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Recurrent polymorphonuclear pleocytosis with increased red blood cells caused by varicella zoster virus infection of the central nervous system:

Case Report and Review of the Literature

Aaron Haug^a, Ravi Mahalingam^a, Randall J. Cohrs^a, D. Scott Schmid^b, John R. Corboy^a, and Don Gilden^{a,c,*}

^a Department of Neurology, University of Colorado Denver School of Medicine, Aurora, CO, USA

^b National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, GA, USA

^c Department of Microbiology, University of Colorado Denver School of Medicine, Aurora, CO, USA

Abstract

We describe an immunocompetent 45-year-old woman who had four episodes of neurological disease (meningoencephalitis, multifocal vasculopathy, myelitis and inflammatory brain stem disease) produced by varicella zoster virus (VZV) over an 11-month period, all in the absence of rash. The cerebrospinal fluid (CSF) contained anti-VZV IgG antibody, but not VZV DNA throughout her illness, reaffirming the superiority of detection of anti-VZV IgG in CSF compared to VZV DNA in diagnosing VZV infection of the nervous system. Moreover, 3 of 7 CSF samples examined during the 11 months showed a VZV-induced pleocytosis consisting predominantly of polymorphonuclear cells (PMNs), and 4 of 7 samples also contained increased numbers of red blood cells (RBCs). Because increased PMNs and RBCs in CSF can also occur in patients with central and peripheral nervous system disease produced by cytomegalovirus (CMV), the differential diagnosis of chronic nervous system infection with increased PMNs and RBCs in CSF should include analyses for both VZV and CMV.

Keywords

VZV; Recurrent vasculopathy; Myelitis; Polymorphonuclear cells; Red blood cells

1. Introduction

The protean clinical manifestations of varicella zoster virus (VZV) infection of the central nervous system (CNS) include meningoencephalitis, multifocal vasculopathy and myelitis, all of which can occur without rash [1]. The cerebrospinal fluid (CSF) usually contains a mild

*Corresponding author: 12700 E. 19th Avenue, Mail Stop B182, Aurora, CO 80045, USA. Tel.: 1-303-724-4326; fax: 1-303-724-4329 don.gilden@ucdenver.edu.

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pleocytosis, mostly mononuclear, although PMNs occasionally predominate. In addition, the CSF may contain increased red blood cells (RBCs). We describe a patient who developed multiple episodes of CNS disease in the absence of rash, serologically confirmed to be caused by VZV, with increased PMNs and RBCs in the CSF on multiple occasions.

2. Case report

In November 2008, a 45-year-old woman developed confusion, an expressive aphasia and a mild spastic paraparesis with left-sided hyperreflexia. Brain magnetic resonance imaging (MRI) revealed bilateral non-enhancing cortical and subcortical T2 hyperintensities (Fig. 1) as well as leptomeningeal enhancement. The CSF contained 26 white blood cells (WBCs), 100% mononuclear cells (MNCs), 387 RBCs, protein 140 mg%, and glucose 58 mg%. CSF IgG was 24.1 mg%, which constituted 17.2% of total CSF protein (normal 3-13%), and there were no oligoclonal bands. PCR for VZV, herpes simplex virus (HSV-1 and HSV-2) and human herpesvirus-6 DNA was negative. No studies for antiviral antibodies in CSF were performed. CSF angiotensin-converting enzyme level was normal. Six days later, the CSF contained 24 WBCs, 100% MNCs, 2 RBCs, protein 83 mg%, and glucose 53 mg%. PCR was again negative for VZV, types HSV-1 and -2 DNA. CSF paraneoplastic antibody panel, VDRL and cytology were negative. CSF IgG was 14.4 mg%, which constituted 17.3% of total CSF protein. Brain biopsy of the gray matter, white matter and meninges showed no evidence of inflammation, granulomas or vasculitis. She was treated with acyclovir, 10 mg/kg intravenously 3 times daily for two days and oral prednisone 40 mg daily for three months, but when MRI and PCR revealed lack of evidence of HSV encephalitis, the acyclovir was discontinued. At discharge, neurological signs consisted of mild expressive aphasia, mild paraparesis, increased deep tendon reflexes (DTRs) in the legs and decreased sensation to all modalities in the right leg. In January 2009, brain MRI revealed decreased leptomeningeal enhancement along with a new T2 hyperintense signal in the periaqueductal gray matter. In March 2009, neurological signs consisted of mildly impaired recall, mild right leg weakness and diffuse hyperreflexia.

