NEUROLOGICAL COMPLICATIONS OF VARICELLA ZOSTER REACTIVATION: A CASE SERIES

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Introduction

Varicella-zoster virus (VZV) causes a wide spectrum of neurological syndromes, with involvement of central or peripheral nervous system. Although VZV-related syndromes are well recognized, only rare cases are reported in literature.
Here, we describe two cases of cranial multinevritis and two of myeloradiculitis following VZV reactivation in immunocompetent patients.

Post-VZV Cranial Multinevritis

Case 1
Age: 69 years old
Sex: F
Past medical history: uneventful
Chief complain: right otalgia, followed by faringodinia, hypophonia, swelling difficulties and right facial nerve palsy.
Neurological examination: right facial nerve palsy, right hearing loss, right soft palate palsy, ataxic gait with right latero-deviation
Investigations:
•HIV: negative
•ORL evaluation: right hemi-larynx vesicular rash, right recurrent laryngeal nerve palsy
•Brain MRI: T1 contrast enhancement of tympanic duct and of tympanic tract of right facial nerve
•EMG: absence of voluntary recruitment from right facial nerve innerved muscles
•CSF: 20 lymphomonocitic cells/mm^3; albumin 37 mg/dl (v.n. 30); albumin transfer ratio 1,1%; VZV DNA not detectable
•pure tone audiometry: right neurosensory hypoacusia
Treatment: iv Acyclovir for one week followed by oral acyclovir and prednisone

Case 2
Age: 67 years old
Sex: F
Past medical history: uneventful
Chief complain: left otalgia and local vesicular rash, soon followed by left facial nerve palsy and left hypovisus
Neurological examination: left facial nerve palsy, left earing loss, horizontal right beating ny, positive Romberg sign, ataxic gait with right latero-deviation, worsened visual suppression
Investigations:
•HIV: negative
•Pure tone audiometry: sensoryneural hypoacusia
•ORL evaluation: left external acoustic meatus vesicular rash
•Brain MRI: negative
•VEP: bilateral increased cortical latency
•BAEP: left increased latency
•CSF: 240 lymphomonocitic cells/mm3; albumin 59 mg/dl (v.n. 10-30); transfer ratio 1,5%; VZV DNA: 520 copies/ml;
Treatment: iv Acyclovir, followed by oral acyclovir and prednisone

Post-VZV Myelo-radiculitis

Case 3
Age: 63 years old
Sex: M
Past medical history: recent surgical treatment of intestinal sub-occlusion
Chief complain: right T2-T3 VZV reactivation treated with oral Acyclovir, followed after ten days by paraesthesia at both upper limbs, soon extended to the thorax and lower limbs, and motor impairment at left lower limb
Neurological examination: hypo/dysesthesia involving the four limbs and thorax and for asymmetric tendon reflexes (sx>dx)
Investigations:
•HIV: negative
•Cervical spinal cord MRI: normal
•SEP: peripheral and cervical components not evocable from lower limbs
•CSF: 2 cells/mm^3; albumin 45 mg/dl, transfer ratio 1.3%; VZV DNA undetectable
Treatment: iv Acyclovir and steroid, followed by oral Acyclovir and prednisone

Case 4
Age: 80 years old
Sex: F
Past medical history: MGUS, hypertension, dyslipidaemia
Chief complain: left S3 VZV reactivation orally treated with Acyclovir
followed, after one month, by development of hypo/dysaesthesia at T10 level, extended to lower limbs, and urinary retention.
Neurological examination: severe proximal and distal, lower limbs weakness (MRC 3.5/5); hypoesthesia at lower limbs up to D11 level
Investigations:
•Routine blood exams: lymphocytopenia; HIV and CMV serology: negative
•Spinal cord MRI: normal
•EMG: signs of pyramidal tract involvement
•SEP: increased central latency from lower limbs
•VEP: increased cortical latency, OS > OD
•CSF: 4 lymphomonocytes/mm^3, CSF VZV DNA: not detectable; blood VZV DNA: 270 copies/ml
Treatment: iv Acyclovir and steroid, followed by oral Acyclovir

Conclusions

Our cases are examples of VZV-related peripheral and central nervous system involvement.
The absence of detectable viral DNA in the CSF can be explained by: i) previous treatment with orally administrated Acycovir, ii) the rapid clearance of VZV DNA from CSF, iii) imperfect diagnostic accuracy for CSF viral detection in real-life settings, with a negative predictive value of 82% and a positive predictive value of 54%.
The localisation of involved structures far from initial site of virus reactivation can be explained by transaxonal virus spreading along ganglionic afferent fibres to producing occlusion of small vessels with resultant microinfarction, but also rises the hypothesis of post-infectious immune mediated mechanisms.

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