Case Report

Post-varicella Guillain Barré syndrome

Ahmed I. Hamad, *FRCP, cFAAN,* ***Wisam K. Ghadban,*** *MRCP,* ***Aymen A. Hamad,*** *CABM,* ***Hassan J. Al-Hail,*** *FACHARZT.*

ABSTRACT

Varicella-zoster virus (VZV) infection has not often been related to the development of Guillain Barré Syndrome (GBS). However, the close temporal relationship noted between varicella infection and the onset of GBS suggests the participation of VZV in the etiopathogenesis of GBS. Very few cases have been reported showing this relationship. This report describes 2 cases highly suggestive of this association.

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Guillain Barré syndrome (GBS) is an acute demyelinating polyneuropathy. The exact pathogenesis is not known, but it is suggestive of an autoimmune mechanism.[1,2] Preceding infection with various infectious agents may trigger the process. Viruses such as cytomegalovirus, Ebstein Barr virus and human immune deficiency virus have been convincingly associated with GBS. Other viruses such as varicella-zoster have been rarely reported.[3] Varicella neurological complications are rare, encephalitis being the most common (1:1000),[4] and GBS the least common (1:15000).[5-8] We report on 2 cases of GBS following varicella encountered over a 12-year period.

Case Report. ***Patient One.*** A 42-year-old Qatari male soldier was admitted with difficulty in swallowing, weakness and numbness of his limbs. He gave a history of chicken pox 20 days prior to admission, which had almost cleared. Examination showed bilateral facial weakness, proximal power: 4/5, areflexia, glove and stocking hypoesthesia. There was reduced nerve conduction velocity, normal compound muscle action potential, delayed F latency and conduction block in 3 nerves. Cerebrospinal fluid showed acellular fluid with slight increase in protein (0.62gm/l). The patient recovered completely after 3 months with mild residual facial palsy.

Patient 2. A 30-year-old Bengali male laborer was admitted with abdominal pain, vomiting and generalized tonic clonic convulsions. He was well until 3 weeks prior to admission when he had chicken pox. His examination was normal apart from confusion, shortness of breath and chicken pox rashes on his body and face (**Figure 1**). His blood tests showed that he was in acute renal failure (urea 30, creatinine 1100, 24-hour urine protein 4gm/L, without cells or casts), chest x-ray showed pulmonary edema. He was put on a ventilator, computerized tomography brain was normal and electroencephalogram showed mild generalized slowing. He underwent 5 series of dialysis within 5 days after which his kidney function improved. No more seizures occurred after admission, and the ventilator was disconnected after 5 days. At that time he was conscious, oriented and complained of generalized weakness and shortness of breath. Examination showed power of 3/5 in arms and 2/5 in legs, areflexia, normal sensation, nerve conduction study showed reduced conduction velocity with conduction block and reduced amplitude suggestive of axonal damage. Cerebrospinal fluid showed acellular fluid and high protein (2.1 gm/l). Due to

From the Department of Neurology, Hamad General Hospital, Doha, *Qatar*.

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Address correspondence and reprint request to: Dr. Ahmed Hamad, Consultant Neurologist, Hamad General Hospital, PO Box 3050, Doha, *Qatar*. Tel. +974 4392773. Fax. +974 4392273. E-mail: aehamad@yahoo.com

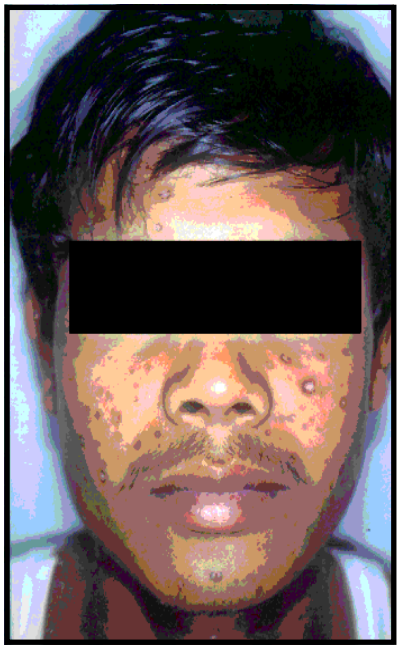

Figure 1 - Patient 2 showing crusted varicella skin eruptions on the face.

impending respiratory failure he was ventilated again for 2 more weeks, after which he slowly recovered and one month after discharge he was completely recovered with no residual weakness.

Discussion. Our first case showed a temporal relationship between GBS and varicella. The 2nd case had acute renal failure secondary to acute viral interstitial nephritis that lasted only 5 days. The clinical picture and electromyogram is against the axonal neuropathy of acute illness. The accelerated neuropathy of renal failure is rare and occurs in cases of established renal failure who have rapid deterioration, usually with hypertension or diabetes.[9] This makes GBS following varicella the most likely diagnosis.

Reported cases in literature recount a severe disease course requiring ventilation and treatment with either IVIG[2] or plasmophoresis,[7] with residual weakness. However, our patients had benign course with full recovery indicating a benign course of GBS following varicella. These 2 cases, and others reported, suggest the participation of varicella-zoster virus in the pathogenesis of GBS.

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