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Sensorineural Hearing Loss in a Patient with Type 2 Diabetes Mellitus

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Abstract: ***A Balinese woman, 52 years old, complained of hearing loss in both ears with dizziness. Patient had history of diabetes mellitus and hypertension, not taking medication regularly. She was also suffering from chronic kidney disease stage 4, dyslipidemia. Based on audiometric test, the result was sudden sensorineural hearing loss moderate severe dextra et sinistra. In addition to insulin therapy, antihypertensive, anti-platelet aggregation, folic acid and statin, patient also given oxygenation, steroid, vitamin B complex, pentoxifylline and hyperbaric therapy. After two weeks the patient's hearing improves, although not yet returned to normal***

Keywords : sudden hearing loss, sensorineural hearing loss, diabetes mellitus

1. Introduction

Diabetes mellitus is a chronic metabolic disorder characterized by hyperglycemia and changes in fat and protein metabolism. This disorder results in a number of microvascular complications that commonly affect the eyes and kidneys, which is accompanied by diffuse polyneuropathy in both somatic and autonomic nerve fibers(1). Neural networks and blood vessels play an important role in the function of auditory organs. Diabetes mellitus can damage the nerve cells and blood vessels, so it can also bring a negative impact on the organ of hearing. Very likely there is a relationship between the function of the organ of hearing with DM, because the disease affects the organs rich in blood vessels such as cochlea and/or the central nerves including the brain that plays a role in the auditory hearing (1).

Hearing loss in diabetic patients is usually bilateral, progressive, sensorineural especially at high frequency (1). However, in some cases people with diabetes mellitus, hearing loss may arise suddenly. Occurrence of cochlear ischemia or occlusion of blood vessels may be caused by thrombosis, embolus, vasospasm, or reduced blood flow. Some researchers report a strong association between diabetes mellitus and hearing loss (1,2). Quoted from Austin DF, Fukushima et al, suggest a link between diabetes mellitus and pathology changes in cochlea in the form of vascular stria thickening, vascular stria atrophy, and reduced hair loss, but no change in the spiral ganglion compared with controls. Bainbridge et al, in his study of type 2 DM patients with microvascular complications using pure audiometric measuring instruments found a strong association between hearing loss and type 2 DM (3).

2. Case

A Balinese woman, 52 years old, came to the hospital with a hearing loss complaint that occurred five days before admission. Hearing loss is felt more severe on the left ear than the right ear. Hearing loss was not accompanied by a buzzing sound in the right or left ear. The presence of fever, cough, nausea, vomiting, shortness of breath was denied. Patients also complained dizzines that occured along with decreased hearing so the patient difficult to perform usual daily activities. Eat and drink, urinate and defecate within normal limits.

Patient had history of diabetes mellitus since 2012 and did not take medication regularly. Drugs that are sometimes taken by patient was 5 mg tablet gibenclamide. Patient also had history of high blood pressure that is known since 2014. Patients never take medication for high blood pressure. History of heart disease, kidney, trauma, and drug allergy was denied by the patient. The patient's mother is said to have diabetes mellitus. Currently no family member has the same complaints as the patient. In his daily life the patient is a housewife.

From the physical examination obtained, the general condition of severe illness, with blood pressure 160/90 mmHg, pulse rate 98 times per minute, respiration rate 19 times per minute, axillary temperature 36.8 degrees Celsius, level of consiousness E4V5M6. There was a decrease in hearing ability in both ears, left ear was more severe than right ear. From the results of laboratory tests obtained, the full blood count: WBC 14.07 10^3/uL (Neu 11,32, Lym 2.06, Mo 0,59, Eo 0,01, Ba 0,06), Hb 9.65 g/dL, MCV 88,05 fL, MCH 30,05 pg, Hct 28,27%, Plt 350,40 10^3/uL, D-dimer 1,23. Chemical blood: SGOT 13,7 U/L, SGPT 17,50 U/L, albumin 2,8 g/dL, BUN 40,0 mg/dL, creatinine 2,87 mg/dL, sodium 130 mmol/L, potassium 5.6 mmol/L, blood sugar at 297 mg/dL, total cholesterol 203 mg/dL, LDL 136 mg/dL, HDL 53 mg/dL, triglyceride 203 mg/dL. The result of chest x-ray was the absent of lung infiltrates or nodules, normal bronchovascular pattern, with cardiomegaly with cardio thoracic ratio (CTR) 63%. From the electrocardiogram obtained normal sinus rhythm, with a frequency of 93 times per minute. Patients were further diagnosed with: Type 2