On 6-30-09, the patient developed acute low back pain. Neurological examination revealed marked tenderness to light palpation along the entire spine, but no other signs. MRI of the thoracic and lumbar spine showed an enhancing T2-hyperintense lesion from T2-11 and mild spinal cord swelling. Throughout the day, she developed increasing leg weakness and urinary retention. Neurological examination revealed a spastic paraparesis, sensory loss to all modalities from T3-11 on the left, hyperactive DTRs in the legs and a left extensor plantar response. The CSF contained 2978 WBCs, 90% PMNs, 10% MNCs, 678 RBCs, protein 247 mg%, and glucose 47 mg%. CSF bacterial cultures were negative. PCR for VZV, cytomegalovirus (CMV), HSV-1 and -2 DNA, and for enterovirus and West Nile virus was negative. She was treated with vancomycin, 1000 mg intravenously twice daily and ceftriaxone 2000 mg intravenously twice daily for 6 days and methylprednisolone 1000 mg intravenously daily for 3 days. Her back pain decreased in a few days, but her neurological deficits remained. On 7-3-09, the CSF contained 95 WBCs (22% PMNs, 72% MNCs, 5% eosinophils, 1% basophils), 24 RBCs, protein 74 mg%, and glucose 51 mg%. Bacterial cultures were negative. She was discharged on oral prednisone 60 mg daily and intermittent straight catheterization.

On 7-6-09, the serum from 6-30-09 was reported to contain anti-VZV IgM antibody, and she was immediately treated with acyclovir, 10 mg/kg intravenously three times daily for 2 weeks, and oral prednisone, 60 mg daily for 2 days. The CSF of 6-30-09 was also later reported to contain anti-VZV IgG antibody, but not anti-HSV IgG or anti-CMV IgG antibody, and the serum/CSF ratio of anti-VZV IgG antibody was reduced (ratio 2.6:1) compared to albumin (ratio 89:1) and total IgG (ratio 151:1), consistent with intrathecal synthesis of anti-VZV IgG antibody. On 8-11-09, the neurological examination was normal.

On 8-20-09, she felt her “legs give out” and complained of diffuse numbness as well as neck pain radiating down the spine. Neurological examination revealed a decreased attention span, word-finding difficulties, a spastic paraplegia and extensor plantar responses. On 8-24-09, a brain MRI showed a new enhancing T2 signal in the meninges over the left superior frontal gyrus, and MRI of the cervical and thoracic spine revealed a diffuse T2 hyperintensity involving the entire cervical spine and the thoracic spine from T1-8 (Fig. 2). The CSF was xanthochromic and contained 48 WBC, 49% PMNs, 51% MNCs, 990 RBCs, protein 283 mg% and glucose 46 mg%. Bacterial cultures were negative, and CSF neuromyelitis optica antibody was negative. Although VZV PCR was negative, the CSF contained anti-VZV IgG antibody, but not anti-HSV IgG antibody. The serum/CSF ratio of anti-VZV IgG antibody was reduced (ratio 1.32:1) compared to albumin (ratio 19:1) and total IgG (ratio 65:1). She was treated with acyclovir 10 mg/kg intravenously three times daily for 3 weeks, methylprednisolone 1000 mg intravenously daily for 2 days and valacyclovir 1 gm daily for 2 months. Examination at discharge revealed a flaccid paralysis in both legs, decreased sensation to all modalities below T3, hyperreflexia in the arms with clonus at the wrists and bilateral extensor plantar responses. On 9-17-09, the CSF contained 8 WBCs, 100% MNCs, 29 RBCs, protein 94 mg% and glucose 46 mg%. PCR for VZV DNA was negative; the CSF contained anti-VZV IgG antibody, but not anti-HSV IgG antibody, and the serum/CSF ratio of anti-VZV IgG antibody was reduced (ratio 2.5:1) compared to albumin (ratio 68:1) and total IgG (ratio 141:1).

