

The value of detecting anti-VZV IgG antibody in CSF to diagnose VZV vasculopathy

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Abstract—Background: Factors that may obscure the diagnosis of varicella zoster virus (VZV) vasculopathy include the absence of rash before TIAs or stroke as well as similar clinical features and imaging, angiographic, and CSF abnormalities to those of other vasculopathies. Diagnosis relies on virologic confirmation that detects VZV DNA, anti-VZV IgG antibody, or both in the CSF. **Methods:** We reviewed our current 14 cases of patients diagnosed with VZV vasculopathy based on combined clinical, imaging, angiographic, or CSF abnormalities. All CSFs must have been tested for VZV DNA by PCR and for anti-VZV IgG antibody by enzyme immunoassay and found to be positive for either or both. Of the 14 subjects, 8 had a history of recent zoster, whereas 6 had no history of zoster rash before developing vasculopathy. **Results:** All 14 subjects (100%) had anti-VZV IgG antibody in their CSF, whereas only 4 (28%) had VZV DNA. The detection of anti-VZV IgG antibody in CSF was a more sensitive indicator of VZV vasculopathy than detection of VZV DNA ($p < 0.001$). **Conclusions:** In varicella zoster virus (VZV) vasculopathy, the diagnostic value of detecting anti-VZV IgG antibody in CSF is greater than that of detecting VZV DNA. Although a positive PCR for VZV DNA in CSF is helpful, a negative PCR does not exclude the diagnosis of VZV vasculopathy. Only when the CSF is negative for both VZV DNA and anti-VZV IgG antibody can the diagnosis of VZV vasculopathy be excluded.

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Primary varicella zoster virus (VZV) infection causes chickenpox (varicella). After varicella, virus becomes latent in ganglia along the entire neuraxis. Decades later, VZV may reactivate and cause shingles (zoster), which may be complicated by vasculopathy. VZV vasculopathy may also develop after varicella.

Typical clinical features of VZV vasculopathy include 1) rash with CNS disease; 2) MRI/CT abnormalities indicating cerebral ischemia/hemorrhage; 3) angiographic evidence of narrowing/beading in cerebral arteries; and 4) a CSF mononuclear pleocytosis. Diagnosis is not always straightforward as 1) many individuals with pathologically and virologically verified disease do not have rash; 2) neurologic disease usually develops weeks to months after rash, and TIAs or completed strokes are often attributed to arteriosclerotic disease rather than virus infection in cerebral arteries; and 3) other vasculopathies can produce the same neurologic symptoms, signs, imaging, angiographic, and CSF abnormalities.

Proof of VZV vasculopathy requires virologic confirmation by detection of VZV DNA or anti-VZV IgG antibody in CSF or both. Before PCR, detection of

antibody in CSF was used to diagnose VZV vasculopathy.^{1–3} Because PCR could be performed more quickly and proved to be extremely valuable to diagnose HSV encephalitis,⁴ clinicians used PCR, even to the exclusion of anti-VZV IgG antibody detection, to diagnose VZV vasculopathy. However, our analysis of patients with a presumptive diagnosis of VZV vasculopathy revealed that CSF did not always contain VZV DNA but did contain anti-VZV IgG antibody. This led us to compare the value of detecting VZV DNA to that of anti-VZV IgG antibody in CSF to diagnose VZV vasculopathy.

Methods. We analyzed our published and current unpublished cases for subjects with neurologic symptoms or signs, imaging, and angiographic or CSF abnormalities consistent with CNS vasculopathy. There were 14 patients, of whom 8 had zoster rash with neurologic symptoms and signs (table 1). The minimum workup in these patients included CT/MRI, a CSF exam, and virologic studies to confirm VZV. Of the six patients with neurologic disease without rash, the workup also included multiple studies to rule out coagulopathy, rheumatologic disorders, and other infectious causes of vasculopathy. Of the 14 patients, 3 had four-vessel angiography, 6 had MR angiography (MRA), 1 had both, and 4 had no vascular studies; the findings are listed in table 2. Of

