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Dengue infection-associated brachial plexopathy: Report of the first case and review of the literature

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Abstract

Brachial plexopathy is an uncommon neurologic disease which is associated with many conditions including infectious and non-infectious conditions. Many viral infections have been reported to be associated with brachial plexopathy. To the best of our knowledge, dengue infection-associated brachial plexopathy has never been reported in the literature. We report here a case of dengue infection complicated by bilateral brachial plexopathy, and also review all reported cases of viral infection-associated brachial plexopathy in the English literature.

INTROUDUCTION

Brachial plexopathy (neuralgic amyotrophy, Parsonage-Turner syndrome), an uncommon neurologic disease, is characterized by acute neuropathic pain and weakness of the upper extremities.[1,2] It is associated with many conditions including hereditary disease, trauma, surgery, irradiation, pregnancy, vaccination, autoimmune diseases, and infections.[2] Dengue virus is a member of the family Flaviviridae, and there are 4 serotypes with closely related antigenicity.[3] Dengue virus can cause a wide spectrum of manifestations ranging from asymptomatic, benign febrile illness (dengue fever) to severe or fatal disease (dengue hemorrhagic fever and dengue shock syndrome).[3] Neurologic complications reported in association with dengue infection include encephalopathy, transverse myelitis, Guillain-Barré syndrome, mononeuropathy, polyneuropathy and aseptic meningitis.[4,5] To the best of our knowledge, dengue infection-associated brachial plexopathy has never been reported in the literature. We report here a case of dengue infection complicated by bilateral brachial plexopathy, and also review all reported cases with viral infection-associated brachial plexopathy in the English literature.

CASE REPORT

A 62-year-old woman living in Bangkok presented with fever orthopnea and severe pain at her neck and interscapular area which was aggravated by movement for 3 days. She had preexisting hypertension and dyslipidemia. The patient denied any recent trauma. Physical examination revealed an acutely distressed Thai female patient with blood pressure of 160/100 mmHg, pulse rate of 102/minute, respiratory rates of 20/minute and 28/minute upon upright and supine positions, and temperature of 36.3°C. Neurological examination revealed bilateral winging of the scapulae (more on the right), bilateral diaphragmatic paralyses and weakness of the following muscles, right biceps brachii Medical Research Council (MRC) grade 4/5, right serratus anterior muscle grade 3/5, and bilateral infraspinatus muscles grade 3/5. There was no muscle atrophy. The biceps brachii, triceps, and brachioradialis reflexes were reduced bilaterally. Other examinations were unremarkable.

A complete blood count showed hemoglobin of 12.6 g/dL, white blood cell count of 10,600/mm^3 (60% neutrophils, 30% lymphocytes, and 10% monocytes), and a platelet count of 342,000/mm^3. Other blood chemistry tests were normal. Cerebrospinal fluid (CSF) analysis revealed a leukocytes count of 18 cells/μL (100% lymphocytes), glucose of 75 mg/dL (blood glucose of 141 mg/dL), and protein of 37.9 mg/dL. HIV serology was negative. A presumptive diagnosis upon admission was Guillain-Barré syndrome, and hence intravenous immunoglobulin (IVIG) was given to the patient. However, after a careful review of her history and physical examination,

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a diagnosis of bilateral brachial plexopathy was made, and IVIG was discontinued after 2 days of administration.

Electrodiagnostic tests were performed four weeks after the onset of her symptoms. Motor nerve conduction studies of both median and ulnar nerves, with stimulation up to the Erb's points as well as the F wave latencies were normal. Phrenic nerve conduction studies showed absence of the compound muscle action potential (CMAP) on the right and reduced CMAP amplitude on the left. Sensory nerve conduction studies of median, ulnar, superficial radial, and medial antebrachial cutaneous nerves were normal, but reduced sensory nerve action potential (SNAP) amplitude of the right lateral antebrachial cutaneous nerve was noted. Concentric needle electromyography showed a mild-to-moderate degree of denervated motor unit potentials and reduced recruitment pattern in the supraspinati and infraspinati bilaterally, the right serratus anterior and the right biceps brachii. These findings were consistent with bilateral brachial plexopathy mainly involving upper trunk, with bilateral phrenic and right long thoracic nerve involvement (Table 1). Magnetic resonance imaging (MRI) of the cervical and thoracic spine and brachial plexus showed no abnormalities.

