

## DISCUSSION

Meckel's diverticulum was originally described by Fabrius Hildanus in 1598. However, it is named after Johann Friedrich Meckel, who established its embryonic origin in 1809. The most common presentations of Meckel's diverticulum are gastrointestinal bleeding from associated ectopic gastric mucosa, diverticulitis, perforation and neoplasia, uncommon presentations include axial volvulus of the diverticulum and internal herniation<sup>5</sup>. Small bowel obstruction can be the result of intussusceptions, strangulation due to a mesodiverticular band or volvulus. Few cases of intestinal obstruction due to enterolith have been reported<sup>6</sup>. It was postulated that calcium from the intestinal contents gets precipitated which initiates the formation of stone. But in our case there was no stone or whatsoever. The faecal matter first got trapped in the pouch of diverticulum and then progressed to involve the adjacent loop of ileum. It formed a firm to hard mass which prevented to crush the mass into small pieces which were then milked into the large intestine. The cause of faecal impaction couldn't be elucidated. May be the narrow mouth of the diverticulum initiated the process of faecal impaction and then followed to involve the adjacent ileal loop resulting in formation of a hard mass causing obstruction. After emptying, diverticulectomy was done as it was

narrow mouthed. The adjacent ileum was normal and there was no induration of the adjacent ileal mucosa. So the adjacent ileal loop was not resected. Therefore, diverticulectomy only with sparing of the adjacent ileal loop is justified in our case.

## CONCLUSION

Meckel's diverticulum is itself a rare entity and most commonly it presents as gastrointestinal bleeding. Obstruction if at all is due to adhesions and kinks or volvulus but that occurring due to faecal impaction is very rare and should be treated as any other intestinal obstruction.

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## Case Report

# Gullian- Barre Syndrome associated with Herpes Zoster Virus Infection - A Case Report.

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**Abstract:** Gullian- Barre Syndrome (GBS) is a rare disease manifesting as severe, generalized, flaccid paralysis & areflexia. GBS resulting from Herpes zoster is a rare & unusual association. The pathogenesis of post zoster demyelinative polyneuropathy & other widespread complications of herpes zoster is poorly understood. We report a case of GBS following a recent herpes zoster attack.

## INTRODUCTION

Since the first report in 1961, only few cases of Gullian- Barre Syndrome following herpes zoster have been reported in the literature<sup>1,2</sup>. GBS usually occurs as a post – infective immune- mediated phenomenon. Many precipitants have been recognised & it is known that Varicella zoster virus (VZV) infection may trigger GBS. However this is rare. VZV is a herpes virus which causes both varicella (chicken pox) and herpes zoster (shingles). It is postulated<sup>3</sup> that during the viraemic phase of a primary varicella infection, the virus reaches the skin & travels along the sensory axons to the dorsal root ganglion, where it can remain in a latent phase for years before it is reactivated. A case of GBS following herpes zoster viral infection is being reported here for its rarity.

## CASE REPORT

A 42 year old male patient presented with 3 days history of cough, fever & multiple bullous eruptions on the right side of face & trunk. Six

days later, he reported sudden onset sharp lumbar pain which radiated to the right leg accompanied by bilateral lower limb muscle cramps. Four days later, he developed parasthesia, numbness & tingling sensation in both feet followed by weakness of both lower & upper limbs. He was unable to walk & speak properly. There was no history of bladder or bowel impairment, no history of injury, vaccination or exposure to toxins or loss of taste sensation. On examination, he was conscious, oriented, afebrile, BP 120/80 mmHg, pulse 94/min, regular. There was scarring & crusting over the distribution of eruptions on the right side of face & trunk. Bell's palsy was present on the right side. He had more weakness of lower limbs (2/5) as compared to upper limbs (4/5). Deep tendon reflexes were absent & plantars were flexor bilaterally. Sense of vibration, joint position & pinprick was impaired distally in the limbs. All the muscles were not unduly tender. Laboratory investigations revealed Hb 11.2 g/dl; TLC 11300/mm<sup>3</sup>; DLC P73, L27, E0, B0; ESR 47mm/hour; B.urea 37mg%; S.creatinine 1.2%; urine C/E normal. CT Brain, CSF examination & chest X ray was normal. CT myelography was normal. Serological tests for syphilis & systemic lupus

erythromatosus were all negative. Nerve conduction studies showed increased latency & decreased amplitudes suggestive of predominantly acute inflammatory demyelinating polyneuropathy. Keeping in the view the history & clinical symptomatology of the patient, the diagnosis of herpes zoster leading to severe polyneuropathy in the form of Gullian-Barre Syndrome was made. The patient was put on oral acyclovir 800mg 5 times daily for 7 days & showed marked improvement in the power in case of upper limbs & lower limbs with significant improvement in the speech & language. The patient was discharged after 7 days in a stable condition & is on regular follow up.

