Uzhhorod National University

Medical Faculty 2

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Management of chronic complications of diabetes mellitus

Guidelines for workshops

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Preface

In the guidelines presented data on the etiology, pathogenesis, clinical symptoms, diagnosis, differential diagnosis, treatment and prevention of chronic complications of Diabetes Mellitus. Recommended as a supplementary educational footage for students of IV, VI course of Medical faculty.

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CONTENT

CLASSIFICATION OF COMPLICATIONS OF DIABETES MELLITUS

Many people with diabetes mellitus eventually develop complications, especially if it is not controlled well. Even if diabetes is controlled well, complications can still occur. There are two important facts concerning complications of diabetes mellitus (DM). The first one is that short-term complications can be easy fixed and that proper treatment of DM usually delays complications. The second one is that long-term complications are difficult to control and can cause someone dies earlier than normal.
Complications of DM can be acute or chronic.

Acute complications include the following:

- comatose states, like ketoacidosis, hypoglycemia, hyperosmolar coma, etc.
- Local allergic reactions

Chronic complications are further subdivided into **macrovascular, microvascular and miscellaneous.**

Macrovascular complications:

- Atherosclerosis (it affects small arterioles)
- Cerebro-vascular disease (atherosclerosis of the internal carotid and vertebro-basilar arteries and their branches predisposes to cerebral ischemia)
- Ischemic heart disease (coronary atherosclerosis often occurs earlier and is more severe and extensive, than in those without diabetes, increasing risk of ischemic heart disease)
- Ischemia of lower limb (ie, gangrene): severe atherosclerosis of the ilio-femoral and smaller arteries of the lower limbs predisposes to gangrene. Ischemia of single toe or ischemic areas on the heel are characteristic of diabetic peripheral vascular disease. This is due to the involvement of much smaller and more peripheral arteries.
- Atherosclerosis of renal arteries and their intrarenal branches causes chronic nephron ischemia. It is a significant component of multiple renal lesions in diabetes.

Microvascular complications

Peripheral neuropathy with trophic ulceration:

Four types of diabetic neuropathies develop, including (1) peripheral distal symmetrical polyneuropathy, predominantly sensory; (2) autonomic neuropathy; (3) proximal painful motor neuropathy; and (4) cranial mononeuropathy (ie, III, IV, VI). Sensory and autonomic neuropathy is due to axonal degeneration and segmental demyelisation. Motor neuropathy and cranial mononeuropathy is due to vascular disease in blood vessels supplying nerves.

Peripheral neuropathy is the damage of nerve fibers outside of the brain or spine. Peripheral neuropathy can cause a gradual loss of sensation starting at the hands and feet, which sometimes moves up arms and legs. Loss of feeling and poor blood

circulation makes the body more susceptible to ulcers (open sores) and gangrene (tissue death due to poor blood supply or infection of wound). Peripheral neuropathy can also couses dizziness when standing up as well as impotence in men.
Ulcers (open sores) on the feet in severe cases can develop into gangrene – is death of a tissue, usually due to loss of blood supply.

Diabetic retinopathy, cataract, glaucoma

Diabetic retinopathy is due to retinal small vessel abnormality leading to hard exudates, hemorrhages, and microaneurysms (it does not affect acuity). Proliferative retinopathy develops due to extensive proliferation of new retinal small blood vessels. A sudden loss of vision can occur due to vitreous hemorrhage from proliferating new vessels or retinal detachment. Edema and hard exudate or retinal ischemia leads to maculopathy, that causes a marked reduction of acuity. Cataract is frequent in people with diabetes also. Glaucoma relates to the neovascularization of the iris, rubeosis iridis.

Diabetic nephropathy

Hyaline arteriosclerosis, a characteristic pattern of wall thickening of small arterioles and capillaries is responsible for ischemic changes in kidneys leads to diabetic nephropathy, which is characterized by proteinuria, glomerular hyalinization (Kimmelstiel-Wilson), and chronic renal failure.