On 10-6-09, she developed bilateral arm numbness and weakness. Neurological examination revealed a mild receptive aphasia and spastic weakness of both arms; the lower extremity signs were unchanged. The CSF contained 6 WBCs, 98% MNCs, 0 RBCs, protein 136 mg% and glucose 51 mg%. PCR for VZV and CMV was negative; the CSF again contained anti-VZV IgG antibody, but not anti-HSV or CMV IgG antibody, and the serum/CSF ratio of anti-VZV IgG antibody was 2.2:1. Brain MRI showed increased signal involving the corticospinal tracts and ventral cervico-medullary junction. MRI of the cervical and thoracic spine revealed less spinal cord swelling and leptomeningeal enhancement. She was treated with acyclovir 10 mg/kg intravenously three times daily for 2 weeks and valacyclovir 1 gram twice daily for 2 months. Table 1 summarizes the CSF findings for this patient.

In 1989, at age 25, the patient had had a monophasic episode of demyelinating disease involving the brain and brainstem. On 7-1-89, a brain biopsy identified an acute inflammatory demyelinating lesion with relative axonal sparing. The inflammatory response consisted of perivascular lymphocytes, plasma cells and plasmablasts. There was a moderate reactive gliosis and monocytic plasmocytic infiltration into parenchyma with focal clustering of foamy macrophages. No further episodes of demyelinating disease ever occurred, MRI scanning 20 years later revealed no multiple sclerosis (MS) plaques, and the CSF did not contain oligoclonal bands. Thus, her neurological disease in 1989 was most likely acute disseminated encephalomyelitis. While VZV infection can be associated with disseminated encephalomyelitis [2], our patient's monophasic episode was not preceded by varicella or zoster. The patient was lost to follow-up, but reported the onset of generalized tonic-clonic seizures in 1995.

3. Discussion

We describe an immunocompetent 45-year-old woman who had four episodes of neurological disease over an 11-month period, all in the absence of rash. The initial attack was characterized by an acute meningoencephalitis and multifocal vasculopathy. Seven months later, she developed chronic myelitis. Six weeks after the onset of myelitis, she developed recurrent multifocal vasculopathy and increasingly severe myelitis that rendered her paraplegic. Two months later, she developed inflammatory brain stem disease. Anti-VZV IgG antibody with reduced serum/CSF ratios of anti-VZV IgG antibody was detected on four occasions, while

VZV DNA was not found at any time, once again demonstrating the greater usefulness of detection of anti-VZV IgG in CSF compared to VZV DNA in diagnosing VZV infection of the nervous system [3,4]. The presence of anti-VZV IgM antibody combined with repeated detection of anti-VZV IgG antibody in CSF reflects chronic active disease. In contrast, in a patient with recurrent VZV myelopathy, anti-VZV IgG antibody in CSF was detected only during acute episodes, but not during symptom free intervals [5]. VZV is well documented to cause myelitis, including recurrent myelopathy [5,6], along with coexisting meningoencephalitis and multifocal vasculopathy [7-9]. Furthermore, given that all neurological complications of VZV reactivation can occur without rash [1], and that about 37% cases of VZV vasculopathy develop without rash [4], including CNS disease in immunocompetent patients [3,10-14], it is not surprising that none of our patient's episodes of neurological disease was associated with zoster rash.

Of the 7 CSFs analyzed in our patient, the pleocytosis was predominantly PMNs on 3 occasions (90%, 22% and 49%, respectively); strikingly, when 90% of the CSF WBCs were PMNs, there were 2978 WBCs (Table 1). Other cases of CNS VZV infection with high PMN percentages have also been reported. Most similar to our patient was one with VZV encephalomyelitis and a CSF pleocytosis of 1760 cells of which 40% were PMNs [15]. Further, in a series of patients with VZV myelitis [6], patient 2 had a CSF pleocytosis of 296 cells with 85% PMNs, and patient 3 had a CSF pleocytosis of 500 WBCs with 67% PMNs and 8 days later 178 WBCs with 65% PMNs. Two patients with VZV multifocal vasculopathy had a pleocytosis of 34 WBCs with 79% PMNs [16,17], and 10 WBCs with 52% PMNs [18]. Moreover, 4 of our patient's 7 CSF examinations were marked by increased numbers of RBCs, notably on 8-25-09, when the CSF contained 990 RBCs and was xanthochromic and the patient became paraplegic (Table 1). Increased CSF RBCs have been found in VZV myelitis [6] and VZV multifocal vasculopathy [18].