Diabetes Mellitus, dyslipidemia, chronic kidney disease (CKD) stage IV caused by diabetic kidney disease (DKD), mild normochromic normositer anemia on CKD, hyperkalemia, stage II hypertension, cardiomegaly caused by suspect diabetic cardiomyopathy dd/ Hypertensive heart disease (HHD) Patient received therapy: Diet CKD 35 kcal and 0.8 grams protein per kilogram body weight per day, infusion of NaCl 0.9% 12 drops per minute, insulin aspart 12 units every 8 hours, insulin glargine 12 units every 24 hours, simvastatin tablet 20 mg every 24 hours, valsartan tablet 80 mg every 24 hours, amlodipine tablet 5 mg every 24 hours, kalitake 1 sachet every 8 hours, folic acid tablet 2 mg every 12 hours, acetosal tablet 80 mg every 24 hour. The monitor is performed for fasting blood sugar and two hours after each meal every day.

Patient was also treated by Ear, Nose and Throat (ENT) Department. Based on audiometric test, the patient was diagnosed with sudden sensorineural hearing loss dextra et sinistra (SSHL D et S). Patient received therapy: 4 liters of oxygen per minute through nasal cannula for 15 minutes every 6 hours, ranitidine 50 mg every 12 hours intravenously, methyl prednisolone 31.25 mg every 12 hours intravenously tappering off, vitamin B complex 1 tablet every 12 hours, pentoxyfilin 400 mg tablet every 12 hours, hyperbaric therapy five times. Monitor performed on improvement of the patient's hearing. After undergoing treatment for approximately two weeks, the patient's hearing improved, although not yet returned to normal.

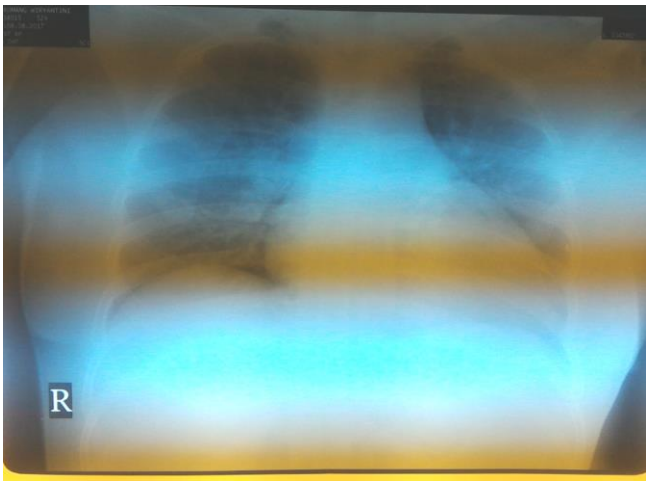

Figure 1: Chest X-ray of the patient

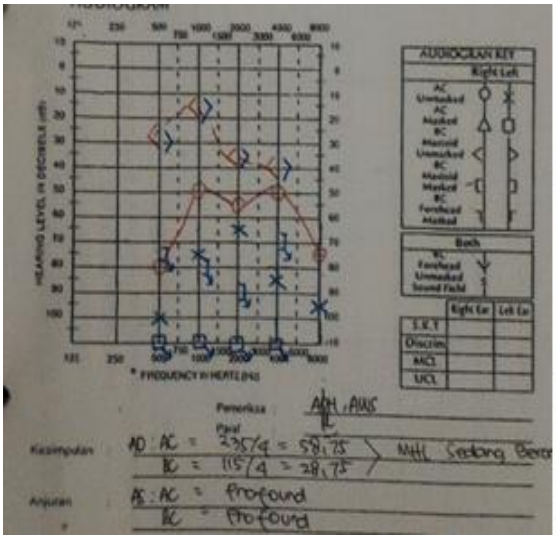

Figure 2: Result of Audiometric Test

Figure 3. Picture of the patient

3. Discussion

The number of diabetes mellitus, especially type 2 diabetes, according to the WHO report, in 2000 reached 171 million people and will be predicted to reach 366 million in 2030 or an increase of 114%. While in Asians, the increase will reach 141% in the same period (3). Quoted from Austin DF, Bainbridge et al., Obtained from 5742 participants on a national health and nutrition examination in which patients with diabetes mellitus suffered more hearing loss than those who did not diabetes mellitus. Hearing loss was 31% of patients with diabetes at 4000 Hz and 34% at 8000 Hz (2.5). Dalton DS et al., in the study of the association of type 2 diabetes and hearing loss, resulted in hearing loss as measured by pure audiometric tone of type 2 diabetes mellitus by 59% compared to 44% (4). Patient in this case was woman, Balinese, 52 years old and had uncontrolled diabetes mellitus for five years.