Miscellaneous complications

Skin infections:

People with diabetes are susceptible to various types of infections. The most common sites affected are the skin and urinary tract system. High level of sugar in the blood makes much more difficult for the body to fight against infections. This can lead to infections of the urinary tract, which is the part of the body that deals with the formation and excretion of urine (pee). Skin infections can also result, as vaginal yeast infections (a type of infection of the female reproductive organ). Increased risk of staphylococcal follicular skin infections, superficial fungal infections, cellulitis, erysipelas, and oral or genital candida infections exists. These patients develop frequent lower urinary tract infections and are at increased risk of acute pyelonephritis.

Necrobiosis lipoidica

Local fat atrophy or hypertrophy at injection sites usually improves by switching to human insulin and injecting it directly into the affected area. Patients do not require any specific treatment of local fat hypertrophy, but injection sites should be rotated.

DIABETIC NEPHROPATHY

Diabetic nephropathy (intercapillary glomerulosclerosis) is a significant life-threatening kidney disease, that is due to the adverse effects of glucose-induced preglomerular vasodilation on glomerular hemodynamics and usually occurs in patients with onset of the diabetes before 20 years-age. Diabetic nephropathy does not occur in all diabetics. The overall risk of developing diabetic nephropathy varies between about 10% of type II diabetics (diabetes of late onset) to about 30% of type I diabetics (diabetes of early onset). There are many factors, that affect the individual risk of developing diabetic nephropathy. These factors include:

- poor blood sugar control
- high blood pressure
- family anamnesis of kidney disease or hypertension (relatives have had them)
- diabetes began in teens, etc.

Some polymorphisms in the various factors involved in its pathogenesis, it can modulate course of this disease from one person to other. Although end-stage renal disease (ESRD) is one of the most severe complications of type 1 DM, the incidence of ESRD has been very low, 2.2% at 20 years after diagnosis and 7.8% at 30 years after diagnosis.

Diabetic nephropathy is connected with different processes, including diffuse glomerulosclerosis, thickening of glomerular capillars' basic membrane, arterio- and arteriolosclerosis, tubular-interstitial fibrosis, etc. Clinical symptoms can be defined after 12-20 years of DM. But some functional changes and anatomical disorders develop much earlier. Thus, already in diabetes onset the inlargement of kidneys, increase of glomerular filtration rate (GFR) are observed. After diabetes compensation, kidney size becomes normal, but GFR is still higher, than normal for 2-5 years. Kidney biopsy, performed in this period, establishes thickening of glomerular capillars' basic membrane, which indicates the initial (histological) stage of diabetic nephropathy. Inspite of anatomical disorders progression, there are no more clinical signs in patients during 12-18 years. Transit proteinuria, that occurs after physical activity or orthostasis, is the first clinical symptom of diabetic nephropathy. Tiny amount of protein, appearing in the urine, is called microalbuminuria (30-300 mg/day); kidney function may be normal at this point. Later it transforms into stabile proteinuria (more than300 mg/day) with normal or decreased GFR. Significant increase of proteinuria (more than 3 g/day)
is followed by disproteinemia with hypoalbuminemia, hypergammaglobulinemia, etc. At the same time nephrotic syndrome develops in 40-50% of diabetic patients. The existance of stabile proteinuria for 2-3 years is followed by azotemia (rest nitrogen more than 100 mg%), increased blood level of creatinine and urea are established, GFR decreases. Such condition leads to the development of clinical syndrome of renal insufficiency in half of diabetic patients, in 80-90% –with hypertension, combined with cardiac decompensation or pulmonary edema.

Pathogenesis of diabetic nephropathy

Reversible changes:
1. Rised GFR without the increase of renal plasma flow.
2. Proteinuria with hyperglycemia, insulin deficiency, more intensive in case of physical activity or orthostasis.
3. Accumulation of immunoglobulins, protein degradation products in mesangium, its hyperplasia.
4. Decrease of ability of distal renal tubules to secrete hydrogen ions.