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Table 1 VZV vasculopathy: Clinical and virologic features

Case no.	Age, y	Sex	Relevant underlying disorders	Rash	CSF pleocytosis	MRI/CT focal lesions	Angiographic or MRA abnormalities	CSF		Reduced serum/CSF ratio of VZV IgG	Ref.
								VSZ DNA PCR	VZV IgG		
1	18	F	None	—	+	+	ND	—	+	+	5*
2	54	M	AIDS	—	+	+	ND	+	+	+	5*
3	34	M	AIDS	+	—	+	ND	—	+	+	5*
4	28	M	HIV	—	+	+	—	+	+	+	6*
5	71	M	Leukemia	+	—	+	+	—	+	+	7*
6	76	F	None	+	—	—	—	+	+	+	7*
7	51	F	CREST syndrome	—	—	+	+	—	+	+	8*
8	56	F	None	—	—	+	+	—	+	+	†
9	77	F	None	+	—	+	+	—	+	+	†
10	52	F	None	+	+	+	+	—	+	+	†
11	52	M	HIV	+	+	+	—	+	+	+	†
12	76	F	Lymphoma	—	+	+	—	—	+	+	†
13	58	M	None	+	—	+	+	—	+	+	†
14	88	M	None	+	+	?‡	ND	—	+	+	†

* Published authors' cases.

† Unpublished authors' cases.

‡ MRI abnormality consistent with ischemic disease or head injury.

ND = not done; VZV = varicella zoster virus; MRA = MR angiography; CREST syndrome = calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia.

the 14 subjects, none had previous TIAs, heart disease, or diabetes. Disorders relevant to VZV reactivation are noted in table 1. Every subject received IV acyclovir (data not shown) and remained stable or improved except for Subject 2 who had AIDS and died despite acyclovir therapy. To confirm the diagnosis of VZV vasculopathy, the same CSF sample from every subject had to

have been tested for both VZV DNA and anti-VZV IgG antibody and found to be positive for either or both.

VZV DNA. CSF samples (>500 μ L) from all 14 subjects (table 1) were centrifuged at 300 *g* for 10 minutes and resuspended in 500 μ L of the supernatant. Smaller volumes (<500 μ L) were used directly as described.⁹ In brief, CSF samples were mixed with an

Table 2 VZV vasculopathy: Vascular studies and results

Case no.	Age, y	Sex	Angiography	Results	MRA	Results	Ref.
1	18	F	ND	ND	ND	ND	5*
2	54	M	ND	ND	ND	ND	5*
3	34	M	ND	ND	ND	ND	5*
4	28	M	ND	ND	Yes	Normal	6*
5	71	M	ND	ND	Yes	Normal	7*
6	76	F	ND	ND	Yes	Normal	7*
7	51	F	Yes	Stenotic MCA and PCA	Yes	Normal	8*
8	56	F	Yes	Multifocal MCA stenosis	ND	ND	†
9	77	F	ND	ND	Yes	Stenotic MCA	†
10	52	F	ND	ND	Yes	Multifocal MCA stenosis	†
11	52	M	ND	ND	Yes	Normal	†
12	76	F	Yes	Normal	ND	ND	†
13	58	M	Yes	Multifocal stenosis in MCA and VA; possible VA dissection	ND	ND	†
14	88	M	ND	ND	ND	ND	†

* Published authors' cases.

† Unpublished authors' cases.

ND = not done; VZV = varicella zoster virus; MRA = MR angiography; MCA = middle cerebral artery; PCA = posterior cerebral artery; VA = vertebral artery.

equal volume of 2× resuspension buffer (10 mM Tris-HCl pH 8.3, 0.9% NP-40, 0.9% Tween-20, and 240 µg/mL of proteinase K) and incubated at 56 °C for 1 hour. Proteinase K was inactivated at 95 °C for 10 minutes. Samples (40 µL) were then used for nested PCR using primers specific for VZV ORF 29 or HSV-1 LAT. This method detects one copy of VZV DNA per microgram of total DNA.

