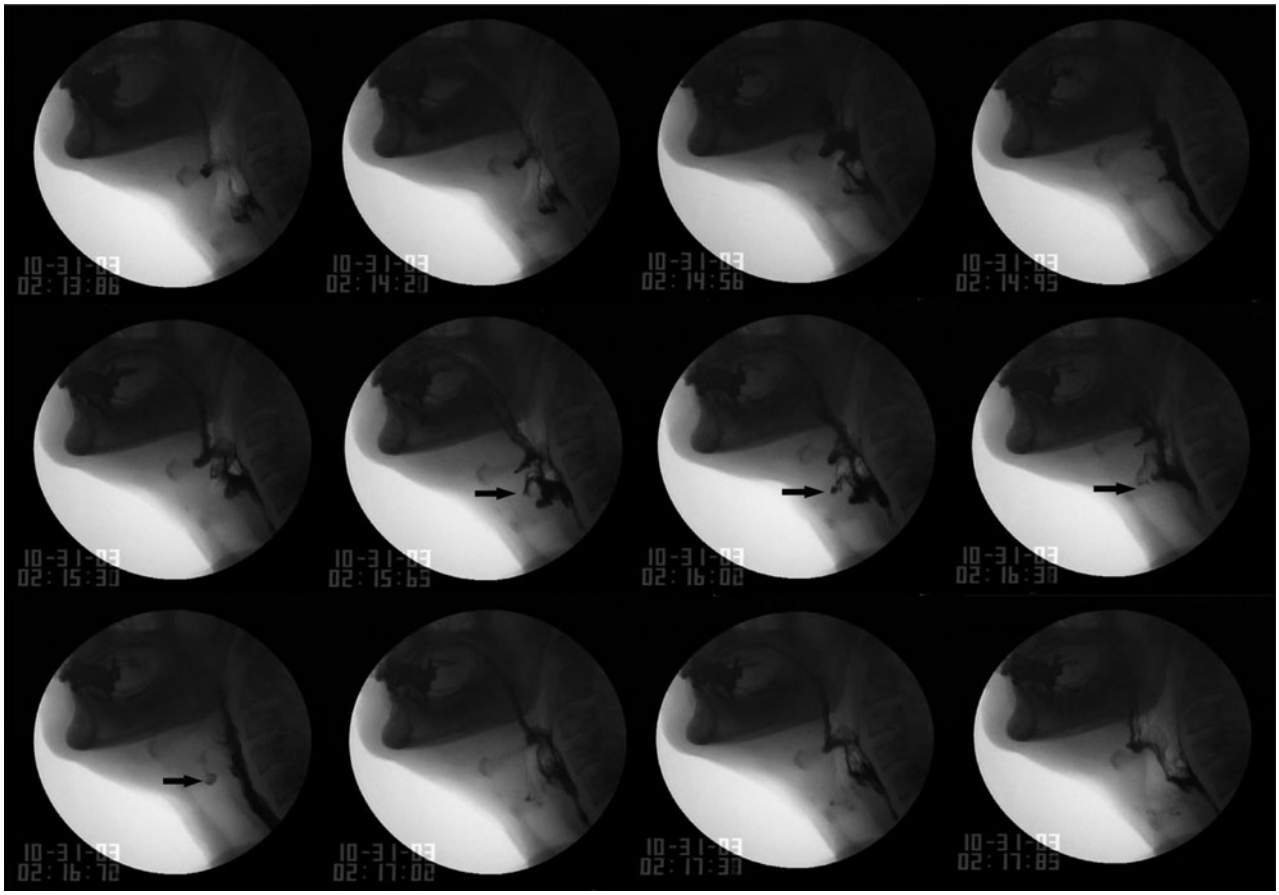

FIG. 2
Videofluoroscopy, lateral views, for case 2. Aspiration (arrow) is observed during laryngeal downward movement on the swallowing.

varicella-zoster virus antibody level at the onset of paralysis was low, but had increased by the time the paired serum was tested. Our cases one, two and three showed the former pattern, while cases four and five showed the latter pattern.

- **This was a study of varicella-zoster infection in patients presenting with glossopharyngeal and vagal palsies**
- **The authors present the pathological and clinical presentations, and argue for the use of antiviral therapy in such cases**

Based on the results from the serum complement fixation test and the enzyme immunoassay varicella-zoster virus IgG test, the laryngeal paralysis in cases one, two and three was diagnosed as being of varicella-zoster virus origin. These results indicate the necessity of serial serological testing in patients with idiopathic vocal fold paralysis. The high IgM values in cases four and five may suggest an increased varicella-zoster viral load. In addition, a serological response indicating no antibody titre fluctuation due to varicella-zoster virus reactivation can occur, as seen in our case three. It therefore appears important to evaluate the presence of varicella-zoster virus DNA, by polymerase chain reaction,[23–25] in saliva, blisters, CSF etc, before making a diagnosis.

In order to more precisely evaluate a palsy of the IXth and Xth cranial nerves, we employed videofluoroscopy[26] and examined pharyngeal wall contraction, laryngeal elevation and aspiration. Cases three and four seemed to show only unilateral recurrent laryngeal nerve paralysis, judging from an unremarkable curtain sign. However, videofluoroscopy demonstrated the existence of Xth cranial nerve palsy, based on the finding of weak unilateral pharyngeal wall contraction. In cases one, two and five, vocal fold mobility recovered more slowly than pharyngeal wall contraction and laryngeal elevation, while cases three and four showed no remarkable findings except for vocal fold paralysis. The recurrent nerve is thus considered to be the most vulnerable of all the pharyngolaryngeal branches of the Xth cranial nerve, because the recurrent laryngeal nerve travels a long distance from the Xth cranial nerve ganglion to the pharyngolaryngeal region.