Irreversible changes:
1. Increase of collagen synthesis in glomerular base membrane.
2. Hyaline arterioles sclerosis with juxta-glomerular apparatus damage.
3. Atherosclerosis of arteries with kidney failure.
4. Necrosis of renal papilla.

Diabetic nephropathy has latent, symptomatic and terminal forms. The stages of diabetic nephropathy are established according to the classifications of Mogensen (1983), based on the laboratory-clinical data:
I. The stage of hyperfunction – occurs during onset of diabetes and is characterized by hyperfiltration, hyperperfusion, hypernephrotrophy and normoalbuminuria (<30 mg/day).
II. The stage of initial renal changes. It is characterized by thickening of glomerular basic membrane, expansion of mesangium, hyperfiltration and normoalbuminuria (<30 mg/day). These changes occur in diabetes duration over 5 years.
III. The stage of initial diabetic nephropathy (incipient nephropathy) develops after 7-15years after diabetes onset. It is characterized by microalbuminuria (30-300 mg/day), normal or increased GFR.
IV. The stage of apparent diabetic nephropathy (overt nephropathy) occurs after 10-30 years of diabetes existance. It is characterized by proteinuria (more than 0,5 g of protein per day), arterial hypertension, decrease of GFR. These symptoms are caused by the sclerosis of 50-70% of glomerules.
V. The stage of chronic renal insufficiency (uremia) – End-Stage Renal Disease. In this case the GFR in less than 10 ml/min, renal changes indicates total glomerulosclerosis, that develops 20-40 years after diabetes onset.
First I-III stages are pre-clinical and reversible.

Prognosis
Controlling blood glucose, Hb A1c, lipids, blood pressure, and weight are important prognostic factors and predict the development of long-term macrovascular and microvascular complications. More than 60% of patients with type 1 DM fare reasonably well over the long term. Many of the rest will develop blindness, end-stage renal disease, and, in some cases, early death. If patient with Type 1 DM survives the period 10-20 years after onset of disease without fulminant complications, he or she has a high probability of reasonably good health. Other factors affecting long-term outcomes are the patient's education, awareness,

motivation, and intelligence level.

DIABETIC NEUROPATHIES

Diabetic neuropathies are the family of nerve disorders caused by diabetes. People with diabetes can, over time, have damage to nerves throughout the body. Neuropathy leads to numbness and sometimes pain and weakness in hands, arms, feet, and legs. Problems may also occur in every organ system, including the digestive tract, heart, and sex organs. People with diabetes can develop nerve problems at any time, but as longer person has diabetes, the risk is greater.

An estimated 50 % of those with diabetes have some form of neuropathy, but not all with neuropathy have symptoms. The highest rates of neuropathy are among people who have had the disease for at least 25 years.

Diabetic neuropathy also appears to be more common in people who have had problems controlling their blood glucose levels, in those with high level of blood fat and blood pressure, in overweight people, and in people over the age of 40. The most common type is peripheral neuropathy, also called distal symmetric neuropathy, which affects the arms and legs.

Causes

The causes are probably different for different varieties of diabetic neuropathy. Researchers are studying the effect of glucose on nerves to find out exactly how prolonged exposure to high glucose causes neuropathy. Nerve damage is likely due to a combination of factors:

- metabolic factors, such as high blood glucose, long duration of diabetes, possibly low level of insulin, and abnormal blood fat levels
- neurovascular factors, leading to damage of blood vessels that carry oxygen and nutrients to the nerves
- autoimmune factors that cause inflammation in nerves
- mechanical injury to nerves, such as carpal tunnel syndrome
- inherited traits that increase susceptibility to nerve disease
- lifestyle factors such as smoking or alcohol use

Symptoms

Symptoms depend on type of neuropathy and which nerves are affected. Some people have no symptoms at all. For others, numbness, tingling, or pain in the feet is often the first sign. Person can experience both pain and numbness. Often, symptoms are minor at first, and since most nerve damage occurs over several years, mild cases may go unnoticed for a long time. Symptoms may involve the sensory or motor nervous system, as well as the involuntary (autonomic) nervous system. In some people, mainly those with focal neuropathy, the onset of pain may be sudden and severe.

