

Case Report on Bell's Palsy and Diabetic Ketoacidosis due to Uncontrolled Diabetes

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Abstract

Diabetic ketoacidosis (DKA) is a life-threatening condition seen in diabetes in which body cannot use glucose as a fuel instead uses fat and produces ketones (chemicals) and finally results in diabetic ketoacidosis. Bell's palsy is a sudden weakness or paralysis of the muscles on one side of the face due to mal-function of cranial nerve VII (facial nerve), which stimulates the facial muscles. Bell's palsy affects about 23 of 1,00,000 people at some time. The cause of Bell's palsy is unknown, but it may involve swelling of the facial nerve as a reaction to an immune disorder or a viral infection. Swelling causes the nerve to be compressed and its blood supply to be reduced. In this case facial nerve has got damaged due to diabetic ketoacidosis (mononeuropathy). Risk factors are the virus that have been linked to Bell's palsy include the virus that causes: cold sores and genital herpes (herpes simplex), chickenpox and shingles (herpes zoster), mononucleosis (Epstein-Barr), cytomegalovirus infections, respiratory illnesses (adenovirus), German measles (rubella), mumps (mumps virus), and flu (influenza B). Clinical features include numbness in the face, headache, eye can't get closed and gets dry resulting in eye damage. Diagnosis is done by CT-scan, MRI scan and blood tests. Treatment involves corticosteroids and anti-viral drugs. Bell's palsy can be cured in few months such as 2 to 3 months with regular medications and physiotherapy.

Keywords: Diabetic ketoacidosis (DKA); Bell's palsy; Mononeuropathy

illnesses (adenovirus), German measles (rubella), mumps (mumps virus), and flu (influenza B) [2,3].

Diabetic Ketoacidosis (DKA)

Diabetic ketoacidosis (DKA) is a life-threatening problem that affects people with diabetes in which the body cannot use sugar (glucose) as a fuel source because there is no insulin or not enough insulin. Hence, fat gets used as the fuel and this fat gets broken down to generate fuel to the body and chemicals called ketones, gets build up in the body which results in DKA [1].

Bell's Palsy

Bell's palsy is sudden weakness or paralysis of the muscles on one side of the face due to mal-function of cranial nerve VII (facial nerve), which stimulates the facial muscles. Bell's palsy affects about 23 out of 1,00,000 people. Generally, the cause of Bell's palsy is unknown, but evidence suggests that it may be caused by viruses. And it involves swelling of the facial nerve as a reaction to an immune disorder or a viral infection. Swelling causes the nerve to be compressed and its blood supply to be reduced [2].

Causes

Evidence suggests that herpes simplex, a viral infection, is usually the cause. Lyme disease is also a common cause of Bell's palsy, especially in the northeastern United States. In blacks, sarcoidosis is common cause. Although the exact reason for Bell's palsy is often linked to exposure to a viral infection. Viruses that have been linked to Bell's palsy include the virus that causes: cold sores and genital herpes (herpes simplex), chickenpox and shingles (herpes zoster), mononucleosis (Epstein-Barr), cytomegalovirus infections, respiratory

Risk Factors

- Pregnant especially during the third trimester or who are in the first week after giving birth.
- People have an upper respiratory infection, such as the flu or a cold.
- People with diabetes.
- People with a family history of recurrent attacks (genetic predisposition) [4].

Symptoms

- Pain behind the ear may be the first symptom, developing several hours or even a day or two before the facial muscles weaken. The weakness reaches its maximum by 48 hours.
- Numbness or heavy feeling in the face, even though sensation remains normal.
- Headache
- When the upper part of the face is affected, closing the eye on the affected side may be difficult. As the eye can't be closed completely, it may become dry resulting in pain and even results in blindness due to absence of lacrimal secretions in the eyes.
- Bell's palsy interferes with the production of saliva and tears. The ear on the affected side may perceive sounds as abnormally loud (a condition called hypeacusis) because the muscle (located in inner ear) that stretches the eardrum is paralyzed. (The facial nerve contains parasympathetic fibers to the nose, palate and lacrimal glands. Hence, damage of facial nerve results in inhibition of secretions in the nose, plate and lacrimal glands) [2].