4. Anatomy of the Ear

The ear consists of three parts, namely the outer ear, middle ear and inner ear. The inner ear consists of two parts, the front part which is the hearing part called the cochlea and the back is the vestibule and the semicircular canal which is the balance organ. The cochlea is a 35 mm long bucket-shaped bone tube, consisting of a vestibule scale, a media scale, and a tympanic scale. The media scale or cochlear has a triangular cross section. The base of the triangle is known by the name of the basilar membrane that forms the basis of the cortic organ. The main components of cortical organs are outer hair cells, inner hair cells, supporting cells (Dieters, Hensen, Claudius), tektoria membranes and reticular lamina complexes. Supporting cells form the structure and support for cortical organ metabolism (6).

Inner Ear Vascularization

Arterial blood vessels for the inner ear are supplied by the labyrinth arteries or internal auditory arteries that are branches of the anterior-inferior cerebellum artery or directly from the basilar artery. This artery enters the inner ear from the back of the internal acoustic meatus and bifurcates into two namely (6):

1) The anterior vestibular artery that supplies the utriculus and the posterior sacculus extends to the anterior and lateral semicircular canals.
2) Communist cochlear artery, which branches into two namely: posterior cochlearis artery and vestibulokoklear artery that branches again into two, namely the cochlear and vestibular parts. The cochlear branch will supply the blood supply to the inferior part of the cochlear ducts, then join the koklearis ramus originating from the posterior cocklearis artery, whereas the vestibular branch mediates the posterior semicircular canal and most of the sacculus.

The veins of the veins in the inner ear come from the axes of the aquaductus koklearis and the vestibularis aquaductus plexus. Venues from the sensory areas in the vestibular such as the posterior vestibular venules that accommodate drainage of the sacculus and the posterior semicircular canal ampules and the anterior vestibular venules that accommodate the drainage of the utriculus will flow into the coccarate aquaduct plexus. The vestibularis aquaductus plexus is an anastomosis of the veins derived from the vestibular non-sensory areas of semicircular canal. These veins run parallel to the aquaductus and receive flow from the veins in the endolymphatic sac (6).

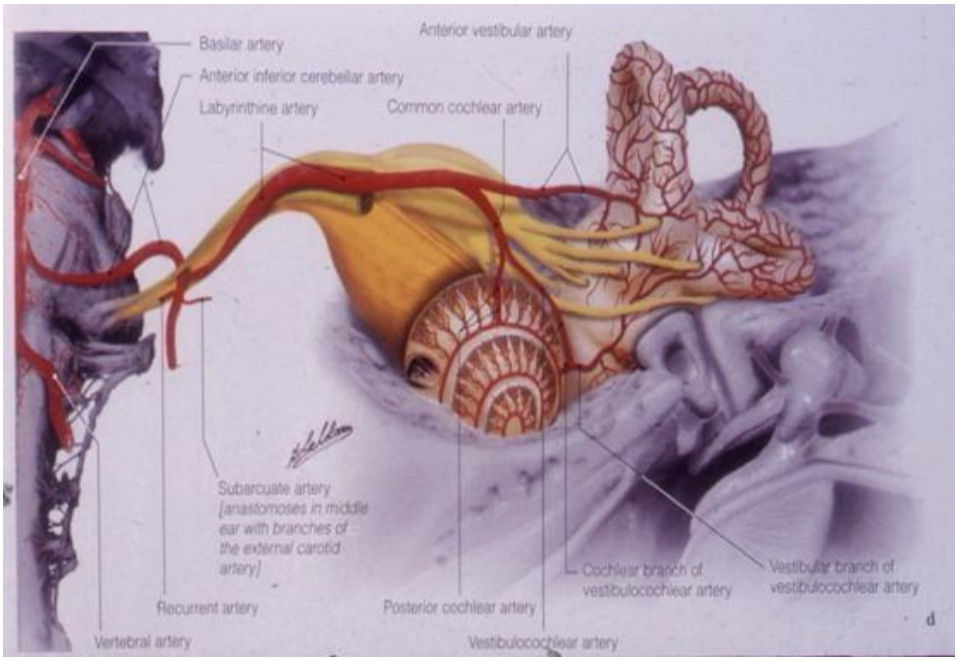

Figure 4: Inner ear vascularization (6)