Symptoms may include

- numbness, tingling, or pain in the toes, feet, legs, hands, arms, and fingers

- wasting of the muscles of the feet or hands
- indigestion, nausea, or vomiting
- diarrhea or constipation
- dizziness or faintness due to a drop in postural blood pressure
- problems with urination
- erectile dysfunction (impotence) or vaginal dryness
- weakness

In addition, the following symptoms are not due to neuropathy but nevertheless often accompany it:

- weight loss
- depression

Types of Diabetic Neuropathy

Diabetic neuropathies can be classified as peripheral, autonomic, proximal, and focal. Each affects different parts of the body in different ways.

1. Peripheral neuropathy causes either pain or loss of feeling in the toes, feet, legs, hands, and arms
2. Autonomic neuropathy causes changes in digestion, bowel and bladder function, sexual response, and perspiration. It can also affect nerves that serve the heart and control blood pressure. Autonomic neuropathy can also cause hypoglycemia (low blood sugar) unawareness, a condition in which people no longer experience the warning signs of hypoglycemia.
3. Proximal neuropathy causes pain in thighs, hips, or buttocks and leads to weakness in legs.
4. Focal neuropathy results in sudden weakness of one nerve, or group of nerves, causing muscle weakness or pain. Any nerve in a body may be affected.

Neuropathy Affects Nerves Throughout the Body

Peripheral Neuropathy

- toes
- feet
- legs
- hands
- arms

This type of neuropathy damages nerves in the arms and legs. The feet and legs are likely tube affected before the hands and arms. Many people with diabetes have signs of neuropathy upon examination but have no symptoms at all. Symptoms of peripheral neuropathy may include

- numbness or insensitivity to pain or temperature
- a tingling, burning, or prickling sensation
- sharp pains or cramps
- extreme sensitivity to touch, even a light touch
- loss of balance and coordination

These symptoms are often worse at night.
Peripheral neuropathy may also cause muscle weakness and loss of reflexes, especially at ankle, leading to changes in gait (walking). Foot deformities, such as hammertoes and the collapse of the mid foot, may occur. Blisters and sores may appear on numb areas of the foot because pressure or injury goes unnoticed. If foot injuries are not treated promptly, the infection may spread to the bone, and the foot may then have to be amputated. Some experts estimate that half of all such amputations are preventable if minor problems are caught and treated in time.

Autonomic Neuropathy

- heart and blood vessels
- digestive system
- urinary tract
- sex organs
- sweat glands
- eyes

Autonomic neuropathy affects the nerves that control the heart, regulate blood pressure, and control blood glucose level. It also affects other internal organs, causing problems with digestion, respiratory function, urination, sexual response, and vision. In addition, the system that restores blood glucose level to normal after a hypoglycemic episode may be affected, resulting in loss of the warning signs of hypoglycemia such as sweating and palpitations.

Unawareness of Hypoglycemia

Normally, symptoms such as shakiness occur as blood glucose levels drops below 70 mg/dL. In people with autonomic neuropathy, symptoms may not occur, making hypoglycemia difficult to recognize. However, other problems can also cause hypoglycemia unawareness to this does not always indicate nerve damage.

Heart and Circulatory System

The heart and circulatory system are the part of the cardiovascular system, which controls blood circulation. Damage to the nerves in cardiovascular system interferes with the body's ability to adjust blood pressure and heart rate. As a result, blood pressure may drop sharply after sitting or standing, causing a person to feel light-headed—or even to faint. Damage to the nerves that control heart rate can mean that it stays high, instead of rising and falling in response to normal body functions and exercise.

