Original Article

Post-Varicella Neurological Complications: A Preliminary Observation from a Tertiary Care Centre of Eastern India

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Abstract

Objectives: The objective of this study is to analyse detailed clinical presentations, imaging findings, and outcome in a series of 17 cases ($n = 17$) with neurological complications following acute varicella infection. **Methods:** It is an observational study on the patients who presented to the neurology outpatient department of our institute with neurological abnormalities following acute varicella infection within the last 3 months. **Results:** Neuroimaging, either computed tomography or magnetic resonance imaging, cerebrospinal fluid analysis, electroencephalography and nerve conduction studies were performed in all the patients along with other specialized investigations as per clinical context. The age of presentation varied from childhood to middle age (median age was 23 years) and range of clinical spectrum was also wide. Peripheral nervous system involvement was more common in the form of Guillain–Barré syndrome (29.4%) and isolated lower motor neuron facial nerve palsy (23.5%) compared to central nervous system (CNS) involvement. CNS involvement was documented in the form of ataxia (11.76%), myelopathy (17.6%), stroke (5.88%) and encephalitis (5.88%). **Conclusion:** Chickenpox is a common viral disease and most patients recover without any complication. Although rare, neurological complications following acute varicella infection may have myriad presentations ranging from lower motor neuron facial palsy to life-threatening encephalitis. Compared to other studies, varicella encephalitis and ataxia were not so common in our study group. Response to therapy was uniformly good except in the patients presenting with ataxia. Response was particularly good to central and peripheral demyelinating disorders.

Keywords: Ataxia, Chickenpox, encephalitis, Guillain–Barré syndrome, myelopathy

Introduction

Chickenpox or varicella, caused by the varicella-zoster virus, is usually characterized by a generalized, itchy, and vesicular rash along with low-grade fever and malaise.[1] Although it is predominantly seen in children, adults are more often affected (15–20%) in tropical countries.[2]

After the primary affection, the virus may remain latent in the central nervous system (CNS) and later reactivation of the virus with replication may lead to zoster (shingles) in tissues innervated by the involved neurons. The zoster in adults is sometimes followed by persistent radicular pain, which is termed as post-herpetic neuralgia.[3]

Neurological complications are rare in acute varicella infection, observed in less than 1% of cases, and may include aseptic meningitis, meningoencephalitis, optic neuritis, cranial nerve palsies, cerebellar ataxia, Guillain–Barré (GB) syndrome and transverse myelitis.[4] Varicella is also an important risk factor for ischemic and haemorrhagic stroke, caused by vasculopathy. In children, one-third of ischemic arteriopathy is caused by varicella, while in adults the risk of stroke is increased by 30% within 1 year of zoster.[5]

There is dearth of literature about acute neurological manifestation of varicella infection. Therefore, we planned this study to document the spectrum of various neurological manifestations observed following acute varicella infection. The wide range of neurological complications following chickenpox demands a prompt diagnosis and focussed management to reduce the immediate complications and improve the long-term prognosis.

Material and Methods

Institutional ethics committee approval was obtained for this observational study. Data of 17 patients, who presented to the neurology outpatients department of our institute with neurological abnormalities following acute varicella infection within the last 3 months, were analysed. The study was conducted between September 2018 and February 2020. Patients with pre-existing neurological illness, neurological manifestation prior to development of

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rash and those who developed neurological complications after herpes zoster were excluded from our study. Detailed neurological history was obtained and examination was done as per the prefixed proforma. Patients were investigated with computed tomography or magnetic resonance imaging (MRI), cerebrospinal fluid analysis (CSF), electroencephalography and nerve conduction studies (NCS), and other specialized investigations as per clinical context. A multi-disciplinary approach was taken and the patients were managed accordingly in consultation with paediatric and radiology department of our institute. They were followed up for 3 months to assess outcome. As cases are heterogeneous and sample size is small for statistical analysis, disease groups with special emphasis on a few individual cases are being discussed.

