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CASE STUDY ON PSEUDOBULBAR PALSY, A NEUROLIGICAL DISORDER- PATIENT MONITORING AND PATIENT COUNSELING BY CLINICAL PHARMACIST

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ABSTRACT

Pseudobulbar palsy is a medical disorder in which there is an inability to control facial movements such as chewing and speaking). It may occur in association with a variety of neurological diseases like amyotrophic lateral sclerosis, Parkinson's disease, Alzheimer's disease, stroke, CADASIL syndrome (cerebral autosomal dominant arteriopathy with sub cortical infarcts and leukoencephalopathy), brain injury and brain tumors. The psychological consequences and the impact on social interactions may commonly misidentify as a mood disorder, particularly depression or bipolar disorder. In pseudobulbar palsy the pathways involving serotonin and glutamate are disrupted. So the effective treatment for many years is with antidepressants like tricyclic antidepressants and SSRI'S (selective serotonin receptor inhibitors). But recently a new drug was approved by FDA Nuedexta a combination of dextromethorphan and quinidine as safe and effective for treating Pseudobulbar palsy.

KEYWORDS: Pseudobulbar palsy, Amyotrophic Lateral sclerosis, Parkinson's disease, Alzheimer's disease, Nuedexta.

INTRODUCTION

Definition

Pseudobulbar palsy is a medical condition characterized by the inability to control facial movements (such as chewing and speaking) and caused by a variety of neurological

disorders. Patients experience difficulty chewing and swallowing, have increased reflexes and spasticity in tongue and the bulbar region, and demonstrate slurred speech (which is often the initial presentation of the disorder), sometimes also demonstrating uncontrolled emotional outbursts.[1]

Etiology

Pseudobulbar palsy is the result of damage of motor fibers traveling from the cerebral cortex to the lower brain stem. This damage might arise in the course of a variety of neurological conditions that involve demyelination and bilateral corticobulbar lesions. This condition is usually caused by the damage (bilateral degeneration) to the neurons of the brain stem, specifically to the corticobulbar tract (upper motor neuron tract to cranial nerve motor nuclei).

Examples include:

Vascular causes: bilateral hemisphere infarction, CADASIL syndrome

Progressive supra nuclear palsy, Brain trauma

Amyotrophic lateral sclerosis

Parkinson's disease and related multiple system atrophy

Various motor neuron diseases, especially those involving demyelination

Multiple sclerosis and other inflammatory disorders

High brain stem tumors

Metabolic causes: osmotic demyelination syndrome

Neurological involvement in Behçet's disease.[2]

Pathophysiology

The proposed mechanism of Pseudobulbar palsy points to the dis inhibition of the motor neurons controlling laughter and crying, proposing that a reciprocal pathway exists between the cerebellum and the brain stem that adjusts laughter and crying responses, making them appropriate to context. [3] The pseudobulbar crying could also be induced by stimulation in the region of the sub thalamic nucleus of the brain.[4]

Signs and symptoms

Slow and indistinct speech

Dysphagia (difficulty in swallowing)

Small, stiff and spastic tongue

Brisk jaw jerk

Dysarthria

Labile affect [5]

Gag reflex may be normal, exaggerated or absent

Examination may reveal upper motor neuron lesion of the limbs

Dribbling persistently.

Facial muscles - may also be paralyzed.

Nasal regurgitation may be present.

Dysphonic.

Emotional liability may also be present.

Diagnosis

Diagnosis of Pseudobulbar palsy is based on observation of the symptoms of the condition. Tests examining jaw jerk and gag reflex can also be performed. It has been suggested that the majority of patients with pathological laughter and crying have Pseudobulbar palsy due to bilateral cortico bulbar lesions and often a bipyrimidal involvement of arms and legs.[6] To further confirm the condition, MRI can be performed to define the areas of brain abnormality. Other tests will depend on the suspected underlying cause but will involve routine blood tests.

Other Investigations

New developments in technology have led to the use of neuro physiological investigations to assess various aspects of speech dysfunction. [7] These include electromagnetic articulography (EMA), electropalatography (EPG) and pressure-sensing EPG.