Cytomegalovirus (CMV) can also cause a persistent pleocytosis in which PMNs predominate. The CSF of a patient with CMV meningoencephalitis had 202 WBCs with 68% PMNs [19], and the CSFs of two patients with progressive CMV polyradiculopathy had 112 WBCs with 80% PMNs [20] and 600 WBCs of which 50% were PMNs [21]. Among three other patients with CMV polyradiculopathy, 4 CSF specimens revealed a CSF pleocytosis of 1080 WBCs (96% PMNs), 693 WBCs (98% PMNs), 780 WBCs (86% PMNs) and 560 WBCs (63% PMNs), respectively [22]. The CSF of a patient with CMV transverse myelitis had 9 WBCs with 37% PMNs [23]. In two patients with CMV ventriculoencephalitis, the CSF contained 15 WBCs (33% PMNs) and 2530 WBCs (90% PMNs), respectively [24]. In one patient with both CMV and HSV infection, the CSF had 3400 WBCs with 84% PMNs [25], and in another patient with both CMV and hepatitis A infection, the CSF contained 226 WBCs with 24% PMNs [26]. While clinicians appreciate that the CSF of patients with acute viral meningitis and meningoencephalitis usually contains a predominance of PMNs for the first 1-2 days of illness [27], the occasional predominance of PMNs in patients with nervous system infections caused by VZV and CMV most likely reflects active disease, as seen in patients with chronic granulomatous disorders. Although MNCs usually predominate just as in CNS disorders produced by VZV and CMV, periodic acute flare-ups are associated with increased numbers of PMNs in CSF [28-30].

Increased RBCs are also found in CSF of patients with CMV disease of the nervous system. The CSF of the five patients mentioned above with respect to CMV polyradiculopathy contained 8700 [20], 360 [21], and 1130, 225, 170, and 70 [22] RBCs, respectively. In the patient with myelitis and coexisting CMV and HSV infection, the CSF contained 14,000 RBCs on one occasion and was "xanthochromic" the second time [25]. Finally, while increased RBCs are found in CSF of patients with acute HSV encephalitis, our focus here is on the diagnostic

value of increased RBCs and PMNs in patients with chronic progressive infection of the nervous system caused by two treatable herpesviruses.

Overall, this important case illustrates the protean manifestations of neurological disease produced by VZV in the absence of rash, the repeated superiority of detecting anti-VZV antibody in CSF compared to VZV DNA to diagnose CNS disease produced by VZV, and the usefulness of increased numbers of PMNs and RBCs in CSF as clues to diagnose chronic VZV infection of the nervous system. The presence of both increased PMN and RBC numbers in patients with chronic nervous system disease warrants consideration of VZV and CMV as possible causative agents, particularly since antiviral treatment can be curative even after months of disease [31]. Patients with VZV vasculopathy and myelitis are typically treated with intravenous acyclovir for 2 weeks based on category 3 evidence (i.e., opinions of respected authorities based on clinical experience, descriptive studies or reports of expert committees), but the determination of optimum dose, duration of antiviral treatment and benefit of concurrent steroid therapy requires prospective studies.