Diagnosis

Bell's palsy is diagnosed based on symptoms, patient's history, computed tomography (CT), magnetic resonance imaging (MRI) and Blood test- to check for Lyme disease or Sarcoidosis. Physical examination including Voluntary facial movements, such as wrinkling the brow, showing teeth, frowning, closing the eyes tightly (inability to do so is called lagophthalmos), pursing the lips and puffing out the cheeks [2].

Treatment

The aims of treatment in the acute phase of Bell's palsy include strategies to speed recovery and to prevent corneal complications. Eye care includes eye patching and lubrication, lubricating drops should be applied frequently during the day and an eye ointment should be used at night. Strategies to speed recovery include physical therapy, corticosteroids and antiviral agents [5-7].

An antiviral drug called Acyclovir is given to prevent the virus from replicating. Corticosteroids, such as Prednisone, are given by mouth to reduce swelling of the nerve. For maximum benefit, treatment should start within 2 days of the development of symptoms and be continued for 1 to 2 weeks.

If paralyzed facial muscles prevent the eye from closing completely, the eye must be protected from dryness to reduce the risk of blindness. Eye-drops consisting of artificial tears or a salt (saline) solution are applied to the eye until it can close completely. An eye patch may also be needed.

Mild electrical stimulation of the nerve and massage of the facial muscles have no proven benefits. If no facial movement has returned after 6 to 12 months, an operation-called hypoglossal-facial anastomosis may be performed to join cranial nerve XII (hypoglossal nerve) to the facial nerve. This operation may partially restore facial movement, but it also causes difficulties in eating and speaking and is therefore rarely performed.

When facial paralysis is partial, most people recover completely within 1 to 2 months whether they are treated or not. When the paralysis is total, the outcome varies. Many people do not recover completely; the facial muscles may remain weak causing the face to droop [2].

Relation between Diabetes and Bell's Palsy

Diabetes complications can include damage to nerves called neuropathy. One of the versions of diabetic nerve damage is the damage of a single nerve (also called mononeuropathy). In this case the single nerve is the facial nerve [5].

Case Study

A male patient of age 35 years visited emergency department in Rhojini Super Specialty Hospital. He complained that he had vomiting of 3 episodes, giddiness, shortness of breath (SOB) and fever one day before his admission. His past history shows that he is a known diabetic and hypertensive patient but non-adherent to medication and had jaundice 4 months back. He is also a chronic alcoholic. On examination patient was irritable. His BP was 130/80 mm Hg, pulse rate (PR) was 142 beats per minute (b/min), respiratory system was normal and clear and glucose-random blood sugar (GRBS) was 445

mg/dl. The provisional diagnosis was made to be Diabetic Ketoacidosis (DKA).

In emergency department, the patient was immediately given normal saline 2 pints with rapid flow. Nebulization was given along with injection (inj.) Human Actropid insulin of 50 U in 50 ml normal saline (NS) at the rate of 8 ml/hr and inj. Sodium bicarbonate 100 ml IV was given immediately.

After stabilization the patient was shifted to MICU (Medical Intensive Care Unit) and there his treatment continued with inj. Sodium bicarbonate 50 ml IV twice daily, an antibiotic inj. cefoperazone sodium 1.5 g IV twice daily in order to prevent hospitalized infections, inj. Pantoprazole 40 mg IV and IV fluid NS 5 pints were given (2 pints in 1 hr, next 2 pints at a rate of 250 ml/hr and last 1 pint at a rate of 150 ml/hr). Neutralizing dose of inj. Human Actropid Insulin 20 U was given in 1 pint DNS at a rate of 30 ml/hr. The patient was advised for laboratory tests such as complete blood picture (CBP), erythrocyte sedimentation rate (ESR), blood urea, serum creatinine, liver function tests, serum electrolytes, urine analysis, chest X-ray, serum amylase, serum lipase and arterial blood gas (ABG) analysis.