Results

During 18-month period, total 17 patients qualified for recruitment (2 of them were below 12 years of age). The latency for the neurological complications varied from 8 to 38 days. The age range of the adult patients was 19–39 years, mostly below 30 years. The demographic and clinical profile of the study subjects is depicted in Table 1.

GB syndrome

We documented five cases of GB syndrome (Cases 1–5); all of them were adult males and one had associated human immunodeficiency virus (HIV) infection. All presented within 3 weeks (range 12–21 days) after the appearance of rash. Electrophysiological studies revealed presence of acute demyelinating polyradiculoneuropathy (AIDP) in two patients and acute motor sensory axonal neuropathy (AMSAN) in the rest. Except the immune-compromised patient, all had albumino-cytological dissociation in CSF. None of them received anti-viral therapy during the course of illness. All of them recovered well with intravenous immunoglobulin (IV-IG) therapy though one of them required mechanical ventilation. Case 3 was a 36-year-old HIV-1-infected gentleman, who was on anti-retroviral therapy (tenofovir, lamivudine and efavirenz), presented with double vision, unsteadiness of gait and dysphagia to liquids after 15 days of rash. After admission, he developed dimness of vision. Clinical examination revealed bilateral disc oedema, asymmetric bilateral abducens nerve palsy and palatal weakness with absent gag reflex, 3/5 power in all 4 limbs as per Medical Research Council (MRC) scale, areflexia and positive Romberg's sign. NCS revealed bilateral absent H-reflex and non-persistent F-waves. MRI with gadolinium contrast of brain, optic nerve and whole spine was normal. Visual evoked potential (VEP) study detected bilateral demyelinating retino-optic pathway dysfunction. He was treated with IV-IG 25 g daily for 5 days. He had improvement of vision within 14 days and motor power improved to 4/5 after 1 month. All other neurological abnormalities got reversed except the deep tendon reflexes which were present but diminished.

The clinical course of the GB syndrome patients in our study has been summarized in Table 2.

Facial nerve palsy

Four adult patients of isolated lower motor neuron (LMN) facial nerve palsy (Cases 6–9) presented between 8 and 11 days after the appearance of rash. Three out of four individuals were male and all were immunocompetent. Detailed otorhinolaryngological examination did not reveal any erythematous and vesicular rash of skin of ear canal, auricle and/or oropharyngeal mucous membrane. Glycemic status, HIV serology, skiagram of chest, serum angiotensin-converting enzyme, MRI brain and imaging of mastoid air cells were normal. Facial nerve NCS study was done on the day of presentation (in the 2nd week, between days 8 and 11 following the onset of facial palsy) and they revealed axonal pathology in one patient and normal pattern in the rest. All the patients were treated with oral valacyclovir 1 g for 7 days and oral prednisolone in tapering dose for 15 days. All of them had complete or near-complete functional recovery (House–Brackmann Facial Nerve Scores 7–8) except the male patient (case 9) with axonal pathology in NCS having significant residual weakness (House–Brackmann Facial Nerve Score 4) on follow-up.

Myelopathy

We documented three adult patients of myelopathy following chickenpox (Cases 10–12). Two relatively young patients, one male and one female, had acute transverse myelitis like presentation. Acute onset paraparesis, girdle-like sensation, exteroceptive and proprioceptive sensory loss below umbilicus and urinary retention all developed within 2 days; but they had no abnormality on neuroimaging or CSF analysis. Markers of collagen vascular disease and primary CNS demyelination were negative. Both patients improved significantly after intravenous methyl-prednisolone therapy. Case 10 was a 39-year-old female, after 21 days of development of vesicular rash (which resolved in 10 days), patient started developing weakness. Five days after onset of neuro-deficits, she noticed blurring of vision in her left eye with mild retro-orbital pain which lasted for a day. She took oral acyclovir 800 mg five times a day for 10 days. Clinical examination revealed left-sided disc oedema, normal other cranial nerves function, 0/5 power in both lower limbs as per MRC scale and areflexia in both lower limbs. Though power was 5/5 in upper limbs, limb ataxia and intention tremor along with bilateral gaze-evoked nystagmus were documented suggesting cerebellar dysfunction. Basic blood parameters were within normal limits. MRI with gadolinium contrast of brain, optic nerve, and whole spine was normal [Figure 1, Panel c and d]. CSF analysis showed 12 cells/mm^3 (all lymphocytes), glucose 62% of capillary blood glucose and protein 214 mg/dL. VEP detected bilateral demyelinating retino-optic pathway dysfunction (P100 latency of 150 ms on the left side and 130 ms on the right side). Anti-aquaporin-4 and anti-myelin oligodendrocyte glycoprotein (MOG) antibody were negative. Patient was initially treated with intravenous methylprednisolone at a dose of 1 g/day for 5 days followed by gradual tapering over 2 weeks. However, there was not much improvement. Patient was treated with plasmapheresis for five sessions, following which she showed marked improvement.