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References

1. Gilden D, Cohrs RJ, Mahalingam R, Nagel MA. Varicella zoster virus vasculopathies: diverse clinical manifestations, laboratory features, pathogenesis, and treatment. *Lancet Neurol* 2009;8:731–40. [PubMed: 19608099]
2. Mariotti P, Colosimo C, Frisullo G, Caggiula M, Della Marca GD, Valentini P, et al. Relapsing demyelinating disease after chicken pox in a child. *Neurology* 2006;66:1953–4. [PubMed: 16801673]
3. Gilden DH, Bennett JL, Kleinschmidt-DeMasters BK, Song DD, Yee AS, Steiner I. The value of cerebrospinal fluid antiviral antibody in the diagnosis of neurologic disease produced by varicella zoster virus. *J Neurol Sci* 1998;159:140–4. [PubMed: 9741397]
4. Nagel MA, Cohrs RJ, Mahalingam R, Wellish MC, Forghani B, Schiller A, et al. The varicella zoster virus vasculopathies: clinical, CSF, imaging, and virologic features. *Neurology* 2008;70:853–60. [PubMed: 18332343]
5. Gilden D, Nagel MA, Ransohoff RM, Cohrs RJ, Mahalingam R, Tanabe JL. Recurrent varicella zoster virus myelopathy. *J Neurol Sci* 2009;276:196–8. [PubMed: 18945446]
6. Gilden DH, Beinlich BR, Rubinstien EM, Stommel E, Swenson R, Rubinstein D, et al. Varicella-zoster virus myelitis: an expanding spectrum. *Neurology* 1994;44:1818–23. [PubMed: 7936229]
7. Kleinschmidt-DeMasters BK, Mahalingam R, Shimek C, Marcoux HL, Wellish M, Tyler KL, et al. Profound cerebrospinal fluid pleocytosis and Froin's Syndrome secondary to widespread necrotizing vasculitis in an HIV-positive patient with varicella zoster virus encephalomyelitis. *J Neurol Sci* 1998;159:213–8. [PubMed: 9741410]
8. Miyazaki Y, Riku Y, Goto Y, Mano K, Yoshida M, Hashizume Y. VZV vasculopathy associated with myelo-radiculoganglio-meningoencephalitis: an autopsy case of an immunocompetent 66-year-old male. *J Neurol Sci* 2008;275:42–5. [PubMed: 18757065]
9. Tavazzi E, Minoli L, Ferrante P, Scagnelli P, Del Bue S, Romani A, et al. Varicella zoster virus meningo-encephalo-myelitis in an immunocompetent patient. *Neurol Sci* 2008;29:279–83. [PubMed: 18810606]
10. Nagel MA, Gilden DH. The protean neurologic manifestations of varicella-zoster virus infection. *Cleve Clin J Med* 2007;74:489–504. [PubMed: 17682626]
11. Nau R, Lantsch M, Stiefel M, Polak T, Reiber H. Varicella zoster virus-associated focal vasculitis without herpes zoster: recovery after treatment with acyclovir. *Neurology* 1998;51:914–5. [PubMed: 9748063]