## DISCUSSION

Our case showed the classical clinical features of GBS namely weakness, parasthesias & areflexia etc. GBS following herpes zoster typically has a latent period of two weeks to two months. Shorter latent periods as in our case, are associated with more severe illness<sup>2</sup>. The pathogenesis of GBS following herpes zoster attack is poorly understood. It is generally considered that GBS results from autoimmune mediated damage to the peripheral nervous system triggered by a preceding infectious event<sup>4</sup>. Some authors here proposed that the pathogenesis by which GBS develop following herpes zoster attack is directly related to autoimmune mediated responses initiated by varicella zoster virus reactivation<sup>1</sup>. Specific pathogens could structurally mimic the molecules from human peripheral nerves that induce cross-reacting anti-peripheral nerve myelin autoimmunity<sup>5</sup>. VZV can interfere with adaptive immunity & can become latent for longer periods, residing in the human nervous system<sup>6</sup>. Regarding the reactivation of VZV, it is proposed that it may be due to imbalance of CD<sub>4</sub> and or CD<sub>8</sub> lymphocytes in the blood during the course of herpes zoster occurrence<sup>7</sup>. Antiviral therapy has been proven to decrease new lesions & to prevent herpetic neuralgia in patients with acute herpes zoster<sup>8</sup>. The possibility that humoral factors are involved provides the rationale for the use of plasma exchange<sup>9</sup>. In GBS, there

is an identifiable precipitant in 10% of cases including mycoplasma, Epstein Barr virus or hepatitis A<sup>10</sup>. GBS following herpes zoster virus is rare, although few cases have been reported in Indian literature<sup>11</sup>. The neurological complications of herpes zoster include sensory radiculopathy, transverse myelitis, GBS & encephalopathy<sup>12</sup>. The very uncommon occurrence of GBS with herpes zoster suggests that most individuals with herpes zoster are somehow able to suppress an autoimmune reaction.

## CONCLUSION

This case suggests that herpes zoster per se may sometimes be a sufficient stimulus to drive antibody generation & precipitate severe clinical symptomatology. The association of GBS with herpes zoster is rare. This prompted us to report this rare clinical entity.

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## Case Report

### Management of Lefort Fractures using External Skeletal Fixator : Two Case Reports.

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**Abstract:** We are reporting two cases of complex maxillofacial injuries involving the midface along with mandible managed under local anaesthesia with External skeletal fixation using POP head frame to mandible and maxilla, respectively. The primary purpose of the paper is to describe this conventional technique as an effective, immediate and economical method for the quality treatment of such fractures managed under OPD settings. The benefits to the patient and the hospital are highlighted.

## INTRODUCTION

Many techniques have evolved for treating midfacial fractures beginning with external skeletal fixation, internal K wire fixation, rubber band traction, direct internal wiring and suspension techniques to the modern era of rigid internal fixation<sup>1</sup>.

External skeletal fixation is still advocated as a method of choice for complex maxillofacial injuries. The technique requires no complicated apparatus and minimum infrastructure for quick and efficient immobilization of the middle-third fracture and patients are mobile immediately after the operation.

## CASE-1

A 35 year old male patient reported to Department of Dental and Oral Surgery, LHMC & Associated Hospitals, with a chief complaint of inability to chew food and generalized pain and swelling over face since two days. There was history of RTA two days back with history of LOC of approx 3 hours and bleeding from nose and mouth. After primary stabilization and neurosurgical clearance, at a local hospital, the patient was referred to our centre for the management of facial fractures. On examination, the general condition was fair with GCS =15. There was generalized swelling over face, with bilateral circumorbital ecchymosis and bilateral subconjunctival hemorrhage. Eye movements and vision

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