Table 1: The demographic and clinical profile of the study subjects

Age at presentation (years)/ gender	Latency from rash (days)	Clinical diagnosis	Neuroimaging	CSF study	Others	Treatment	Outcome at 3 months
23/Male	16	GB syndrome (MRS 4 at presentation)	MRI spine - normal	Albumino-cytological dissociation	NCS - AIDP	IV-IG	MRS 2 at discharge
19/Male	12	GB syndrome (MRS 5 at presentation)	MRI spine - normal	Albumino-cytological dissociation	NCS - AMSAN	IV-IG	MRS 2 at discharge, required mechanical ventilation
36/Male	15	GB syndrome (MRS 4 at presentation), external ophthalmoparesis, optic neuritis	MRI brain, orbit and spine - normal	Albumino-cytological dissociation	NCS - AIDP, HIV positive, CD4 count - 458	IV-IG	MRS 2 at discharge
28/Male	14	GB syndrome (MRS 3 at presentation)	MRI spine - normal	Albumino-cytological dissociation	NCS - AMSAN	IV-IG	MRS 2 at discharge
21/Male	21	GB syndrome (MRS 4 at presentation)	MRI spine - normal	Albumino-cytological dissociation	NCS - AMSAN	IV-IG	MRS 2 at discharge
22/Female	9	Right LMN facial nerve palsy	MRI brain - normal	Normal	None	Oral valacyclovir and prednisolone	House-Brackmann Facial Nerve Score 8
29/Male	8	Left LMN facial nerve palsy	MRI brain - normal	Normal	None	Oral valacyclovir and prednisolone	House-Brackmann Facial Nerve Score 7
32/Male	11	Right LMN facial nerve palsy	MRI brain - normal	Normal	None	Oral valacyclovir and prednisolone	House-Brackmann Facial Nerve Score 8
24/Male	9	Left LMN facial nerve palsy	MRI brain - normal	Normal	None	Oral valacyclovir and prednisolone	House--Brackmann Facial Nerve Score 4
39/Female	21	Dorsal myelopathy (MRS 4 at presentation)	MRI spine - normal [Figure 1; Panel c,d]	Albumino-cytological dissociation	Anti-aquaporin 4, anti-MOG - negative	IV methylprednisolone and plasmapheresis	MRS 2 at discharge
21/Female	16	Dorsal myelopathy (MRS 4 at presentation)	MRI spine - normal	Normal	Anti-aquaporin-4, and anti-MOG antibody - negative	IV methylprednisolone	MRS 2 at discharge
21/Male	14	Dorsal myelopathy	MRI brain with whole spine - normal	Normal	ANA, ENA profile, anti-aquaporin-4, anti-MOG - negative	IV methylprednisolone	MRS 2
25/Female	28	Cerebellar ataxia	MRI brain - normal	Normal	PET scan - negative	IV methylprednisolone	No improvement (MRS 4)
20/Female	38	Cerebellar ataxia	MRI brain - normal	Normal	PET scan - negative, autoimmune encephalitis profile - negative	IV methylprednisolone and IV-IG	No improvement (MRS 4)
9/Female	14	Right hemiparesis, focal seizure (MRS 5 at presentation)	MRI brain - left MCA territory infarct. MRA - left MCA narrowing [Figure 1; Panel a,b]	Cell count of 20 mm-3 and normal glucose and protein	ANA, ANCA, CRP - negative	Inj. acyclovir and Inj. levetiracetam	MRS 3 at discharge
32 years/ Female	23	ADEM	MRI brain - bilateral fluffy white matter opacity in brain and brainstem [Figure 1; Panel e,f]	Albumino-cytological dissociation, OCB - absent	ANA, ENA profile, anti-aquaporin-4, anti MOG - negative	IV methylprednisolone	MRS 3

Contd...