Anti-VZV IgG antibody. CSF samples from all 14 subjects (table 1) were analyzed for anti-VZV IgG antibody by enzyme immunoassay (EIA) at the Viral and Rickettsial Disease Laboratory of the California Department of Health Services.¹⁰ In brief, each CSF sample was diluted 1:20 and 1:200 and tested against VZV-infected and -uninfected cell lysates. An optical density index (ODI) was calculated for each sample, and an ODI greater than 1.00 at 1:200 dilution was considered positive for the presence of anti-VZV IgG antibody. For many years, this protocol has been used to test more than 1,600 CSF samples from subjects with clinical features of viral encephalitis for antibody to herpes simplex virus (HSV), VZV, Epstein-Barr virus, human herpesvirus-6, measles virus, mumps virus, western equine encephalitis virus, and West Nile virus. CSF samples that contained antibody to viruses other than VZV did not test positive for anti-VZV IgG antibody. With use of this EIA test, all CSF samples that were positive for anti-VZV IgG antibody in patients with clinical and laboratory features of VZV vasculopathy were found to be negative for anti-HSV IgG antibody. Furthermore, the EIA did not detect anti-VZV IgG antibody in normal human CSF. Finally, if anti-VZV IgG antibody was present in the CSF, intrathecal synthesis of anti-VZV IgG antibody must have been demonstrated by showing that the serum/CSF ratio of anti-VZV IgG antibody was reduced compared with the serum/CSF ratio of albumin and total IgG as already described.¹¹

Results. Based on combined clinical, imaging, angiographic, or CSF abnormalities, we identified 14 subjects with a presumptive diagnosis of VZV vasculopathy whose CSFs were tested for both VZV DNA and anti-VZV IgG antibody. Every subject was positive for either VZV DNA or anti-VZV IgG antibody or both, which confirmed the diagnosis of VZV vasculopathy. In addition, all CSFs were negative for control HSV DNA and anti-HSV IgG antibody. Of the 14 subjects, only 4 (28%) had VZV DNA in their CSF while all (100%) had anti-VZV IgG antibody in their CSF (table 1). The significance of detection of anti-VZV IgG antibody in the CSF was verified by the demonstration of intrathecal synthesis as described above. Based on a sample size of $n = 14$, the sensitivity of PCR as a test for VZV vasculopathy is 28% (4/14) with 95% confidence limits (8 to 58%) for binomial proportions. Based on a sample size of $n = 14$, the sensitivity of anti-VZV IgG antibody in the CSF as a test for VZV vasculopathy is 100% (14/14) with 95% confidence limits (77 to 100%) for binomial proportions. As a test for VZV vasculopathy, the sensitivity of detection of VZV IgG is greater than the sensitivity of detection of VZV DNA by PCR ($p < 0.001$) via McNemar test for correlated proportions.

Discussion. Our current analysis of 14 subjects with virologically verified VZV vasculopathy reveals that the detection of anti-VZV IgG antibody in the CSF is a more useful diagnostic test than detection of amplifiable VZV DNA. Furthermore, when anti-VZV IgG antibody was found in CSF, we calculated the serum/CSF ratio of anti-VZV IgG antibody and showed that the ratio was reduced vs the serum/CSF ratio of albumin and total IgG. Our demonstration of intrathecal synthesis of anti-VZV IgG antibody eliminated any possibility of contamination of the CSF by blood, which, in nearly all adults, contains antibodies to the virus. Overall, whereas a positive PCR is help-

ful, a negative PCR result does not exclude the diagnosis, and the detection of anti-VZV IgG antibody in CSF is the best test to diagnose VZV vasculopathy. Of our 14 subjects, if antibody screening had not been done, the diagnosis of stroke caused by VZV vasculopathy would have been missed in 10 (71%) of them.