Digestive System

Nerve damage to the digestive system most commonly causes constipation. Damage can also cause the stomach to empty too slowly, a condition called gastroparesis. Severe gastroparesis can lead to persistent nausea and vomiting, bloating, and loss of appetite. Gastroparesis can make blood glucose level fluctuate widely as well, due to abnormal food digestion.
Nerve damage to the esophagus may make swallowing difficult, while nerve damage to the bowels can cause constipation alternating with frequent, uncontrolled

diarrhea, especially at night. Problems with the digestive system may lead to weight loss.

Urinary Tract and Sex Organs

Autonomic neuropathy most often affects the organs that control urination and sexual function. Nerve damage can prevent the bladder from emptying completely, allowing bacteria to grow in the bladder and kidneys and causing urinary tract infections. When the nerves of the bladder are damaged, urinary incontinence may result because a person may not be able to sense when the bladder is full or control the muscles that release urine.

Neuropathy can also gradually decrease sexual response in men and women, although the sex drive is unchanged. A man may be unable to have erections or may reach sexual climax without jaculating normally. A woman may have difficulty with lubrication, arousal, or orgasm.

Sweat Glands

Autonomic neuropathy can affect the nerves that control sweating. When nerve damage prevents the sweat glands from working properly, the body cannot regulate its temperature properly. Nerve damage can also cause profuse sweating at night or while eating.

Eyes

Finally, autonomic neuropathy can affect the pupils of the eyes, making them less responsive to changes of light. As a result, a person may not be able to see well when the light is turned on in dark room or may have trouble driving at night.

Proximal Neuropathy

- thighs
- hips
- buttocks

Proximal neuropathy, sometimes called lumbosacral plexus neuropathy, femoral neuropathy, or diabetic amyotrophy, starts with pain in either the thighs, hips, buttocks, or legs, usually on one side of the body. This type of neuropathy is more common in those with type 2 diabetes and in older people. It causes weakness in legs, manifested by an inability to go from a sitting to standing position without help. Treatment for weakness or pain is usually needed. The length of the recovery period varies, depending on the type of nerve damage.

Focal Neuropathy

- eyes
- facial muscles
- ears
- pelvis and lower back
- thighs
- abdomen

Occasionally, diabetic neuropathy appears suddenly and affects specific nerves, most often in head, torso, or leg. Focal neuropathy may cause:

- inability to focus the eye

- double vision
- aching behind one eye
- paralysis on one side of the face (Bell's palsy)
- severe pain in the lower back or pelvis
- pain in the front of a thigh
- pain in the chest, stomach, or flank
- pain on the outside of the shin or inside the foot
- chest or abdominal pain that is sometimes mistaken for heart disease, heart attack, or appendicitis

Focal neuropathy is painful and unpredictable and occurs most often in older people. However, it tends to improve by itself over weeks or months and does not cause long-term damage. People with diabetes also tend to develop nerve compressions, also called entrapment syndromes. One of the most common is carpal tunnel syndrome, which causes numbness and tingling of the hand and sometimes muscle weakness or pain. Other nerves susceptible to entrapment may cause pain on the outside of the shin or the inside of the foot.

Diagnosis

Neuropathy is diagnosed on the basis of symptoms and a physical exam. During the exam doctor may check blood pressure and heart rate, muscle strength, reflexes, and sensitivity to position, vibration, temperature, or a light touch.

Doctor may also do other tests to help determine the type and extent of nerve damage.