Polymerase chain reaction (PCR) tests of the CSF for varicella-zoster virus, herpes simplex virus Type 1 and 2, cytomegalovirus, Epstein-Barr virus, pan-enteroviruses, and West Nile virus were negative. Further investigations were carried out to determine the causative agent in this patient. Enzyme-linked immunosorbent assay (ELISA) for dengue virus IgM and IgG[6] were 115 and 3 units in the serum, and 0 and 15 units in the cerebrospinal fluid, respectively. All hemagglutination inhibition (HAI) titers[7] for dengue viruses 1, 2, 3, and 4 were 1: 80 in the serum. In addition, ELISA for Japanese encephalitis virus IgM in the serum and cerebrospinal fluid were 94 and 0, respectively. Reverse-transcription nested PCR test for 3' untranslated region (UTR) for dengue virus was performed as described by Putcharoen *et al*.[8] showed positive results in the serum and peripheral blood mononuclear cells, and negative result in the cerebrospinal fluid. PCR test for Japanese encephalitis virus showed negative results in all samples tested. On further questioning, the patient did not reveal a history of dengue infection.

Three days after hospitalization, the patient developed ventilatory failure requiring bilevel positive airway pressure (BiPAP) ventilatory support. Seven days after hospitalization, there were an absence of shoulder pain and a partial improvement of her weakness. She was discharged home after one month of hospitalization, and continued to have ventilatory support from BiPAP at night. She was doing well without BiPAP ventilatory support with near complete recovery of weakness of all muscles when last seen 6 months after being discharged from the hospital.

DISCUSSION

Brachial plexopathy is a subject of controversy regarding the anatomical localization of the disease between anterior horn cells and brachial plexus. Recently, due to the advanced electrodiagnostic tests and magnetic resonance imagings, most investigators believe that the disease is primarily on the brachial plexus.[9] Neurological complications is common for several members of the family Flaviviridae including Japanese encephalitis, WNV, St Louis encephalitis, and Murray Valley encephalitis.[10] Furthermore, most viruses in this family have been reported to cause a poliomyelitis-like syndrome. Based on the electrodiagnostic and imaging studies as well as autopsy findings, most patients had anterior horn cell disease and some had evidence of radiculitis.[10] Dengue virus is also a member of the family Flaviviridae, and it would not be surprising if dengue infection can also be complicated with encephalitis, poliomyelitis-like syndrome, and brachial plexopathy. The first patient with dengue-associated diaphragmatic paralysis was reported by Chien *et al*.[11] The patient was a 34-year-old man who developed bilateral phrenic nerve palsies 3 weeks after recovery from dengue infection. Unfortunately, there were no much details in electrodiagnostic and imaging studies, and the authors concluded that the patient had postinfectious phrenic neuropathy.

Brachial plexopathy has been reported to be associated with several viral infections including herpes zoster virus (HZV), Epstein-Barr virus (EBV), parvovirus B19, cytomegalovirus (CMV), hepatitis E virus (HEV), human immunodeficiency virus (HIV), and West Nile virus (WNV). To our knowledge, dengue-associated brachial plexopathy has never been reported. There are 25 reported patients with viral infection-associated brachial plexopathy in the English literature (Table 2).[12-34] Among DNA viruses, HZV is the most common causative agent (7 patients), followed by parvovirus B19 (7 patients, one with CMV co-infection), EBV (4 patients), and CMV (one patient). Among RNA viruses, there are 2 each of HEV and HIV, one each of

Table 1. A summary of electrophysiologic study in our patient.