Table 1: Contd...

Age at presentation (years)/ gender	Latency from rash (days)	Clinical diagnosis	Neuroimaging	CSF study	Others	Treatment	Outcome at 3 months
6/Female	14	Known NMOSD on rituximab. Presented with encephalopathy, GTCS (MRS 5 at presentation)	MRI (previous) - demyelination in brain, cervical cord and bilateral optic nerve	Could not be done	None	Inj. acyclovir, Inj. meropenem, levetiracetam and midazolam	MRS 6

ADEM: Acute demyelinating encephalomyelitis, AIDP: acute inflammatory demyelinating polyneuropathy, AMSAN: acute motor sensory axonal neuropathy, ANA: antinuclear antibody, ANCA: antineutrophil cytoplasmic antibody, CRP: c-reactive protein, ENA: extractable nuclear antigen, GB syndrome: Guillain-Barré syndrome, IV: intravenous, IV-IG: intravenous immunoglobulin, LMN: lower motor neuron, MCA: middle cerebral artery, MOG: myelin oligodendrocyte glycoprotein, MRA: magnetic resonance angiography, MRI: magnetic resonance imaging, MRS: Modified Rankin Scale, NCS: nerve conduction study, NMOSD: neuromyelitis optica spectrum disorders, OCB: oligoclonal antibody, PET: positron emission tomography

Table 2: The clinical profile and course of the Guillain-Barré syndrome patients

	NCS	Duration of hospital stay (days)	Ventilator requirement	Cranial nerve involvement	Autonomic involvement	MRC sum score on presentation	MRC sum score on day 14	MRC sum score on day 28	MRC sum score on day 42
Case 1	AIDP	18	No	Yes	No	26	32	51	52
Case 2	AMSAN	31	Yes	Yes	Yes	8	14	23	32
Case 3	AIDP	20	No	No	Yes	25	43	49	54
Case 4	AMSAN	23	No	No	No	22	38	43	51
Case 5	AMSAN	19	No	No	No	36	49	52	57

AIDP: Acute inflammatory demyelinating polyneuropathy, AMSAN: acute motor and sensory axonal neuropathy, MRC: medical research council, NCS: nerve conduction study

Cerebellar ataxia

Two young females had acute cerebellitis which developed in 4 weeks and 5 weeks, respectively, after appearance of rash (Cases 13 and 14). None of them received anti-viral therapy during the illness. Acute cerebellitis was established by clinical clues.[6] One of them had ataxic dysarthria. The diagnosis was clinical, as the temporal association of ataxia with typical rash of chickenpox seldom requires further diagnostic testing.[7] Both had normal MRI brain and normal CSF study. Anti-thyroid peroxidase antibody and autoimmune encephalitis and paraneoplastic panel were negative. 18-Fluoro-deoxyglucose positron emission tomography whole body did not reveal any evidence of increased metabolic activity. They responded poorly to intravenous methylprednisolone at a dose of 1 g/day for 5 days followed by oral tapering over 2 weeks. One of the patients received IV-IG for 5 days without much improvement.