Pathophysiology of Hearing Loss in Type 2 Diabetes Mellitus

DM is a group of metabolic diseases with characteristics of hyperglycemia that occur due to abnormalities of insulin secretion, insulin work or both. Chronic hyperglycemia in diabetes mellitus is associated with long-term damage, dysfunction or failure of several organs, especially the eyes, kidneys, nerves, heart and blood vessels. Hearing loss in patients with Type 2 DM has characteristics, progressive or acute, type sensorineural especially at high frequencies. Several studies have found a vascular abnormality in the inner ear that histopathologically shows microangiopathic changes that form precipitates in the blood vessel wall resulting in thickening seen with periodic acid schiff (PAS). This microangiopathic disorder occurs mainly in the vascular stria capillary vessels, subsequently occurring in the internal auditory artery, modiulus, in the spiral ganglion vasa nervosum and demyelinization of the auditory nerve (2.12).

The mechanism theory of hearing impairment in patients with DM is due to microangiopati. These changes occur thoroughly in blood vessel capillaries with clinical manifestations especially in the kidneys, heart, brain, retina and peripheral nerves. Microangiopathy is also experienced by blood vessels in the inner ear. Microangiopathy in the

labyrinth especially concerns the stria vascularis, internal auditiva artery (2.5). The exact mechanism of this change can not be explained, but when it comes to the fact that further complications of DM occur in insulin-dependent cells and tissues for glucose transport, it seems that hyperglycemic play a major role in the process. Prolonged hyperglycaemia, has been known to stimulate the reaction of non-enzymatic protein glycosylation, which takes place on various tissues of the body. Several clinical studies provide information on the correlation between the duration of hyperglycaemia and microangiopathy progression in DM patients. Controlled status of glycemia close to normal limits may inhibit or even prevent microangiopathy (13).

Glucose is bound to proteins by non-enzymatic chemical reactions. This process begins with the attachment of glucose to the amino acid group, which continues with a series of biochemical reactions with the result of the formation of amadory product, the reaction further produces a final product called advanced glycosilation end product (AGEP) which is irreversible. This glycosylation reaction occurs in long live proteins, including collagen and basement membranes of blood vessels. One form of AGEP in DM is 2-furoyl-4 (5)-(2-furanyl)-1-H-imidazole (FFI) which is heavily buried in DM body tissue. In this glycosylation reaction, free radicals are formed as a result of glucose-auto oxidation that takes place at the time of AGEP formation of the amadory product, which is highly reactive oxidant which has muscle-toxic properties such as the effects of denaturation and aggregation (13,21). Increased production of AGEP reduces the elasticity of blood vessel walls (arteriosclerosis) and results in the attachment of plasma proteins on the basement membrane, so that the blood vessel wall thickened with increasingly narrow lumen. Pathological changes that occur in microangiopathy are basically (9):

a) Thickening of the basement membrane of the capillary vessels resulting in narrowing of the capillary lumen.
b) Hemodynamic changes result in organ dysfunction.
c) Changes in blood viscosity and platelet function that spur the formation of microtrombus as a result of microvascular blockage.

As a result of microangiopathy of the cortic organ will occur atrophy and reduced hair cells. Neuropathy occurs due to microangiopathy in nerve vasa nervosum VIII and spiral ligament venous resulting in spiral ganglion atrophy and demyelination of nerve fibers VIII. Wackym and Linthicum (1986) suggest that hair cells are atrophied by the accumulation of metabolic toxic substances in endolymphs due to disruption of blood vessel absorption around endolymphatic sacs. Hearing loss mainly occurs at high frequencies. This appears to be related to the lack of tissue glycogen as an energy source in DM patients. Transduction in cortical organs requires energy (ATP), which is derived from glycogen (3).