The detection of anti-VZV IgG antibody in our 14 subjects was highly specific. The EIA test used herein has been performed on more than 1,600 CSF samples from subjects with migraine, epilepsy, as well as other infectious CNS disorders that turned out to be encephalitis caused by HSV, Epstein-Barr virus, human herpesvirus-6, measles virus, mumps virus, western equine encephalitis virus, and West Nile virus. None of these CSF samples contained IgG antibody to VZV. An additional control for specificity was provided by the demonstration that anti-HSV IgG antibody was not found in any of our 14 CSFs that were positive for anti-VZV IgG antibody. Finally, the demonstration of intrathecal synthesis of anti-VZV IgG antibody as described eliminates the possibility of false-positive detection of anti-VZV IgG antibody due to serum contamination.

Typically the accuracy of a test is determined by its comparison with a gold standard. In HSV encephalitis, the diagnostic value of PCR was shown by its comparison with HSV-positive brain biopsies, which has served as the gold standard for diagnosis. Unfortunately, in VZV vasculopathy, a gold standard has not been established. Nevertheless, compared with the common clinical practice of testing for VZV DNA in CSF only, testing for anti-VZV IgG antibody identifies more cases of VZV vasculopathy.

Another relevant issue is a comparison of the presence and persistence of VZV DNA and anti-VZV IgG antibodies in CSF of zoster patients with and without vasculopathy. To our knowledge, only one study has examined CSF for VZV DNA and anti-VZV IgG antibodies and performed MRI in multiple zoster patients.¹² In 46 patients, a single CSF sample was taken from 24 patients 1 to 18 days after zoster, two CSF samples were taken from 11 patients 7 to 47 days after zoster, and three CSF samples were taken from 11 patients 31 to 171 days after zoster. VZV DNA was found in CSF from only 10 of 46 (24%) patients with zoster, only one CSF contained VZV DNA as late as day 15, and none of 12 initially PCR-negative patients became PCR positive in the second sampling 6 to 34 days later. Similarly, anti-VZV IgG antibody was found in 10 of 44 (23%) patients with zoster, and only 3 of these 10 CSFs were positive for anti-VZV IgG antibody 31 to 105 days after zoster. Importantly, one of these three patients had MRI abnormalities indicating CNS involvement; however, no information regarding CNS disease was provided for the other two patients. Unfortunately, there was no clear comparison of VZV DNA and anti-VZV IgG antibody in the CSF of zoster patients with and without vasculopathy. Nevertheless, only a minority of patients with zoster had VZV DNA and anti-VZV

IgG antibody in their CSF after resolution of rash. Thus, the detection of anti-VZV IgG antibody in CSF of all eight of our zoster patients with vasculopathy is remarkable. Even more significant is the fact that the CSF of all 6 of our 14 (43%) subjects with vasculopathy without any history of zoster rash also contained anti-VZV IgG antibody.

In seven additional cases of VZV vasculopathy described in the literature, the CSF was also analyzed for both VZV DNA and anti-VZV IgG antibody and was positive for either or both; furthermore, when anti-VZV IgG antibody was present, intrathecal synthesis was confirmed.¹³⁻¹⁹ In five of seven of these cases, anti-VZV IgG antibody, but not VZV DNA, was found in CSF. Although those reports support our finding that detection of anti-VZV IgG antibody is superior to the detection of VZV DNA to diagnose VZV vasculopathy, the cases were not included in our analysis because of possible variability in PCR and EIA techniques among different laboratories.

Anti-VZV IgG antibody is found more often than VZV DNA in the CSF of most patients with VZV vasculopathy most likely because disease usually lasts for week to months. For example, clinical experience with acute HSV-1 encephalitis has shown that during the first week of disease, the CSF is positive for HSV-1 DNA by PCR and negative for antibody to HSV-1, whereas during the second week of disease, viral DNA begins to disappear from the CSF as anti-HSV-1 antibody begins to be detected.⁴ In our 14 subjects, the average time from onset of neurologic symptoms to virologic analysis was 4.8 months (data not shown); thus, it is not surprising that most subjects did not have VZV DNA in their CSF but did have anti-VZV IgG antibody.

