Blindness is possibly the handicap that is most feared by people. There have been many patients who have said to me: if I lose my vision I may as well die. I disagree with this sentiment of course, as I know blind people who live fuller lives than some sighted people. However, we cannot deny the fact that blindness impairs all aspects of life: self-care, ambulation, employment, leisure. Patients may be unable to identify the correct medication or give themselves insulin.

In developed countries, diabetic retinopathy is the second commonest cause of blindness, after age-related macular degeneration, which is the most common cause of blindness in working-age adults. Unlike age-related macular degeneration, however, diabetic blindness is largely preventable. According to the World Health Organisation (WHO), 150 million of people worldwide have diabetes. After 15 years, 2% go blind and 10% are visually handicapped, in Singapore, the National Health Survey in 2004 revealed that 8.2% of the adult population has diabetes, and therefore at risk of developing diabetic retinopathy.

Much of the visual problems can be prevented by control of the risk factors, early detection of retinopathy and appropriate treatment.

Risk Factors
Randomised controlled studies have shown that well-controlled blood sugar, hypertension, and hyperlipidaemia reduce the onset, as well as the progression of diabetic retinopathy.

The Diabetes Control & Complications Trial (DCCT, 1994) showed that intensive control of diabetes reduced the rate of progression of diabetic retinopathy from 49% to 17.1%.

The United Kingdom Prospective Diabetes Study (UKPDS, 1998) showed that intensive control of diabetes reduced the risk of diabetic retinopathy by 21% at 12 years; and that tight control of blood pressure (<150/85mm Hg) led to a 34% reduced risk of retinopathy progression and a 47% reduced risk for visual deterioration. The Action to Control Cardiovascular Risk in Diabetes Study (ACCORD, 2010), showed that intensive cholesterol therapy significantly reduced the rate of progression of diabetic retinopathy when compared with standard treatment (6.5% with fenofibrate and simvastatin versus 10.2% simvastatin alone).

Whilst patients do need to take some responsibility for their own health, the onus is on us as doctors to continue emphasising the necessity to have their eyes screened, particularly those who are poorly controlled systemically. For every patient that loses vision, we should be able to tell ourselves that we did our best to prevent it.
Development of Diabetic Retinopathy

Patients with insulin-dependent diabetes (IDDM) are more likely to develop retinopathy, and this is probably related to the greater difficulty in maintaining euglycaemia. On the other hand, patients with non-insulin-dependent diabetes (NIDDM) are more likely to either present with retinopathy at diagnosis of diabetes, or develop diabetic retinopathy soon after diagnosis of diabetes, as the onset of diabetes may have been years before actual diagnosis, giving plenty of time for the retinopathy to develop. Screening for diabetic retinopathy should thus begin at diagnosis in NIDDM and after 3-5 years in IDDM. Retinopathy can progress rapidly in diabetic pregnancies, and screening should be stepped up during this time.

Diabetic retinopathy is a microangiopathy, as is diabetic nephropathy and diabetic peripheral neuropathy. The pathophysiology of diabetic retinopathy is based on two events:

1. leakage and exudation of fluid, protein and lipid from the retinal capillaries, and
2. capillary closure, leading to retinal haemorrhages, ischaemia and subsequently, neovascularisation through the mediation of VEGF (vascular endothelial growth factor).

As the retinopathy evolves, it goes through a series of stages which form the basis of diabetic retinopathy classification. These have significance in the prognosis and indications for treatment.

There are essentially two types of diabetic retinopathy:

- nonproliferative diabetic retinopathy, which can be subdivided into mild, moderate or severe, based on the amount and nature of retinal haemorrhages, and signs of ischaemia, such as venous beading and intraretinal microvascular abnormalities, and
- proliferative diabetic retinopathy, where neovascularisation occurs. Neovascularisation is the growth of abnormal vessels, which can lead to vitreous haemorrhage and fibrovascular proliferation with traction retinal detachment. If neovascularisation of the iris (rubeosis) occurs, rubeotic glaucoma develops, with subsequent optic nerve damage and further loss of vision.