Although the duration of laryngeal palsy in cases four and five was longer than that in the other

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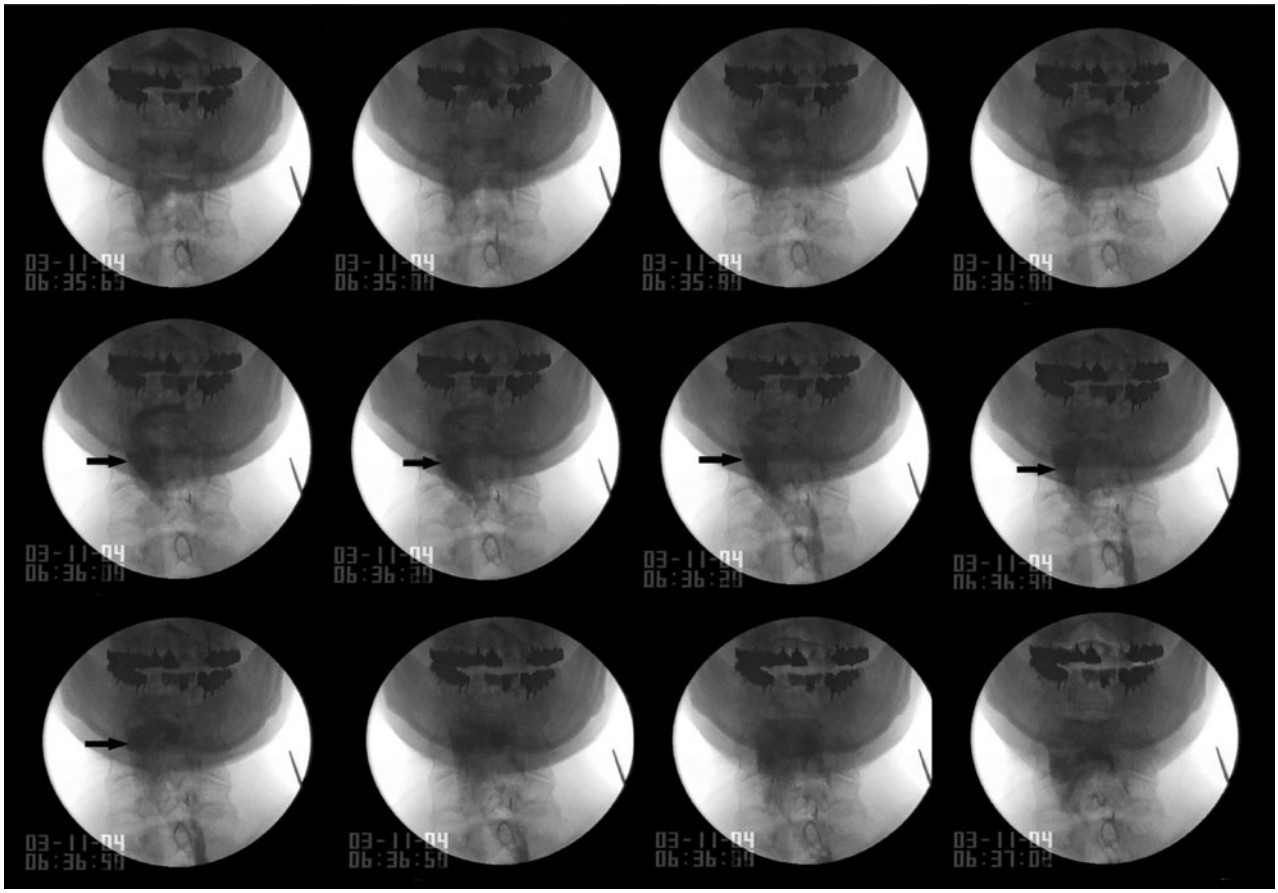

FIG. 3

Videofluoroscopy, anteroposterior views, for case 4. Weak pharyngeal wall contraction is observed for the right lateral wall (black arrow indicates right lateral wall).

cases, the primary symptoms in these cases were less severe than those of other cases, and the degree of the palsy was conversely milder (based on objective evaluation). In addition, in cases four and five, the period from onset of illness to start of therapy was longer than that of other cases, and the serum enzyme immunoassay varicella-zoster virus IgM titres on the first examination were higher than standard values. Therefore, the outcome of associated laryngeal paralysis may depend on both the timing of antiviral therapy commencement, and the enzyme immunoassay varicella-zoster virus IgM antibody level.

Conclusion

In cases of varicella-zoster virus cranial palsy in the earlier stages, it is necessary to suppress the spread of varicella-zoster virus and also to reduce the vicious cycle of oedema, constriction and ischaemia, by administering antiviral therapy and steroids.[27,28] Associated laryngeal paralysis may be due to various aetiologies, including demyelinating disorders, vascular disease and viral infection. An accurate diagnosis of varicella-zoster virus palsy may sometimes be missed due to the absence of skin eruptions, or to a stroke-like onset. When patients show unilateral palsy of the IXth and Xth cranial nerves with associated laryngeal paralysis, varicella-zoster virus should be considered in the differential diagnosis. Moreover, our study indicates that varicella-zoster virus serological tests and videofluoroscopy are useful, both in making an accurate diagnosis and in elucidating the pathophysiology of associated laryngeal paralysis due to varicella-zoster virus.

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