Although the detection of anti-VZV IgG antibody in CSF appears to be superior to the detection of VZV DNA to diagnose VZV vasculopathy in adults, data are still insufficient for the pediatric population. Whereas numerous cases of stroke after varicella have been reported, only a few were tested for both VZV DNA and anti-VZV IgG antibody. Among these cases, three reports of VZV vasculopathy in children revealed only anti-VZV IgG antibody and not VZV DNA in the CSF,¹⁷⁻¹⁹ consistent with our current findings. In contrast, there are two other reports of VZV vasculopathy in children whose CSF contained VZV DNA but not anti-VZV IgG antibody. The first patient developed VZV vasculopathy 10 days after varicella; thus, it is not surprising that during acute disease, VZV DNA, but not anti-VZV IgG antibody, was found.¹⁵ In the second case, VZV vasculopathy developed 20 months after varicella.¹⁴ In this instance, the most likely reason for the presence of VZV DNA and not anti-VZV IgG antibody in the CSF is that VZV reactivated to produce vasculopathy without rash 2 years after varicella and virologic analysis was performed immediately.

The possibility exists that at the time of presentation of VZV vasculopathy, the CSF of some patients may be negative for both VZV DNA and anti-VZV

IgG antibody. For example, there is a report of a 16-month-old girl who developed seizures, acute focal neurologic deficits, and multiple infarcts on MRI without any history of varicella.¹⁸ Although virologic analysis did not detect VZV DNA or anti-VZV IgG in the CSF at the time of presentation, anti-VZV IgG antibody was present in the CSF 12 weeks later, and virologic confirmation was further substantiated by intrathecal synthesis of anti-VZV IgG antibody. Another report describes a 7-month-old boy who developed an intracranial hemorrhage secondary to focal arteritis in the left anterior cerebral artery 2 months after varicella²⁰; virologic analysis did not detect VZV DNA or anti-VZV IgG in the CSF at the time of presentation, and no biopsy or follow-up virologic analysis was performed on the CSF. Such unusual cases indicate the need to repeat virologic testing in cases of suspected VZV vasculopathy.

In our 14 subjects, there was no apparent correlation between the detection of VZV DNA or anti-VZV IgG antibody in the CSF with 1) a CSF pleocytosis, 2) imaging and vascular abnormalities, 3) large, small, or mixed cerebral artery involvement (data not shown), and 4) the presence or absence of zoster rash. However, analysis of a larger number of cases of VZV vasculopathy could reveal significant associations. Although some subjects with virologically verified VZV vasculopathy fulfill all four typical clinical criteria (a history of zoster rash followed by neurologic symptoms and signs of CNS disease, MRI/CT abnormalities, angiographic changes, and a CSF pleocytosis), most subjects with VZV vasculopathy do not have all four features. For example, 6 of our 14 (43%) subjects did not have rash, and 7 of 14 (50%) did not have a CSF pleocytosis. One of our patients (table 1, Subject 6) with posterior ciliary artery VZV vasculopathy after zoster rash had a negative MRI and vascular studies; the most likely explanation is that restriction of disease to a single small artery would not be associated with an abnormal MRI or angiogram. Another three subjects (table 1, Subjects 4, 11, and 12) had negative vascular studies and positive MRI; this is not surprising as the sensitivity of detecting stenosis in CNS vasculitis is only 62 to 79% for MRA²¹ and 90% for conventional angiography.²²

Finally, as six cases of VZV vasculopathy without rash came from analysis of CSFs of patients with vasculitis/vasculopathy of unknown etiology, the diagnostic workup of all such individuals should include CSF analysis not only for VZV DNA but also for anti-VZV IgG antibody. Future studies will be needed to determine the incidence of VZV vasculopathy among patients with vasculitis/vasculopathy of unknown etiology.

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