1. A **comprehensive foot exam** assesses skin, circulation, and sensation. The test can be done during a routine office visit. To assess protective sensation or feeling in the foot, a nylon monofilament (similar to a bristle on a hairbrush) attached to a wand is used to touch the foot. Those who cannot sense pressure from the monofilament have lost protective sensation and are at risk for developing foot sores that may not heal properly. Other tests include checking reflexes and assessing vibration perception, which is more sensitive than touch pressure.
2. **Nerve conduction studies** check the transmission of electrical current through the nerve. With this test, an image of the nerve conducting an electrical signal is projected onto a screen. Nerve impulses that seem slower or weaker than usual indicate possible damage. This test allows the doctor to assess the condition of all the nerves in the arms and legs.
3. **Electromyography (EMG)** shows how well muscles respond to electrical signals transmitted by nearby nerves. The electrical activity of the muscle is displayed on a screen. A response that is slower or weaker than usual suggests damage of the nerve or muscle. This test is often done at the same time as nerve conduction studies.
4. **Quantitative sensory testing (QST)** uses the response to stimuli, such as pressure, vibration, and temperature, to check for neuropathy. QST is

increasingly used to recognize sensation loss and excessive irritability of nerves.

5. **A check of heart rate variability** shows how the heart responds to deep breathing and to changes in blood pressure and posture.
6. **Ultrasound** uses sound waves to produce an image of internal organs. An ultrasound of the bladder and other parts of the urinary tract, for example, can show how these organs preserve normal structure and whether the bladder empties completely after urination.
7. **Nerve or skin biopsy** involves removing a sample of nerve or skin tissue for examination by microscope. This test is most often used in research settings.

Treatment

The first step is to bring blood glucose level within the normal range to prevent further nerve damage. Blood glucose monitoring, meal planning, exercise, and oral drugs or insulin injections are needed to control blood glucose level. Although symptoms may get worse when blood glucose is first brought under control, over time, maintaining lower blood glucose level helps lessen neuropathic symptoms. Importantly, good blood glucose control may also help to prevent or delay the onset of further problems.

Additional treatment depends on the type of nerve problem and symptom, as described in the following sections.

Foot Care

People with neuropathy need to take special care of their feet. The nerves of the feet are the longest in the body and are the ones most often affected by neuropathy. Loss of sensation in the feet means that sores or injuries may not be noticed and may become ulcerated or infected. Circulation problems also increase the risk of foot ulcers.

More than half of all lower limb amputations in the United States occur in people with diabetes – 86,000 amputations per year. Doctors estimate that nearly half of the amputations caused by neuropathy and poor circulation could have been prevented by careful foot care. Here are the steps to follow:

- Clean your feet daily, using warm – not hot – water and a mild soap. Avoid soaking your feet. Dry them with a soft towel; dry carefully between your toes.
- Inspect your feet and toes every day for cuts, blisters, redness, swelling, calluses, or other problems. Use a mirror (laying a mirror on the floor works well) or get help from someone else if you cannot see the bottoms of your feet. Notify your health care provider of any problems.
- Moisturize your feet with lotion, but avoid getting it between your toes.
- After a bath or shower, file corns and calluses gently with a pumice stone.
- Each week or when needed, cut your toenails to the shape of your toes and file the edges with an emery board.

- Always wear shoes or slippers to protect your feet from injuries. Prevent skin irritation by wearing thick, soft, seamless socks.
- Wear shoes that fit well and allow your toes to move. Break in new shoes gradually by wearing them for only an hour at a time at first.
- Before putting your shoes on, look them over carefully and feel the insides with your hand to make sure they have no tears, sharp edges, or objects in them that might injure your feet.
- If you need help taking care of your feet, make an appointment to see a foot doctor, also called a podiatrist.

Pain Relief

To relieve pain, burning, tingling, or numbness, the doctor may suggest aspirin, acetaminophen, or nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen. (People with renal disease should use NSAIDs only under a doctor's supervision.) A topical cream called capsaicin is another option. Tricyclic antidepressant medications such as amitriptyline, imipramine, and nortriptyline, or anticonvulsant medications such as carbamazepine or gabapentin may relieve pain in some people. Codeine may be prescribed for a short time to relieve severe pain. Also, mexiletine, used to regulate heartbeat, has been effective in treating pain in several clinical trials.