AGEP occurs in several cellular structures and various tissues. Due to hyperglycemia and the activation of CCP enzymes is the buildup of AGEP in type IV collagen. Type IV collagen is found in the periphery of the auditory system which includes the textural membrane, the basement membrane, the vascular stria, the enameled nerve fibers, the spiral ligaments, the spiral prominence, the spiral limbus, the media scale, and the epithelial cells. AGEP accumulation that occurs in collagen is caused by post-translational protein modification and increased cross-linking protein. Hence the hypothesis that AGEP buildup causes the membrane becomes more fibrous and more inflexible, resulting in inarticulation with outer hair cells and decreased sound transduction. This cellular change causes cell communication to decline, both within the cell itself and between cells. If it occurs in the cochlear of a DM patient, the perilimfa and endolymphal fluids become inhibited so that it may harm the homeostasis of the cochlear ions (12).

The function of hemostasis in the body is determined by various factors, namely: blood vessels, platelets, coagulation and fibrinolysis. Changes in the pathophysiology of blood flow may occur in diabetes mellitus including increased blood plasma viscosity, increased fibrinogen levels, decreased fibrinolytic activity, platelet hyperactivity, increased plasma coagulability. Factors involved are: Hyperaggregation and platelet dysfunction, increased von Willebran factor (vWF), increased prothrombotic factors such as tissue factor (TF) and fibrinogen, increased plasminogen activator inhibitor-1 (PAI-1), and decreased bioavailability of nitric oxyde (NO) (9).

The vascular endothelial cells are between the bloodstream and blood vessel walls in the regulation of blood function. Endothelial cell functions include maintaining blood vessel tone, hemostasis and antithrombotic function (antitrembosit, anticoagulant, fibrinolysis), and inflammation. Endothelial cells produce NO, which acts as a vasodilator of blood vessels, platelet inhibitors, inhibits migration and smooth muscle cell proliferation. NO is synthesized by endothelial cells from L-arginine by the enzyme NO synthase. NO biological effects on blood vessels: Vasodilators for blood vessels, as platelet inhibitors, are anti-inflammatory and anti-atherogenic, mediators of apoptosis for cells and smooth muscle. In patients with diabetes mellitus, a decrease in NO release, decreased bioavailability of NO, a defect response to NO that will lead to a protrombotic condition (9).

In DM patients increased platelet activity. Some investigators report an increase in spontaneous platelet aggregation in vitro and increased adhesion function associated with increased vWF levels. In DM, hyperglycemic conditions will alter platelets by disrupting calcium balance resulting in platelet hyperactivity and platelet aggregation including platelet form changes and mediator release (9).

Patients in this case come with a sudden hearing loss complaint on both ears, perceived hearing loss in the left ear is more severe than the right ear. Patients with a history of diabetes for five years and uncontrolled. From the laboratory examination found an increase in D dimer value of 1.23. This indicates the presence of thrombus that tends to occur in DM patients and the cause of vascularization disorders in the inner ear, causing a sudden hearing loss.

5. Diagnosis

Diagnosis of hearing loss is established from auditory results in the form of pure tone audiometry and otoacoustic emission (OAE) (6.12).

Pure tone audiometry

Pure tone audiometry is a tool for measuring one's ability to hear pure tone sounds. This tool can produce pure sounds of several frequencies of 250, 500, 1000, 2000, 4000, and 8000 Hz. Sensoryineural hearing impairment is enforced when there is a decrease in auditory acuity characterized by increasing pure threshold value of air conductivity and bone conductivity (BC) with a difference between the two not more than 10 dB with a hearing threshold greater than 25 dB. The degree of deafness is calculated using the Fletcher index. In determining the degree of deafness, only air shear thresholds are calculated. Deafness, according to ISO (International Standrad Organization): 0 - 25 dB: normal,> 25 - 40 dB: light deafness,> 40 - 55 dB: moderate deafness,> 55 - 70 dB: moderate to severe deafness,> 70 - 90 dB : severe deafness,> 90 dB: very severe deafness (6).

Otoacoustic emission (OAE)

Otoacoustic emission is the cochlear response produced by the outer hair cells emitted in the form of acoustic energy. Use of OAE to analyze hearing impairment, more objective and more sensitive than a pure tone audiometer. The OAE examination is an electrophysiological examination to assess the function of the cochlea objectively, automatically (using pass/refer criteria) (6.8). The diagnosis of sudden sensorineural hearing loss of dextra et sinistra (SSHL D et S) in this patient is established from a pure tone audiometric examination. Sensorineural hearing loss in this patient occurs for high-frequency tunes.