Stroke

Case 15 was a 9-year-old girl presented with right hemiparesis, dysarthria and focal seizure with impaired consciousness after 2 weeks of rash. Clinical examination revealed right-sided upper motor neuron type facial palsy, normal other cranial nerves function, 2/5 power in right upper and lower limbs as per MRC scale. Her MRI revealed narrowed M1 segment of left MCA with poor distal flow and infarct [Figure 1, Panel a,b]. The CSF showed 20 cells/mm3 (all lymphocytes), glucose 69% of capillary blood glucose and protein 44 mg/dL. Comprehensive work-up for autoimmune, vasculitis, or prothrombotic state did not reveal any abnormality. She was treated with injection acyclovir 10 mg/kg IV 8 hourly for 7 days and intravenous methylprednisolone at a dose of 500 mg/day for 5 days followed by gradual tapering over next 2 weeks along with physiotherapy. She recovered well over next few months (power of 4/5 as per MRC scale in the affected limbs at 90 days' follow-up), and at last visit, she had modified Rankin scale of 3 with improving vasculopathy.

Acute disseminated encephalomyelitis

Case 16 was a 32-year-old female, educated up to higher secondary, who was admitted with behavioural abnormality, emotional incontinence, unsteadiness of gate, weakness of all four limbs and urinary incontinence, started after 23 days of rash. Clinical examination revealed disorientation to time, place and person, lacking social inhibition and spastic quadriparesis (MRC grade 4 in all four limbs). Cerebellar signs were positive including spastic ataxic dysarthria. Contrast MRI of brain, optic nerve and whole spine revealed bilateral T2 hyperintensities in brain, brainstem, cerebellum and upper cervical cord [Figure 1, Panel e,f]. CSF showed 6 cells/mm^3 (all lymphocytes), glucose 71% of capillary blood glucose and protein 123 mg/dL; oligoclonal bands were absent. VEP was normal. Anti-aquaporin-4 and anti-MOG antibody were negative. Patient was treated with intravenous methylprednisolone at a dose of 1 g/day for 5 days followed by oral tapering over 2 weeks. The higher functions improved

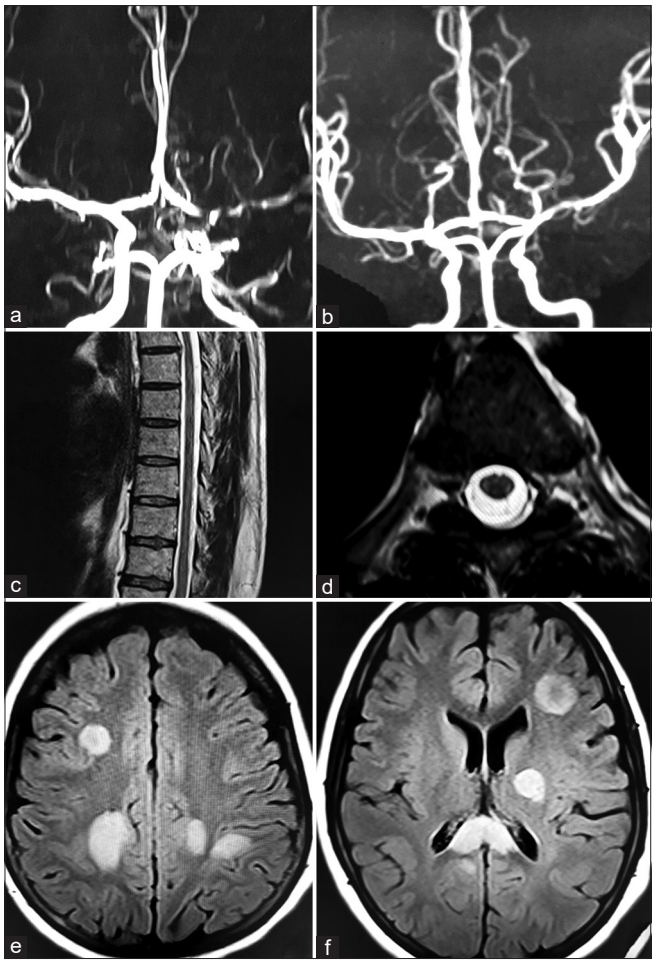

Figure 1: Gradual improvement of middle cerebral artery calibre and distal flow in the magnetic resonance time of flight angiography imaging at presentation (Panel a) and 6 months later (Panel b) in a patient of post-varicella acute left middle cerebral artery territory infarct (Case 15); magnetic resonance T2-weighted image in sagittal (Panel c) and axial (Panel d) image showing no signal change in dorsal cord (Case 10); magnetic resonance T2-weighted brain image in axial section at centrum semiovale (Panel e) and basal ganglia level (Panel f) showing bilateral asymmetric fluffy white matter signal changes with involvement of corpus callosum (Case 16)

in 2 weeks. Her upper motor neuron signs were normalized at 3 months' follow-up.

