Abstract

Bulbar Palsy is B/L impairment of function of cranial N. 9, 10, 11, 12 at lower motor neuron level either at nuclear or fascicular level in medulla or B/L lesion of lower cranial N. outside brainstem.

We present case of viral hepatitis who initially presented with classical signs and symptoms of hepatitis B followed by characteristic features of bulbar palsy in form of difficulty in swallowing and slurring of speech reduced gag reflex, weak palatal movement. Other causes for bulbar palsy were excluded and indirect laryngoscopy confirmed presence of bulbar palsy. Patient had no previous neurological abnormality, there are many studies in the past for association of hepatitis B and bulbar palsy but no one confirmed about a direct association between hepatitis B and bulbar palsy. To best of knowledge this is the first case report which shows direct association between hepatitis B and bulbar palsy.

Introduction

B/L weakness or paralysis of muscles supplied by cranial nerves 9, 10, 11, 12 due to lesion at lower motor neuron level either in medulla or B/L lesion of lower cranial N. outside brainstem is termed bulbar palsy.\(^1\) Cause may be genetic, due to Kennedy disease, acute intermittent porphyria, medullary infarction, motor neuron disease, syringobulbia, GBS, polio, Lyme disease, multiple viral infection, brainstem glioma, botulism. Patient present with difficulty in swallowing, chewing, talking, drooling of saliva, and nasal regurgitation. There will be reduced gag reflex weak palatal movement weakness of face/tongue muscle, tongue atrophy/fasciculation. Jaw jerk may be normal or absent. There may be gagging and choking and death due to pneumonia.\(^2,3\)

Hepatitis B and Bulbar Palsy

Previous studies done in this field showed association of hepatitis B and bulbar palsy with use of hepatitis B adult vaccine. Many patients in west who received adult hepatitis B vaccination later progressed to bulbar palsy.\(^4\) Some case reports also show that during follow up of hepatitis B patient they developed bulbar palsy signs/symptoms, majority of them are biased because patient was taking some antiviral drugs particularly abacavir/ lamivudine and as because of
association of these drugs with signs and symptoms of bulbar palsy. No definite causal association could be established in these studies as patient improved after stopping abacavir / lamivudine therapy. 5

Case Report

A 32 yr old male presented to us with c/o mild grade fever, nausea/vomiting, loss of appetite, yellowish discolouration of sclera and urine for last 8-10 days. Investigations done were serum bilirubin T/D/I – 2.42/1.5/.92, SGOT – 870, SGPT – 2361, PT (test) – 18 second, (control) – 14 second, INR – 1.28. Patient’s HBsAg was positive, hepatitis C negative, HIV I and II negative, anti nuclear antibody negative, Anti HBc IgM positive – 17.7 u/ml. HBeAg positive. Ultrasound favoured diagnosis of acute viral hepatitis. Patient was kept on symptomatic treatment and then followed after 2 weeks. Patient developed improvement in acute viral hepatitis sign and symptom but developed difficulty in deglutition for solids not for liquid, patient also developed dry cough, patient also developed slurring of speech suggesting involvement of 9th and 10th cranial nerve palsy. Follow up investigation showed SGOT - 77, SGPT – 219, serum bilirubin T/D/I - 1.02/.51/.51. Codeine and antihistaminic were added to treatment. In next follow up slurring of speech progressively increased compared to previous follow up visit. He also had difficulty in swallowing now both for liquid and solid. Patients also had increased dry cough despite of codeine. Neurological examination favours 9th, 10th LMN palsy. Thyroid function tests WNL, MRI brain was normal, symptoms of Viral Hepatitis disappeared but neurological features persisted. ENT opinion was taken and indirect laryngoscopy confirmed 9th, 10th LMN palsy (Figure 1). Patient’s SGPT was 62, SGOT was 73. After that Methyl cobalamin + pregabalin added (150 microgram + 75 mg OD dose). During subsequent follow up patient recovered completely from acute viral hepatitis but neurological features showed improvement. Investigations were serum bilirubin T/D/I -.94/0.30/0.64, SGPT- 27, SGOT – 23, HBsAg – negative.

Discussion

In our patient there was no previous history of any neurological abnormality/ vaccination available and patient developed bulbar palsy after he got infected with hepatitis B. Signs and symptoms, clinical examination, indirect laryngoscopy confirmed presence of bulbar palsy. In the past and during whole treatment course and follow up no antiviral drug was given.

Conclusion

To conclude our case is a rare case because none of the studies done in past have shown direct association between hepatitis B and LMN bulbar palsy.

References

1. Adams et al, Principal of Neurology sixth edition, p 1091.
2. Neuron disease factsheet NINDS Bethesda M.D.