Proliferative diabetic retinopathy tends to progress more quickly and with graver consequences in younger patients, as they have a greater capacity to develop fibrovascular proliferation with attending traction retinal detachment.

Diabetic Maculopathy refers to nonproliferative changes occurring at the macula, in the form of retinal oedema, lipid and protein exudation or capillary nonperfusion. These changes, because they involve the macula, result in loss of vision (Fig 1).

Diabetic maculopathy tends to be a greater problem in older patients, whose microvasculature may be compromised by age, hypertension and hyperlipidaemia.

It is important to realise that vision is normal in both nonproliferative and proliferative diabetic retinopathy if the macula is not involved. Thus, it is possible to have fairly advanced diabetic retinopathy that is asymptomatic. This is the reason why screening for diabetic retinopathy is essential to reduce diabetic visual loss.

Fig 1. Nonproliferative diabetic retinopathy with clinically significant macular oedema (maculopathy)

Fig 2. Proliferative diabetic retinopathy with fibrovascular proliferation in the right eye (a) and pre-retinal/vitreous haemorrhage in the left eye (b). Despite advanced disease, vision is good because the macula is unaffected.
Management of Diabetic Retinopathy

The management of diabetic retinopathy encompasses a wide range of approaches. They include:

- Management of risk factors: obsessive control of blood sugar (HbA1c<6.5-7mg%), hypertension (BP<130/85) and hyperlipidaemia through lifestyle modification and medication.
- Regular screening for diabetic retinopathy
- Timely and appropriate treatment of diabetic retinopathy
- Education of patients and their caregivers so that they can understand and buy into the need for all the above measures, and be effective partners in the fight against their own diabetic visual loss. Education of health care professionals in the holistic management of diabetic patients also plays a significant role.

Screening is effective for diseases in which the target population is defined, which can be detected at the stage that treatment can be usefully instituted, where the screening process is not too invasive, and where there are few false positive and false negative results.

Diabetic retinopathy is thus an ideal disease to screen. Screening is done in polyclinics, GP practices, hospitals, and the Diabetic Society of Singapore. Studies to improve diabetic screening methods using technology to capture images and transmit them to a central reading centre are ongoing, in anticipation of an increasing load of diabetic patients.

The polyclinics, general practitioners and endocrinologists in Singapore have done a good job in having their diabetic patients screened for diabetic retinopathy. The management of systemic risk factors has improved over the years. Consequently, the impression of ophthalmologists (who have been managing diabetic retinopathy patients for many years) is that the proportion of patients presenting with advanced untreated diabetic retinopathy has fallen.

However, we do still see some patients presenting with advanced, untreated disease because they were never screened or because they defaulted on screening or follow-up sessions. We have all experienced patients who refuse to have their eyes screened or to control their systemic risk factors, against their doctors’ advice. Some of our patients fail to return for follow-up when they have mild nonproliferative retinopathy, only coming back years later when they have developed visual loss from irreversible diabetic maculopathy or proliferative retinopathy. Whilst patients do need to take some responsibility for their own health, the onus is on us as doctors to continue emphasising the necessity to have their eyes screened, particularly those who are poorly controlled systemically. For every patient that loses vision, we should be able to tell ourselves that we did our best to prevent it.

Treatment of Diabetic Retinopathy

For decades, the mainstay of diabetic retinopathy treatment has been laser photocoagulation for maculopathy and proliferative retinopathy, and microsurgery in the form of vitrectomy, sometimes requiring delicate excision of the fibrovascular membranes, in more advanced proliferative retinopathy.

We have now entered a new era where intravitreally injected drugs (eg, anti-VEGF such as ranibizumab) can be used to improve vision in diabetic macular oedema, or reduce bleeding during surgery. It is imperative for ophthalmologists today to keep abreast of the new treatment strategies, in order to provide the best chance of improving and maintaining vision for our patients.

Conclusion

The battle against diabetic retinopathy blindness is fought in many arenas. We all have a part to play in combating this devastating handicap, and will share the credit when we succeed in preventing blindness from diabetic retinopathy.