Severe diabetic retinopathy at presentation in a young man

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Summary: A 22 year old man presented with a severe ischaemic diabetic retinopathy and, in spite of photocoagulation therapy, was blind in one eye 16 months after diagnosis. Four similar cases of aggressive diabetic retinopathy are reviewed. There is increasing evidence that a small group of young men with insulin dependent diabetes (IDD) may develop severe diabetic retinopathy at, or soon after, diagnosis.

Introduction

Many factors have been suggested as associated with the development of diabetic retinopathy. Duration of diabetes\(^1\) is the best recognized, while mean clinic blood glucose concentrations are high, particularly in those with severe retinopathy.\(^1,2\) Other factors implicated in the development of diabetic retinopathy include male sex,\(^3\) cigarette smoking,\(^4,5\) hypertension,\(^3\) hyperlipidaemia\(^6\) and alcohol consumption.\(^7\) Patients with non-insulin dependent diabetes (NIDD) may have severe diabetic retinopathy at presentation of their disease\(^8\) possibly associated with prolonged, asymptomatic hyperglycaemia. Severe retinopathy is exceptionally rare, however, in young patients with insulin dependent diabetes (IDD).\(^9\)

We report here a young man who presented as an emergency with a severe pre-proliferative retinopathy and was found to be in mild diabetic ketoacidosis. Diabetic eye disease progressed rapidly and within 9 months of presentation he was registered as partially sighted. Similar cases of aggressive diabetic retinopathy in young men with IDD are reviewed.

Case report

A 22 year old unemployed storeman was admitted as an emergency with a 10-day history of a painful, red right eye, headache and photophobia. On direct questioning he gave a history of thirst, polydipsia and polyuria. He had smoked 30 cigarettes/day for many years but had a low alcohol intake. There was no family history of diabetes mellitus.

On examination he was thin, ketotic, with a blood pressure of 130/70 mmHg. An acute ischae-
months after early diabetic retinopathy changes. Figure 1 Ischaemic retinopathy with cotton wool spots, early new vessels and macular oedema in the left eye 3 months after presentation.

Discussion

The presenting feature of acute iritis in our patient was presumably related to the severity of the ocular ischaemia. Ischaemia has been previously identified as a cause of iritis in aortic arch syndrome and internal carotid artery stenosis. This ischaemic iritis is often followed by ruberosis, as in our case. Sixteen months after presentation our patient had total destruction of one eye, while extensive therapy had stabilized the other eye, which appeared to be maintaining useful vision. Our patient is unusual because of the presence of severe diabetic retinopathy at presentation of his disease.

Diabetic retinopathy is rarely seen within 5 years of diagnosis of IDD in young patients as the development of retinopathy is related to duration of disease. In children under 15 years of age who had been diabetic for less than 5 years, even minor diabetic retinopathy changes identified by fluorescein angiography were rarely observed. In a series of over 5,000 newly diagnosed diabetics only 1.5% of patients in the 20–39 year age group had retinopathy, and of these 9 young patients with retinopathy, 7 were controlled on diet or oral hypoglycaemic therapy and only 2 had IDD. None of the patients had evidence of an accelerated type of retinopathy, but of these 9 young patients, 8 were male.

Severe diabetic retinopathy at presentation of IDD in young patients has been described on two previous occasions while our patient is the fifth reported case of severe proliferative retinopathy presenting at, or shortly after, diagnosis in young patients. At least two, including our patient, were heavy cigarette smokers, one was hypertensive, one gave a history of excessive alcohol intake, and one had been involved in severe trauma prior to presentation of diabetes. All 5 patients have been young men and men are recognized as being at increased risk of developing proliferative diabetic retinopathy. Of the other factors implicated in the development of severe diabetic retinopathy, only heavy cigarette smoking was implicated in our case.

In two of the three cases of severe diabetic retinopathy in which details of follow-up are given, the retinopathy advanced rapidly in spite of photocoagulation therapy, as in our case. In the fourth case, yttrium implantation resulted in an improvement in the retinopathy, although photocoagulation was subsequently necessary. Diabetic eye disease may progress rapidly in such patients, and aggressive therapy should be considered at an early stage. Sudden improvement in diabetic control should, however, perhaps be avoided as dramatic improvement in glycaemic control might be associated with a deterioration in retinopathy.

Keen reviewed three trials in which continuous subcutaneous insulin infusion was used to control diabetes intensively in patients with retinopathy. Results pointed towards accelerated worsening of retinopathy in those intensively treated compared to those on normal insulin regimes. Our patient’s insulin dependent diabetes was well controlled quickly as were Cove’s second and Alexander’s patients and in each the retinopathy progressed rapidly within the first few months leading to blindness in two. This is in contrast to those reviewed by Keen where ‘little if any serious deterioration of vision accompanied normoglycaemic re-entry’.

There is increasing evidence that a small group of young men with diabetes are prone to develop severe diabetic retinopathy. Review of our case and similar cases suggests that accelerating factors in this group may be smoking, alcohol abuse, hypertension or previous severe trauma.
References


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