6. Management

Systemic corticosteroids

Various studies on the use of corticosteroids in sudden deafness have been published. There is laboratory evidence suggesting an inflammatory cascade of cell death in sudden deafness patients, modified by steroid therapy. The given corticosteroids are oral, intravenous, and / or intratymic synthetic glucocorticoids, including prednisone, methylprednisolone, dexamethasone. Corticosteroids are thought to have anti-inflammatory effects and ability to increase cochlear blood flow. Currently, the standard of sudden hearing deafness is by tappering off the oral cortiosteroid. A randomized controlled trial comparing oral steroid therapy with placebo in 67 patients showed significantly better improvement in the group of patients with oral steroid therapy than in the placebo group (61% vs. 32%, p <0.05). For maximum treatment results, the recommended oral dose of oral prednisone therapy is 1 mg / kg / day of single dose at a maximum dose of 60 mg / day for 10-14 days. The equivalent dose of prednisone 60 mg is equivalent to 48 mg methylprednisolone and dexamethasone 10 mg. A representative data using treatment regimen with maximum dose for 4 days followed tapering off 10 mg every two days (5).

Prednisone side effects include insomnia, dizziness, weight gain, sweating, gastritis, mood swings, photosensitivity, and hyperglycemia. Other side effects are quite severe, but rarely found, namely pancreatitis, bleeding, hypertension, cataracts, myopathy, opportunistic infections, osteoporosis, and osteonecrosis. Therefore, to minimize risk, patients with systemic medical conditions, such as insulin-dependent diabetes mellitus (IDDM), uncontrolled diabetes, labile hypertension, tuberculosis, and peptic ulcers are not recommended for systemic corticosteroid therapy (5).

Intratimpani corticosteroids

Intratimpani corticosteroid therapy as a substitute for systemic corticosteroid therapy in patients not improving with systemic corticosteroids. Intratimpani corticosteroid therapy can be an alternative for diabetic patients who can not take systemic corticosteroids. Commonly given intratimpani steroids are dexamethasone or methylprednisolone. The concentrations of corticosteroids used varied, most studies recommend dexamethasone 10-24 mg/mL and methylprednisolone 30 mg/mL or more. Adverse effects of intratimpani therapy that should be anticipated are local effects, such as otalgia, dizziness, vertigo, perforation of the tympanic membrane, or infection (otitis media) (5).

Hyperbaric oxygen therapy

Hyperbaric oxygen therapy has been applied as adjunctive therapy in cases of sudden deafness. This therapy provides 100% oxygen with a pressure of more than 1 ATA (atmosphere absolute). This therapy aims to increase the oxygenation of cochlea and perilimfe, so it is expected to deliver oxygen with higher partial pressure to the tissues, especially cochlea which is very sensitive to ischemic state. Guideline American Academy of Otorynolaryngology-Head and Neck Surgery (AAO-HNS), hyperbaric oxygen therapy should be performed within 2 weeks to 3 months from the time of sudden diagnosis of deafness. Younger patients respond better than older patients (age varies between 50-60 years). The important things to consider in hyperbaric oxygen therapy are the benefits and risks of side effects. This therapy has side effects such as damage to the ears, sinuses and lungs due to pressure changes, temporarily worsening myopia, claustrophobia, and oxygen poisoning (5).

Other pharmacological therapy

Vasoactive agents, thrombolytics, vasodilators, or antioxidants have been attempted to improve cochlear blood flow, but there is no evidence of treatment success. Prostaglandin E1 has shown benefit as a vasodilator and inhibitor of platelet aggregation. Naftidrofuril can also be a vasodilator with antagonistic effects on serotonin and thromboxane A2. Ginkgo biloba extracts containing flavones and terpenes can prevent the development of free radicals and act as vasodilators. Pentoxifylline inhibits platelet aggregation and increases the flexibility of erythrocytes and leukocytes thereby improving the viscosity of blood, especially capillaries. Dextrans can improve microcirculation because it has antithrombotic effects. HES (hydroxyethyl starch) reduces hematocrit and platelet aggregation. Clinicians should be aware of the risks of side effects such as allergic reactions, bleeding, hypotension, arrhythmia, seizures, and drug interactions (5).

