OTHER DIABETIC COMPLICATIONS

THE primary complication of diabetes is atherosclerosis, or hardening of the arteries that leads to heart attack, stroke, and amputations. This is discussed elsewhere on this papers (see Goal Setting, Drugs for High Cholesterol, Blood Pressure Management, Coronary Artery Disease Screening). Other common complications seen with diabetes include those affecting the eyes, kidneys and nerves, discussed here.

Diabetes-related eye disease is the most common cause of blindness in the United States. The process that occurs in the eyes is similar to the process that occurs throughout the rest of the body. With poorly controlled diabetes, the elevated blood sugar levels over time damage capillary membrane supporting cells called pericytes and these cells begin to die. As a result, the membrane cells of capillaries begin to leak plasma fluid into the surrounding tissue spaces. Along with fluid, cholesterol, fats and other blood components penetrate tissues as well. Over time, deposits of cholesterol and fats obscure some of the transparency of the retina tissue and reduce vision. Capillaries damaged by loss of pericytes are also prone to rupture and bleeding. Tiny blot hemorrhages can occur as well as larger hemorrhages that pool in front of the retina and obscure vision. Occasionally a large bleed may occur, and a large amount of blood will pour into the retina, then seep out over the surface of the retina and into the vitreous, or central portion of the eye. Such a large bleed can cause partial or total loss of vision in one eye. The time it takes for any of these processes to heal is directly related to the amount of blood in the eye and the state of health of the eye and involved tissues. Large bleeds can be permanent.

Retinal specialists use laser therapy to coagulate areas of leaking blood vessels, to destroy damaged blood vessels or new abnormal blood vessels that grow over the retina. Laser therapy typically will not improve vision, but is very effective at stopping the ongoing damage that occurs with diabetic eye disease so as to prevent further deterioration of vision. Laser therapy is more effective when done early in the course of diabetic eye disease. After substantial bleeding has occurred in a diabetic eye, laser therapy cannot be used because the bleeding obscures the target areas over the retina. The ophthalmologist has to wait until the blood is reabsorbed before they can either see or treat the points of damage and bleeding.

We now also have new medications available that suppress the inappropriate release of growth factors in the back of damaged diabetic eyes. These drugs have been shown to slow the progression of diabetic eye disease and give ophthalmologists more time to address problem areas with laser treatments.

Prevention is much better than treatment of diabetic eye disease. The earlier treatment begins, the more vision is preserved. That is why good diabetic care mandates yearly eye exams by an ophthalmologist.

Diabetic kidney disease results from the same process that leads to diabetic eye disease. High blood sugar levels damage the basement membrane of the filtering apparatus (glomerulus) in the kidney, and over time this membrane becomes inappropriately leaky. The glomerulus of the kidney goes from a fine filter to more like a

sieve. Larger and larger molecules start to leak out of the blood stream into the kidney collecting tubules and the urine. Proteins become lost in the urine and tubular damage to the kidneys also occurs. Ultimately the kidneys fail to filter the blood adequately and this results in the manifestations of kidney failure.

The earliest indication that diabetic kidney damage is beginning to occur is the leaking of minute amounts of albumin into the urine (microalbuminuria). This albumin can be measured in the laboratory. It is not recognizable by the old urine dip sticks that we used to use to check urine sugar and proteins. Microalbuminuria is defined as the presence of more that 30 mg of albumin in the urine over a 24 hour period of time. When the urine microalbumin levels rise to 400-600 mg/24 hours it becomes recognizable on urine dipsticks and is known as macroalbuminuria. Somewhere about this time the damage occurring to the kidney becomes irreversible and a slow relentless decline in kidney function begins, leading to kidney failure in about 10 years. For the above reasons, we screen diabetic individuals annually for urine microalbumin.

High blood pressure will accelerate the damage that diabetes inflicts upon the kidneys. Aggressively controlling high blood pressure will markedly slow the progression of kidney damage occurring under these circumstances. Given that blood sodium, potassium, water and protein content are largely regulated by kidney function, these electrolytes and water balance issues may have to be closely monitored in patients with kidney insufficiency on medications for their diabetes and hypertension.

Diabetic nerve disease results from the same process that leads to diabetic eye and kidney disease. Nerves are living tissue that require a good blood supply for oxygen, nutrients, and to carry waste products away. Capillaries called vaso nevorum are the blood vessels responsible for this task. When these vessels become damaged or leaky, nerve cells start to suffer. High sugar levels may lead to the accumulation of slowly metabolized sugar alcohols in nerve cells that interfere with cellular function. Over time, the nerve cells with the longest fiber length (those supplying the feet and toes with sensation) tend to be the first nerves damaged. Nerve cell death leads to loss of sensation of hot/cold and light touch first in the distal toes, then at the bases of the toes, then at the mid foot, then at the heel, then the ankle, then the leg. This is called “stocking and glove” dysesthesia, because the sensation is lost in a fashion as if you were covering your foot with a stocking or your hand with a glove. This pattern of sensory damage is typical of diabetic nerve damage. Usually the feet, ankles and legs are affected first. Only after that may one see similar symptoms in the fingers and hands. We had thought that this damage was irreversible. However, there is some evidence that some nerve regeneration can occur, and permanent nerve damage may not necessarily be the case. Diabetic nerve damage is of concern because it is one of the primary factors responsible for diabetic foot damage.

For the above reasons, people with diabetes with peripheral neuropathy should be very careful to protect their feet from injury. Don’t use your toe or heel to sense the temperature of bath water. You could easily burn yourself without knowing it. Don’t walk around the house barefoot. If you step on a small pebble or a tack, or bump your foot

under the desk you may not know it and more damage may occur than would otherwise be the case. Wear shoes that are properly fitted to prevent blister formation, calluses or other foot injuries. Inspect your shoes before putting them on to make sure there are no foreign objects such as tacks or paper clips in them. If you have significant foot deformities such as bunions or hammer toes or big calluses, you probably should get diabetic shoes. They are shoes made wider and with deeper toe boxes that allow for more toe space and the placement of custom made shoe inserts which will better distribute weight across the foot and reduce the risk of foot injury.

Revised 3/2019