Post-varicella generalized tonic–clonic seizure and sudden death in a young female with neuromyelitis optica spectrum disorder

A 6-year-old child while on rituximab therapy for neuromyelitis optica spectrum disorder (NMOSD) developed chickenpox (Case 17). After 2 weeks of rash, she developed encephalopathy and generalized convulsion. She received rituximab 3 weeks before development of chickenpox. We treated her with protocol-based aggressive seizure control measures and antiviral therapy in intensive care unit settings. She succumbed within 24 h of her admission. Her CD19 lymphocyte level was 4% on the day of admission. Other investigations could not be performed.

Discussion

Neurological complications of chickenpox are mostly documented after appearance of rash; although in some unusual situation, they can precede the exanthems. Identification of varicella antigens by polymerase chain reaction (PCR) may be helpful in such cases.[8,9] As we did not perform PCR for varicella antigens, some of the cases might have been missed during this period. Encephalitis and cerebellar ataxia are common neurological complications, whereas the unusual manifestations are GB syndrome, facial paralysis, transverse myelitis, aseptic meningitis, cerebral angitis, optic neuritis, meningoencephalitis, ventriculitis, delayed contralateral hemiparesis and peripheral motor neuropathy.[4,9]

Varicella infection is a rare antecedent for Guillain-Barré syndrome (GBS). To find out antecedent infections in GBS, Jacobs *et al*.[10] concluded that one case out of 154 was found in relation to chickenpox. Unlike other studies, it was the most frequently documented complication in our series. All five cases were male in our study with average latency of 15.6 days. None of the patients were vaccinated against chickenpox; neither they received any antiviral therapy. A study conducted by Islam *et al*.[11] in a tertiary care centre of Bangladesh revealed that out of 536 patients diagnosed as GBS, 7 of them had antecedent chickenpox infection within 1 month of onset of weakness, all of them were bed-bound, three patients required mechanical ventilatory support, but all of them had an excellent outcome at 1-year follow-up. Although worldwide AIDP is the most common variant, in our series AMSAN was more common. Previously published reports have shown that post-varicella GB syndrome is mostly demyelinating type, but there are documented cases where it is of axonal type in the form of either AMAN or AMSAN.[12] Few cases of post-infectious concomitant GB syndrome and optic neuritis are documented in literature;[13] but to the best of our knowledge, the Case 10 of our series is probably the first reported case of optic neuritis in association with varicella infection in an immunocompromised patient. All patients recovered well with IV-IG and when required respiratory support was given. No specific diagnostic features distinguish the post-varicella GB syndrome from cases due to other causes. Evolution of weakness after recovering from the viral illness, absence of CSF pleocytosis, presence of albumino-cytological dissociation and good response to IV-IG without antiviral therapy suggests that GB syndrome is an immunological phenomenon triggered by primary varicella infection probably by molecular mimicry.[14]

Second most common neurological complication in our series is LMN facial palsy. All of them were unilateral, though bilateral cases are more commonly reported.[15,16] The latency is minimum for this neurological manifestation (9.25 days on average). Apart from one patient, all had House–Brackmann Facial Nerve Score 7–8 at the end of 3 months.[17] The association between LMN facial palsy and varicella is uncommon and a poorly understood entity. Possible mechanisms are direct nerve

invasion by virus or immune-mediated inflammatory nerve damage. In majority of the published literature, young patients responded well to acyclovir and/or steroids.[15]