Diabetes mellitus therapy

Modality of diabetes mellitus management consists of education, medical nutrition therapy, physical exercise, and pharmacological intervention. Pharmacologic interventions are added if blood glucose targets have not been achieved with dietary and physical exercise settings. Pharmacological therapy includes (7,8,9):

Insulin secretagogues (Sulfonylurea and Glinid)

This sulphonylurea group drug has the main effect of increasing insulin secretion by pancreatic beta cells, and is the primary choice for patients with normal weight and less, but should still be given to patients with more weight. Avoiding prolonged hypoglycemia in various circumstances such as the elderly, renal and hepatic physiological disorders, lack of nutrition and cardiovascular disease, it is not recommended to use sulfonylureas (7,8)

Glinid is a drug that works the same way as sulfonylureas, with an emphasis on increasing first-phase insulin secretion. This class consists of 2 kinds of drugs: Repaglinid (benzoic acid derivatives) and Nateglinid (phenylalanine derivatives). It is rapidly absorbed after oral administration and rapidly excreted by the liver (8).

Insulin sensitivity enhancer (Tiazolidinedion)

Thiazolidinedione (rosiglitazone and pioglitazone) bind to the peroxisome proliferator activated receptor gamma (PPAR-γ), a nuclear receptor in muscle cells and fat cells. This group has the effect of lowering insulin resistance by increasing the amount of glucose transporting proteins, thereby increasing glucose uptake in the periphery. Thiazolidinedion is contraindicated in patients with class I-IV heart failure because it may aggravate fluid retention and liver dysfunction. In patients taking thiazolidindion periodic liver monitoring (8) should be regularly monitored.

Gluconeogenesis inhibitors (Metformin)

This drug has the main effect of reducing the production of liver glucose (gluconeogenesis), as well as improving peripheral glucose uptake. Mainly used in people with diabetes fat. Metformin is contraindicated in patients with impaired renal function (serum creatinine greater than 1.5 mg/dL) and liver, as well as patients with hypoxemia tendencies (eg cerebrovascular disease, sepsis, shock, heart failure). Metformin can have side effects of nausea. To reduce such complaints may be given at or after meals (8).

Alpha Glucosidase Inhibitors (Acarbose)

This drug works by reducing the absorption of glucose in the small intestine, so it has the effect of lowering blood glucose levels after meals. Acarbose does not cause hypoglycemic side effects. The most common side effects are flatulence (8,9).

Insulin

Insulin is needed in rapid weight-loss situations, severe hyperglycemia with ketosis, diabetic ketoacidosis, non-ketotic hyperglycemia hyperglycemia, failure with maxillary oral hypglycemic drug combinations, severe stress (systemic infection, major surgery, acute myocardial infarction, stroke), diabetes uncontrolled gestational mellitus with meal planning, DM patients with severe renal or hepatic dysfunction, and allergy to oral hypoglycemic drugs (8,9).

Patients in this case have received education related to diabetes mellitus and recommended activities for people with the disease. The diet given to this patient is a diet with 2100 kilocalories and 48 grams of protein. Patients also experienced complications of CKD stage IV caused by DKD, so the pharmacological therapy administered was insulin aspart with a dose of 12 units every 8 hours subcutaneously (if eating out one serving). And insulin glargine 12 units every 24 hours subcutaneously. Patients also receive 80 mg of acetosal therapy every 24 hours orally, which acts as an anti-platelet aggregation. Of the ENT department, the patient has been treated with: Oxygen 4 liters per minute through nasal cannula for 15 minutes every 6 hours, ranitidine 50 mg every 12 hours intravenously, methyl prednisolone 31.25 mg every 12 hours intravenous tappering off, vitamin b complex 1 tablets every 12 hours, 400 mg tablet pentoxifylline every 12 hours, and hyperbaric therapy for five times.

7. Summary

We reported a case of a patient, woman, Balinese, age 52, who came with a hearing loss complaint on both ears that occurred five days before admission. Patient has a history of diabetes mellitus disease for five years that are not well controlled. Physical examination by ENT colleagues, ensuring nerve deafness in the right and left ear, especially on high tones. During hospitalization, patients get diabetes mellitus therapy in the form of education and medical nutrition therapy, and pharmacological therapy such as insulin aspart and insulin glargin. Patients also receive 80 mg of acetosal therapy every 24 hours orally, which acts as an anti-platelet aggregation. From the ENT department, patient has been treated with: Oxygen 4 liters per minute through nasal cannula for 15 minutes every 6 hours, ranitidine 50 mg every 12 hours intravenously, methyl prednisolone 31.25 mg every 12 hours intravenous tappering off, vitamin b complex 1 tablets every 12 hours, 400 mg tablet pentoxifylline every 12 hours, and hyperbaric therapy for five times. Along with the controlled blood sugar levels of patients, the patient's hearing gradually improves, but has not been able to return as before.

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