Management [8]

All patients should be referred to neurologists. Patients will need admission if dysphagia is present or symptoms are rapidly progressive.

Postural changes can help with drooling of saliva and may prevent aspiration.

Supportive measures may include baclofen for spasticity, anti cholinergic for drooling, treatment of aspiration pneumonia if it occurs and attention to nutrition - eg, enteral feeding.[9]

Management should involve speech and language therapists and the dietician.

Genetic analysis may be appropriate for cases presenting in childhood.[10]

Treatment

Since pseudobulbar palsy is a syndrome associated with other diseases, treating the underlying disease may eventually reduce the symptoms of pseudobulbar palsy.

Possible pharmacological interventions for pseudobulbar affect include the tricyclic antidepressants, serotonin reuptake inhibitors, and a novel approach utilizing dextromethorphan and quinidine sulfate. Nuedexta is an FDA approved medication for pseudobulbar affect. Dextromethorphan, an N-methyl-D-aspartate receptor antagonist, inhibits glutamatergic transmission in the regions of the brainstem and cerebellum, which are hypothesized to be involved in Pseudobulbar symptoms, and acts as a sigma ligand, binding to the sigma-1 receptors that mediate the emotional motor expression.[3]

CASE STUDY

An 18 year old female was admitted in King George Hospital Visakhapatnam, Andhra Pradesh, India, with a complaint of weakness of left upper and lower limbs since 12 years, and weakness of right upper and lower limbs, cry's and spells since 5years.

History of present illness includes blurring of vision on right lateral gaze, deviation of angle of mouth to left side , flexion tonicity of left upper limb and extensor tonicity of left lower limbs since 10 months, inhale attacks – 10 attacks / day ,both awake and sleep attacks.

History of past medical illness includes, in an asymptomatic patient the present illness started at 6 years of age. No tonic clonic movements of limbs and frothing from mouth. She got admitted in a low grade hospital, where she gained normal sensation after 2-3 hrs. By next day morning she developed weakness of left upper and lower limbs. She was unable to move her left lower limb sideways and had complete ability to move her left upper limb even sideways. She had a deviation of angle of mouth to right side with dabbling of liquid from left angle of mouth. There is no loss of consciousness. There is a history of slurred speech and difficulty in understanding the words spoken by her. At that time, she was unable to get up from bed with support and needing support of family members for turning in bed also. She was discharged from hospital after 10 days. After 6 months she has minimal improvement in her power and was able to walk with minimum support but not completely. Since last 6-7 years patient developed sudden weakness of right upper and lower limbs. She was completely

dependent on her mother for even daily activities. She was feeling tightness of all 4 limbs. There is a history of jerkiness of feet while keeping feet on floor and while trying to walk. She had 2-3 inhale attacks/day since 3 years both awake and sleep attacks.

On admission her body temperature was normal, pulse rate – 82/min, and respiratory rate was found to be 20/min.

MRI of brain revealed old infarcts with porencephalic changes in the right temporoparietal and left frontal lobar regions of brain parenchyma.

CT-CEREBRAL ANGIOGRAM showed narrowing of bilateral M_1 segment of middle cerebral artery and multiple sub acute to chronic infracts at bilateral frontal, right parieto-temporal region, bilateral capsulo-gangilionic region.

CAROTID DOPPLER was found to be normal.
EEG shows focal discharge in temporal lobe.
CRANIAL NERVE EXAMINATION shows:

Table-1 showing cranial nerve examination

Cranial nerve number	Cranial nerves	Examination
I	Olfactory nerve	Unable to tell name of sick
II	Optic nerve	Visual activity 6/18
III , IV , VI	Oculomotor, trochlear, abducens nerve	Emergency medical services required
V	Trigeminal nerve	Normal
VII	Facial nerve	Facial nerve palsy
VIII	Vestibulocochlear nerve	Normal
IX , X	Glossopharyngeal, vagus nerves	Exaggerated gag reflex
XI	Accessory nerve	Normal
XII	Hypoglossal nerve	Normal

Treatment

Table-2 showing drug chart (drugs used for patient in current case study)