In our series and other peer-reviewed literature, signs and symptoms of varicella myelopathy are mostly similar with any post-infectious myelopathy.[18] Unlike other studies, none of our cases had MRI abnormality. The MRI of dorsal spine was conducted within 1 week from the development of clinical features suggestive of myelopathy. Myelopathy with normal imaging is a well-known clinical conundrum. In MRI-negative myelopathy cases, among the other aetiologies, varicella also needs to be ruled out.[19] It has been shown that in immune-mediated myelopathies, initial MRI often remains normal, but diffusion tensor imaging (DTI) done at that time may show dysfunction of the white matter tracts.[20] Parainfectious myelopathies are presumed to be immune-mediated phenomena, which might explain why our patients had a normal MRI. Due to lack of resources, we could not perform DTI and because of logistic issues, we did not obtain a follow-up MRI, which could have shown signal changes. Three proposed pathologic mechanisms of varicella myelopathy are post-infectious immunological process, direct viral invasion of spinal cord and angitis.[21] CSF can be abnormal with cell count not more than 160 mm^{-3} and the protein can be normal to moderately increased.[22] In our series, we documented CSF abnormality (albumino-cytological dissociation) in one case only. Only one out of three patients required plasmapheresis but all recovered at the end of 3 months and became ambulatory. Patient requiring plasmapheresis had albumino-cytological dissociation, though its prognostic significance is not established in literature.

Cerebellitis is the commonest neurological complication of chickenpox occurring in 1 in 4000 cases of children less than 15 years.[6] We documented two cases of acute cerebellitis in young female individuals. One lady had scanning speech, which was seen in one in three cases as per other studies.[23] In children, acute cerebellitis usually results from infectious causes and with complete recovery, whereas autoimmune or paraneoplastic causes are common in adults with poor prognosis.[24] Despite having a prior history of varicella infection, both of our adult patients did not improve with immunomodulatory therapy. We did detailed evaluation to exclude other causes. Few studies suggest use of acyclovir suspecting significant disease severity but others did not support considering its autoimmune pathology.[25]

As documented in literature, most children with typical post-varicella arteriopathy (PVA) develop unilateral cerebral artery stenosis, which has a monophasic course but may progress up to 6 months and gradually normalizes either spontaneously or after acyclovir or steroid therapy between 1 and 14 months after the first clinical event.[26-29] A large number of cases of focal cerebral arteriopathy improves over time, also termed as transient cerebral arteriopathy and a significant portion of those are varicella related, termed as PVA.[30,31] Our patient of ischemic stroke with M1 segment stenosis was treated with antiviral and steroid and she made a significant functional and angiographic improvement within 3 months.

ADEM is a monophasic, usually steroid-responsive immune-mediated demyelinating disorder of CNS, which mostly occurs within 4 weeks after viral infections.[32] Only few cases are documented in literature in adult (about 0.01–0.03%) and the clinical features, treatment response and outcome are unclear.[33] Varicella is an uncommon aetiology of ADEM with an incidence of 0.5–1 in 100,000 varicella infected population.[34] The proposed pathological mechanisms for varicella-associated ADEM can be either molecular mimicry or direct viral invasion.[35] Our patient presented with clinical features and MRI findings of ADEM in after 23 days of chickenpox and responded well to IV methylprednisolone.

Our youngest patient with NMOSD developed generalized seizure followed by altered sensorium after 14 days of development of the exanthem. She received rituximab therapy 3 weeks prior to the fever and her CD19 lymphocyte level was 4% on the day of admission. We presumed the encephalopathy was fulminant relapse of NMOSD triggered by varicella. A few studies have shown that a significant proportion (up to 18%) of seropositive patients with NMOSD may initially present with seizure, encephalopathy or brainstem involvement, especially in children.[36]

Spontaneous recovery after chickenpox is common but disabling or fatal neurological complications may occur in both adults and children. Although ataxia and encephalitis constitute more than 50% of cases of post-varicella neurological complications, we had only two cases (11.7%). In our series, GB syndrome was the most common complication (29.4%), followed by LMN facial palsy (23.5%) with satisfactory outcome. We seek to acquaint clinicians with those complications to avoid delay in diagnosis and appropriate management.

Article footnote

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Conflicts of interest

There are no conflicts of interest.

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