Motor nerve conduction study (NCS) Normal NCS of bilateral median and ulnar nerves as well as F wave latencies Absent compound muscle action potential (CMAP) of right phrenic nerve Reduced CMAP of left phrenic nerve
Sensory NCS Normal NCS of bilateral median, ulnar, superficial radial, and medial antebrachial cutaneous nerves Reduced sensory nerve action potential (SNAP) amplitude of right lateral antebrachial cutaneous nerve
Electromyography Denervation potentials and reduced recruitment pattern of bilateral supra and infraspinatus, right serratus anterior, and right biceps brachii

Sensory NCS

Nerve/sites	Onset latency (msec)	Peak amplitude (μV)	Velocity (m/sec)
Right median - digit II			
Wrist	2.15	43.3	60.5
Left median - digit II			
Wrist	2.05	49.1	63.4
Right ulnar - digit V			
Wrist	2.00	45.7	55.0
Left ulnar - digit V			
Wrist	1.90	39.8	57.9
Right radial - wrist			
Forearm	1.95	37.6	66.7
Left radial - wrist			
Forearm	2.10	44.1	61.9
Right lateral medial antebrachial			
Lateral	2.00	5.4	50.0
Medial	2.00	13.4	50.0
Left lateral medial antebrachial			
Lateral	1.70	32.0	58.8
Medial	1.75	13.6	57.1

Motor NCS

Nerve/stes	Onset latency (msec)	Peak amplitude (μV)	Velocity (m/sec)
Right median - APB			
Wrist	3.00	11.3	
Elbow	7.00	9.9	50.0
Axilla	10.35	9.4	56.7
Extensor pollicis	12.55	9.5	68.2
Left median - APB			
Wrist	2.95	14.7	
Elbow	6.75	12.8	55.3
Axilla	10.05	12.7	54.5
Extensor pollicis	12.25	12.8	63.6
Right ulnar - Abductor digiti minimi			
Wrist	2.05	11.0	
Below elbow	5.70	9.9	56.2
Above elbow	7.55	8.7	59.5
Axilla	10.05	8.3	52.0
Extensor pollicis	12.15	7.7	66.7

Left ulnar - Abductor digiti minimi			
Wrist	2.40	11.4	
Below elbow	5.80	10.0	58.8
Above elbow	7.55	9.8	57.1
Axilla	10.10	8.7	56.9
Extensor pollicis	12.30	8.3	63.6
Right phrenic - Xiphoid process			
SCM		NR	
Left phrenic - Xiphoid process			
SCM	7.05	0.2	

F wave

Nerve	Minimum F latency (msec)
Right median - Abductor pollicis brevis	25.60
Right ulnar - Abductor digiti minimi	25.75
Left median - Abductor pollicis brevis	25.05
Left ulnar - Abductor digiti minimi	26.10

Needle EMG

EMG summary table

	Spontaneous			MUAP			Recruitment
	IA	Fibrillation	PSW	Amp	Duration	PPP	Pattern
Right biceps	N	1+	1+	N	N	N	Mildly reduced
Right infraspinatus	I	None	1+	N	N	+	Mildly reduced
Right supraspinatus	I	2+	1+	N	N	N	Mildly reduced
Right serratus anterior	I	1+	None	N	N	N	Mildly reduced
Right extensor digitorum communis	N	None	None	N	N	N	Normal
Right first dorsal interosseous	N	None	None	N	N	N	Normal
Left infraspinatus	I	2+	2+	N	N	N	Moderately reduced
Left trapezius	I	None	None	N	N	N	Normal
Left extensor digitorum communis	N	None	None	N	N	N	Normal
Right deltoid	N	1+	1+	N	N	N	Mildly reduced

MUAP: motor unit action potential, IA: insertional activity, PSW: positive sharp wave, Amp: amplitude, N: normal, I: increase, PPP: polyphasic potential

WNV and dengue virus (our report) infections. The age ranges from 9 to 86 years. There are 17 (15 DNA and 2 RNA viral infections) and 8 (4 each of DNA and RNA viral infections) patients with and without prodromal symptoms, before a development of brachial plexopathy. Antecedent infection with prodromal symptoms was observed in 100% (4 of 4 patients) and 85.7% (6 of 7 patients) of EBV and HZV brachial plexopathy. In one patient, brachial plexopathy developed 60 days after a previous HZV infection.[13] Our patient had no prodromal period; she had brachial plexopathy in association with low-grade fever. Among 25 patients, 15 (60%) and 10 (40%) patients had unilateral and bilateral neurologic involvement, in comparison with a previous study of neuralgic amyotrophy by van Alfen *et al*. which showed unilateral involvement of 71.5%.[2] Our patient had bilateral brachial plexopathy, more severe on the right side. Regarding neurologic manifestations, 23 (92%) patients had shoulder or upper limb pain, 17 (68%) patients had sensory deficits, and 23 (92%) patients had weakness, in comparison with a study by van Alfen *et al*. which