Other pain treatments include transcutaneous electronic nerve stimulation (TENS), which uses small amounts of electricity to block pain signals, as well as hypnosis, relaxation training, biofeedback, and acupuncture. Walking regularly or using elastic stockings may also help leg pain.

Gastrointestinal Problems

To relieve mild symptoms of gastroparesis—indigestion, belching, nausea, or vomiting—doctors suggest eating small, frequent meals, avoiding fats, and eating less fiber. When symptoms are severe, the doctor may prescribe erythromycin to speed digestion, metoclopramide to speed digestion and help relieve nausea, or other drugs to help regulate digestion or reduce stomach acid secretion.

To relieve diarrhea or other bowel problems, the doctor may prescribe an antibiotic such as tetracycline, or other medications as appropriate.

Dizziness and Weakness

Sitting or standing slowly may help prevent the light-headedness, dizziness, or fainting associated with blood pressure and circulation problems. Raising the head of the bed or wearing elastic stockings may also help. Some people may benefit from increased salt in the diet and treatment with salt-retaining hormones. Others may benefit from high blood pressure medications. Physical therapy can help when muscle weakness or loss of coordination is a problem.

Urinary and Sexual Problems

To clear up urinary tract infection, the doctor will probably prescribe antibiotics. Drinking plenty of fluid will help to prevent another infection. People who have incontinence should try to urinate at regular intervals (every 3 hours, for example) since they may not be able to tell when their bladder is full.

To treat erectile dysfunction in men, doctor will first manage tests to rule out a hormonal cause. Several methods are available to treat erectile dysfunction caused by neuropathy, including taking oral drugs, using a mechanical vacuum device, or injecting a drug called a vasodilator into the penis before sex. The vacuum and vasodilator raise blood flow to the penis, making it easier to have and maintain an erection. Another option is to surgically implant an inflatable or semi rigid device into penis. A constriction ring or penile sling may be helpful. Vaginal lubricants may be useful for women when neuropathy causes vaginal dryness. To treat problems with arousal and orgasm, the doctor may refer the woman to a gynecologist.

DIABETIC RETINOPATHY

Diabetes is a disease that occurs when the pancreas does not secrete enough insulin or the body is unable to process it properly. Insulin is the hormone that regulates the level of sugar (glucose) in the blood. Diabetes can affect children and adults.
Patients with diabetes are more likely to develop eye problems such as cataracts and glaucoma, but the disease's affect on the retina is the main threat to vision. Most patients develop diabetic changes in the retina after approximately 20 years. The effect of diabetes on the eye is called diabetic retinopathy.
Over time, diabetes affects the circulatory system of the retina. The earliest phase of the disease is known as background diabetic retinopathy. In this phase, the arteries of the retina become weakened and leak, forming small, dot-like hemorrhages. These leaking vessels often lead to swelling or edema in the retina and decreased vision.
The next stage is known as proliferative diabetic retinopathy. In this stage, circulation problems cause areas of the retina to become oxygen-deprived or ischemic. New, fragile, vessels develop as the circulatory system attempts to maintain adequate oxygen levels within the retina. This is called neovascularization. Unfortunately, these delicate vessels hemorrhage easily. Blood may leak into the retina and vitreous, causing spots or floaters, along with decreased vision.
In the later phases of the disease, continued abnormal vessel growth and scar tissue may cause serious problems such as retinal detachment and glaucoma.

Signs and Symptoms

The affect of diabetic retinopathy on vision varies widely, depending on the stage of the disease. Some common symptoms of diabetic retinopathy are listed below, however, diabetes may cause other eye symptoms.