His total white blood cells (T.WBC) and neutrophils (N) were found to be increased i.e. T.WBC=13,000 cells/cmm, and N=87%. ESR was increased (ESR=57 in 1st hr). Blood urea and creatinine was normal. Liver function test showed decreased serum glutamic-pyruvic transaminase (SGPT) (SGPT=5.1 U/L). Serum electrolytes were normal. Urine analysis showed presence of albumin, sugar in urine, ketone bodies, epithelial cells, pus cells and red blood cells (RBCs). Chest X-ray was normal. Serum amylase was normal but serum lipase was increased (sr. lipase=85.7 U/L).

On 2nd day, the patient complained about deviation of mouth to left side causing angle mouth drop and unable to close the left eyelid. Patient's fasting blood sugar (FBS) was 212 mg/dl, BP was 90/60 mm Hg, PR was 117 b/min, ABG analysis showed pH was 7.3, PCO₂ was 9.3 mm Hg (highly decreased), PO₂ was 171.9 mm Hg (highly increased), HCO₃ was 4.7 mmol/L (highly decreased). Ultrasound scan of abdomen showed B/L Grade I Renal parenchymal changes. Same medication was continued and IV fluid NS was added at a rate of 100 ml/hr. Patient was advised for neurologist opinion and diagnosed as Bell's palsy.

On the 3rd day, the patient was conscious but irritable. The patient was afebrile and his BP was 100/60 mm Hg, PR was 122 b/min, RR was 18/min and GRBS was 210 mg/dl. High amount of urine sugars were seen with no ketone bodies. ABG analysis showed respiratory alkalosis with metabolic acidosis. Patient's oral intake was decreased. Same treatment was continued with an addition of Tab. Acyclovir 800 mg 5 times.

On 4th day, patient had no complaints. His BP was 110/70 mm Hg, PR was 117 b/min, GRBS was 96 mg/dl. ABG analysis showed respiratory alkalosis with metabolic acidosis. Same medications were given with a change of Tab. Acyclovir 800 mg from 5 times to 3 times (TID) a day, inj. KCL (Potassium chloride) 40 mg in 200 ml NS was given over a period of 2 hrs, Lubricant eye drops, inj. Human Actropid Insulin 10 U and inj. Sodium bicarbonates 50 ml in 25 ml saline BD were administered. Serum electrolytes were normal.

On 5th day, patient had no new complaints. His BP was 130/70 mm Hg, PR was 140 b/min, RBS was 294 mg/dl, patient was febrile (T=100°F), serum electrolytes were normal. ABG analysis showed

Respiratory alkalosis with metabolic acidosis with PH was 7.5, PO₂ was 117 mm Hg, PCO₂ was 20.6 mm Hg). Same medications were continued by holding inj. Sodium bicarbonate. On 6th and 7th day, patient was conscious and coherent with no fresh complaints and same treatment was continued.

On 8th day, patient had no new complaints and on his request he was discharged to higher center for further treatment. And his discharged medications include Tab. Pantoprazole 40 mg OD, Tab. Acyclovir 800 mg TID, Tab. Prednisolone 50 mg OD, Tab. Vitamin supplement 1 tab OD, Lubricating-eye drops TID, Inj. Human Actropid Insulin 10 U s/c 4th hourly.

Conclusion

In this patient, Bell's palsy was seen as a result of uncontrolled diabetes as the patient was non-adherent to his diabetic medications. Bell's palsy is one of the complication of diabetes i.e. diabetic neuropathy which can also result in mononeuropathy. In this case, facial nerve has got damaged due to the uncontrolled diabetes which

also resulted in diabetic ketoacidosis and resulted in Bell's palsy. This condition can be cured in few months such as 2 to 3 months, with regular medications and physiotherapy.

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