12. Häusler M, Schaade L, Kemény S, Schweizer K, Schoenmakers C, Ramaekers VT. Encephalitis related to primary varicella-zoster virus infection in immunocompetent children. *J Neurol Sci* 2002;195:111–6. [PubMed: 11897240]
13. Jacobus GH, Visser CE, Portegies P. Recurrent varicella-zoster virus myelitis in an immunocompetent patient. *Eur Neurol* 2004;52:121–2. [PubMed: 15319558]
14. Habib AA, Gilden D, Schmid DS, Safdieh JE. Varicella zoster virus meningitis with hypoglycorrhachia in the absence of rash in an immunocompetent woman. *J Neurovirol* 2009;15:206–8. [PubMed: 19255900]
15. Devinsky O, Cho ES, Petito CK, Price RW. Herpes zoster myelitis. *Brain* 1991;114:1181–96. [PubMed: 1648419]
16. Case Records of the Massachusetts General Hospital (Case 5 – 1995). *N Engl J Med* 1995;332:452–9. [PubMed: 7824018]
17. Gilden DH, Kleinschmidt-DeMasters BK, Wellish M, Hedley-Whyte ET, Rentier B, Mahalingam R. Varicella zoster virus, a cause of waxing and waning vasculitis: the New England Journal of Medicine case 5-1995 revisited. *Neurology* 1996;47:1441–6. [PubMed: 8960724]
18. Amlie-Lefond C, Kleinschmidt-DeMasters BK, Mahalingam R, Davis LE, Gilden DH. The vasculopathy of varicella-zoster virus encephalitis. *Ann Neurol* 1995;37:784–90. [PubMed: 7778852]
19. Chin W, Magoffin R, Frierson G, Lennette EH. Cytomegalovirus infection: A case with meningoencephalitis. *JAMA* 1973;225:740–1. [PubMed: 4352471]
20. Eidelberg D, Sotrel A, Vogel H, Walker P, Kleefield J, Crumpacker C. Progressive polyradiculopathy in acquired immune deficiency syndrome. *Neurology* 1986;36:912–6. [PubMed: 3012412]
21. Clifford DB, Buller RS, Mohammed S, Robinson L, Storch GA. Use of polymerase chain reaction to demonstrate cytomegalovirus DNA in CSF of patients with human immunodeficiency virus infection. *Neurology* 1993;43:75–9. [PubMed: 8380909]
22. Granter SR, Doolittle MH, Renshaw AA. Predominance of neutrophils in the cerebrospinal fluid of AIDS patients with cytomegalovirus radiculopathy. *Am J Clin Pathol* 1996;105:364–6. [PubMed: 8602619]
23. Miles C, Hoffman W, Lai C, Freeman J. Cytomegalovirus-associated transverse myelitis. *Neurology* 1993;43:2143–5. [PubMed: 8413984]
24. Torgovnick J, Arsura E, Lala D. Cytomegalovirus ventriculoencephalitis presenting as a Wernicke's encephalopathy-like syndrome. *Neurology* 2000;55:1910–3. [PubMed: 11134396]
25. Tucker T, Dix RD, Katzen C, Davis RL, Schmidley JW. Cytomegalovirus and herpes simplex virus ascending myelitis in a patient with acquired immune deficiency syndrome. *Ann Neurol* 1985;18:74–9. [PubMed: 2994553]
26. Tyler KL, Gross RA, Cascino GD. Unusual viral causes of transverse myelitis: Hepatitis A virus and cytomegalovirus. *Neurology* 1986;36:855–8. [PubMed: 3010183]
27. Feigin RD, Shackelford PG. Value of repeat lumbar puncture in the differential diagnosis of meningitis. *N Engl J Med* 1973;289:571. [PubMed: 4723106]
28. Marinho SF, Paciullo VH, Fonseca MO, Khoury Z, Yamin MA, Minkoves R, et al. Persistent neutrophilic meningitis in a patient with the acquired immunodeficiency syndrome. *Rev Soc Bras Med Trop* 1997;30:241–5. [PubMed: 9273571]
29. Al Soub H, Almaslamani M, Al Khuwaiter J, El Deeb Y, Khatib MA. Primary nocardia meningitis in a patient without a predisposing condition: case report and review of the literature. *Scand J Infect Dis* 2007;39:737–41. [PubMed: 17654355]
30. Pinto VL Jr, Lima MA, Bóia MN. Persistent neutrophilic meningitis. *J Neurol Neurosurg Psychiatry* 2009;80:697–8. [PubMed: 19448101]
31. Gilden DH, Lipton HL, Wolf JS, Akenbrandt W, Smith JE, Mahalingam R, et al. Two patients with unusual forms of varicella-zoster virus vasculopathy. *N Engl J Med* 2002;347:1500–3. [PubMed: 12421892]

Fig. 1. MRI brain, T2 Flair axial. (A) Posterior hemispheric swelling and increased signal involving the left medial-occipital gyrus. (B) Gyriform swelling and increased T2 signal involving the right parasagittal parietal and occipital gyri. Left frontal lobe signal is scarring from previous biopsy.

Fig. 2. MRI cervical spine. (A) Sagittal T2 fast-recovery fast-spin echo sequence: abnormal T2 hyperintense signal and expansion involving the medulla and entire cervical spinal cord. (B) Sagittal T1 Flair post-contrast: abnormal leptomeningeal enhancement.

Table 1

CSF profiles.

Date	WBCs	% MNCs	% PMNs	RBCs	VZV DNA	Anti-VZV IgG antibody
11/03/2008	26	100	0	387	-	ND*
11/09/2008	24	100	0	2	-	ND
06/30/2009	2978	10	90	678	-	+
07/03/2009	95	72	22	153	-	ND
08/25/2009	48	51	49	990	-	+
09/17/2009	8	100	0	0	-	+
10/07/2009	6	98	1	0	-	+

* ND = not done.