Table 2. A summary of 25 patients with viral infection-associated brachial plexopathy in the English literature.[12-34]

Virus	Sex, age (Y), country	Prodrome			Neurologic manifestations			CSF	Electro-diagnosis	MRI	Treatment	Outcome
		Clinic	Duration (D)	Involvement	Sensory: Pain	Sensory: Numbness	Weakness					
I. DNA virus												
HZV	F, 78, Australia[12*]	No, coincident rash at left arm	0	L	Shoulder, arm	Dysesthesia at arm	Shoulder, arm, forearm	NA	NA	NA	No	died from acute MI
	M, 54, Turkey[13]	Rash at left arm	60	L	Shoulder, arm, hand	Hyperesthesia, paresthesia at arm	Arm	NA	Upper, middle lower trunk	Unremarkable	Acyclovir	Partial recovery at 4 M
	M, 54, US[14]	Rash at T4	7	B	Neck, shoulders	No	Deltoids, biceps, diaphragms	NA	Middle trunk	Unremarkable	Valacyclovir	Complete recovery of shoulder weakness at 1 M, no recovery of diaphragmatic weakness at 19 M
	M, 74, Korea[15]	Rash at right C5-6	7	R	L5-6	Paresthesia, hyperesthesia	Shoulder, arm, forearm	Pleocytosis, L	Upper, middle trunks	T2 hyperintensity enhancement of plexus	Acyclovir	Nearly complete recovery at 2 M
	F, 76, Korea[15]	Rash at left C8, T1	7	L	Arm	Paresthesia at arm	Arm	NA	Median cord	Enhancement of plexus	Acyclovir	Nearly complete recovery at 2 M
	M, 78, UK[16]	Rash at right C4-6	14	R	Arm	No	Shoulder, arm	Normal	Plexopathy	Unremarkable	Acyclovir	Nearly complete recovery at 12 M
	F, 86, UK[17]	Rash at right shoulder, arm	14	R	Shoulder, arm	Arm	Shoulder,	NA	Radiculoplepathy	Thickening, edematous plexus	Acyclovir	Nearly complete recovery at 6 M
EBV	F, 76, US[18]	Rash at right arm and forearm	1	R	Hand	Hand	Hand, fingers	NA	Radiculoplepathy	NA	Acyclovir	Improvement at 3 M
	M, 19, Canada[19]	Sore throat, rash, lymphadenopathy	25	B	Shoulders	Shoulders	Shoulders	NA	Plexopathy	NA	No	Nearly complete 4 M
	M, 18, Poland[20]	Fever, tonsillitis, lymphadenopathy	7	L	Shoulder, arm	Arm	Shoulder, arm, forearm	Normal	Plexopathy	Unremarkable	No	Nearly complete recovery
	M, 56, US[21]	Fever, OM, lymphadenopathy	2	R	Neck, back	Paresthesia at arm, hand	Shoulder, arm, forearm, hand	Pleocytosis, L	Radiculoplepathy	T2 hyperintensity	No	Complete recovery at 6 M