- Blurred vision (this is often linked to blood sugar levels
- Floaters and flashes
- Sudden loss of vision

Detection and Diagnosis

Diabetic patients require routine eye examinations so related eye problems can be detected and treated as early as possible. Most diabetic patients are frequently examined by an internist or endocrinologist who in turn work closely with the

ophthalmologist.
The diagnosis of diabetic retinopathy is made following a detailed examination of the retina with an ophthalmoscope. Most patients with diabetic retinopathy are referred to vitreo-retinal surgeons who specialize in treating this disease.

Treatment

Diabetic retinopathy is treated in many ways depending on the stage of the disease and the specific problem that requires attention. The retinal surgeon relies on several tests to monitor the progression of the disease and to make decisions for the appropriate treatment. These include: fluorescein angiography, retinal photography, and ultrasound imaging of the eye.
The abnormal growth of tiny blood vessels and the associated complication of bleeding is one of the most common problems treated by vitreo-retinal surgeons. Laser surgery called pan retinal photocoagulation (PRP) is usually the treatment of choice for this problem.
With PRP, the surgeon uses laser to destroy oxygen-deprived retinal tissue outside of the patient's central vision. While this creates blind spots in the peripheral vision, PRP prevents the continued growth of the fragile vessels and seals the leaking ones. The goal of the treatment is to arrest the progression of the disease.
Vitrectomy is another surgery commonly needed for diabetic patients who suffer a vitreous hemorrhage (bleeding in the gel-like substance that fills the center of the eye). During a vitrectomy, surgeon carefully removes blood and vitreous from the eye, and replaces it with clear salt solution (saline). At the same time, the surgeon may also gently cut strands of vitreous attached to the retina that create traction and could lead to retinal detachment or tears.
Patients with diabetes are at greater risk of developing retinal tears and detachment. Tears are often sealed with laser surgery. Retinal detachment requires surgical treatment to reattach the retina to the back of the eye. The prognosis for visual recovery is dependent on the severity of the detachment.

Expectations (prognosis)

Patients who have good control of their blood sugar and blood pressure may improve their outcomes.
Diabetic retinopathy can lead to blindness without treatment.

Complications

- Glaucoma
- Retinal detachment
- Blindness

Prevention

Researchers have found that diabetic patients who are able to maintain appropriate blood sugar level have fewer eye problems than those with poor control. Diet and exercise play important roles in the overall health of those with diabetes.

Diabetics can also greatly reduce the possibilities of eye complications by scheduling routine examinations with an ophthalmologist. Many problems can be treated with much greater success when caught early.

FOOT ULCERS

Classification of diabetic foot ulcers

Most experts use some variant of the classification system developed by Wagner and most currently modified by Brodsky.

Depth-Ischemia Classification of Diabetic Foot Lesions

0 – At-risk foot, no ulceration
Patient education, accommodative footwear, regular clinical examination
1 – Superficial ulceration, not infected
Offloading with total contact cast (TCC), walking brace or special footwear
2 – Deep ulceration exposing tendons or joints
Surgical debridement, wound care, offloading, culture-specific antibiotics
3 – Extensive ulceration or abscess
Debridement or partial amputation, offloading, culture-specific antibiotics

Ischemia

A – Not ischemic
B – Ischemia without gangrene
Noninvasive vascular testing, vascular consultation if symptomatic
C – Partial (forefoot) gangrene
Vascular consultation
D – Complete foot gangrene
Major extremity amputation, vascular consultation

In diabetes mellitus, severe hyperglycemia may result from absolute or relative insulin deficiency. In some patients, the condition may culminate in diabetic ketoacidosis or hyperglycemic hyperosmolar nonketotic coma. Profound hypoglycemia may result from a relative excess of insulin. Symptoms associated with acute hyperglycemia generally develop more slowly (over hours or days) than do symptoms associated with an acute fall in the level of blood glucose (over minutes).
Diabetes mellitus (DM) is a systemic disease that affects essentially every organ of the body. The fatal outcome is related to the development of acute or chronic complications.

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