Brand name	Generic name	ROA	Frequency	Dose
Atorvastatin	Atorvastatin	Oral	O.D	10mg
Ecospirin	Aspirin	Oral	O.D	75mg
B.complex	Vitamin B complex	Oral	O.D	_
Sodium valproate	Sodium valproate	Oral	B.D	500mg
Pantop	Pantoprazole	Oral	O.D	40 mg
Leviteracetam	Leviteracetam	Oral	B.D	500mg
Ultracet	Tramadol and acetaminophen	Oral	S.O.S	37.5mg +325mg
Elavil	Amitriptyline	Oral	O.D	10mg

On 1st day Tablet Atorvastatin 10mg OD is a HMG-CoA reductase inhibitor given to decrease LDL levels in blood, Tablet Ecospirin 75mg OD is a COX-2 inhibitor having anti clotting effects and anti-inflammatory action given to prevent further strokes, Tablet sodium valproate 500mg BD and Tablet leviteracetam 500mg BD are given as anti epileptic drugs. Tablet B complex OD is given as vitamin supplement are given.

From 2nd -7th day Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD a proton pump inhibitor given to decrease acidity caused by drugs are given.

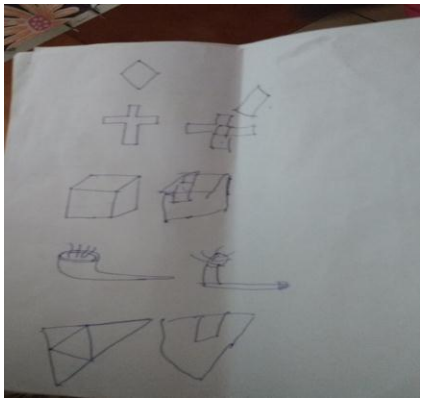
From 8th-12th day patient complained of body pains. So, Tablet Ultracet SOS an opioid analgesic given to relieve pain along with Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD.

From 13th -16th day patient have decreased body pains and physiotherapy was done along with Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD were given.

On day 17th patient was referred to psychiatric department to examine her cry's and spells and treated with Tablet Amitriptyline 10 mg OD used to treat depression caused due to Pseudobulbar palsy along with Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD , Tablet B.complex.

From 18th – 20th day patient was treated with Tablet Amitriptyline 10 mg OD, Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg O.D, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD, Tablet B.complex.

Patient has shown improvement in her health status and discharged on 20th day along with medication Tablet Amitriptyline 10 mg OD, Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD, Tablet B.complex. The patient's representative was counseled regarding patient's disease, proper use of medication, diet to be taken and life style modifications.



Picture -1 showing dysgraphia of patient in Hand writing.

Picture -2 showing wrist drop of left hand patient.

CONCLUSION

Pseudobulbar palsy is a medical condition which limits ability to control the muscles in face. It is usually caused by a variety of neurological disorders resulting in the damage (bilateral degeneration) to the neurons of the brain stem, specifically to the corticobulbar tract (upper motor neuron tract to cranial nerve motor nuclei). The present case is a pseudobulbar palsy where the patient had a weakness of left upper and lower limbs since 12 years. But effective treatment with Tablet Atorvastatin 10mg OD, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD, Tablet B.complex patient showed positive prognosis after a long duration of therapy for 20 days. As a clinical pharmacist we have done the therapeutic drug monitoring (TDM) and drug utilization review for the patient for the occurrence of any drug related side effects or drug interactions and for positive prognosis of the patient with the collaborative effects of physician, clinical pharmacist and the nursing staff. The patient exhibited positive prognosis and discharged with the medication Tablet Atorvastatin 10mg O.D, Tablet Ecospirin 75mg OD, Tablet sodium valproate 500mg BD, Tablet leviteracetam 500mg BD, Tablet pantoprazole 40mg OD, Tablet B.complex. Patient representative was counseled about the disease, proper use of medication, diet to be taken like soft diet and eternal feeding during severe dysphagia, vitamin and fiber rich food should be taken and life style modifications like exercise every day and physiotherapy in weakened muscles until full recovery.

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