Virus	Sex, age (Y), country	Prodrome			Neurologic manifestations			CSF	Electro-diagnosis	MRI	Treatment	Outcome
		Clinic	Duration (D)	Involvement	Sensory		Weakness					
					Pain	Numbness						
B19	F, 26, Canada[22]	Flu-like symptoms	7	L	No	Hand, fingers	Fingers	NA	Plexopathy	Unremarkable	IVIG	No recovery at 18 M
	M, 26, UK[23]	Flu-like symptoms, rash	5	B	Shoulders, arms	Arms	Shoulders, arms, forearms	NA	Plexopathy	NA	No	NA
	F, 23, UK[24]	Rash	14	B	Arms	Right forearm	Shoulders, arms	Normal	Plexopathy	NA	No	Improvement at 6 M
	NA, 23, France[25]	Flu-like symptoms rash, arthralgia	5	L	Shoulder	No	Shoulder	NA	Plexopathy	NA	No	Improvement at 2 M
	F, 38, US[26]	No, coincident rash	0	L	Shoulder, arm	Fingers	No	NA	NA	NA	Steroid	Complete recovery at 3M
	F, 33, France[27]	No, coincident fever, rash, arthritis	0	B	Arms	Forearms	No	Normal	NA	NA	No	Improvement at 3 M
	F, 9, Germany[28]	No	0	R	Shoulder, arm	Paresthesia at hand, fingers	Shoulder	NA	Normal NCV	Unremarkable	No	Complete recovery at 12 M
B19 and CMV	M, 23, Netherlands[29]	Flu-like symptoms, transient rash	10	B	Shoulders, arms, fingers	Hands, fingers	Shoulders, arms,	NA	Plexopathy	Unremarkable	Ganciclovir	Mid recovery at 6 M
II. RNA virus												
HEV	M, 53, UK[30]	No	0	B	Shoulder	Shoulder, arms	Shoulders	NA	Plexopathy	Unremarkable	No	Complete recovery at 24 M
	M, 49, Thailand31	Fever, abdominal pain	4	B	Shoulders	Fingers	Fingers	Normal	Plexopathy	Unremarkable	No	Improvement at 10 M
HIV	M, 35, US[32]	No	0	B	No	No	Shoulders	Normal	Radiculoplexo-pathy	Unremarkable	IVIG	No recovery at 48 M
	M, 32, US[33]	Fever, rash, lymphadenopathy	14	L	Shoulders	No	Shoulders	NA	Radiculoplexo-pathy	NA	N	No recovery at 3 M
WNV	M, 48, US[34]	No, coincident fever, vomiting, confusion	0	L	Neck, back	Arm	Shoulder, arm	Pleocytosis, lymphocy-tosis	Plexopathy	Unremarkable	No	Partial recovery
Dengue virus	F, 62, Thailand (present report)	No, coincident fever	0	B	Neck, shoulders	No	Shoulders, arms, diaphragms	Pleocytosis, L	Plexopathy	Unremarkable	IVIG	Nearly complete recovery at 6 M

*Autopsy findings revealed extensive lymphocytic infiltration, myelin breakdown of brachial plexus; perivascular lymphocytic inflammation of posterior nerve root of C3

M: Male, F: Female, US: the United States, UK: the United Kingdom, Y: year, M: month, D: day, CSF: cerebrospinal fluid, MRI: magnetic resonance imaging, NA: not available, IVIG: intravenous immunoglobulin, EBV: Epstein-Barr virus, HZV: Herpes zoster virus, CMV: Cytomegalovirus, HEV: Hepatitis E virus, B19: B19 Parvovirus, WNV: West Nile virus, B: bilateral, L: left, R: right, NCV: nerve conduction velocity, OM: otitis media, MI: myocardial infarction, L: lymphocytosis

showed that shoulder or limb pain was noted in 90% of all patients.[2] In addition, 2 (including our patient) patients had diaphragmatic paresis. The CSF analysis was performed in 10 patients, and 4 (including our patient) had mild lymphocytic pleocytosis. All except one patient underwent electrodiagnostic study, and had evidence of brachial plexopathy or radiculoplexopathy. The other patient with HZV brachial plexopathy died due to acute myocardial infarction, and postmortem examination revealed extensive lymphocytic infiltration at C3 posterior nerve root and brachial plexus.[12] MRI was performed in 15 patients, 4 patients had T2-weighted hyperintensity and gadolinium T1-weighted enhancement of brachial plexus; the other 11 (including our patient) patients had unremarkable results. The outcomes of brachial plexopathy were excellent. Only 6 patients had mild or no neurologic recovery. Our patient made a nearly complete recovery at 6 months of follow-up.

In conclusion, we report here the first case of dengue infection complicated by bilateral brachial plexopathies. All 25 patients with viral infection-associated brachial plexopathy in the English literature are also reviewed.

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DISCLOSURE

Conflit of interest: None

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