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Preventing diabetes in south Asians

Too little action and too late

The south Asian diaspora in the United Kingdom comprises Indians (predominantly Gujaratis and Punjabis), Sri Lankans, Pakistanis, and Bangladeshis. A dramatic increase in the prevalence of type 2 diabetes in south Asians is observed in many parts of the world including the United Kingdom.[1] While marked cultural and social differences arise within this racial group, south Asians have the unenviable distinction of achieving the highest death rates of coronary heart disease in the United Kingdom,[2] with Bangladeshis being particularly disadvantaged.[3] Much of this excess risk may be attributed to the increased risk of type 2 diabetes (four times that of Europeans), which develops about 10 years earlier than in Europeans,[4] and renal and cardiac complications are encountered more commonly.[5]

Although genetic factors are important, the increased incidence of type 2 diabetes is strongly associated with increasing central (intra-abdominal) obesity and hyperinsulinaemia.[6] South Asians seem to be more insulin resistant, even at an early age.[7] The relation between obesity and insulin resistance may occur at lower levels of fatness in south Asians compared with Europeans, and in recognition of this the World Health Organization has recommended lower levels of body mass index as desirable in south Asians (overweight is classed as a body mass index higher than 23). A more useful clinical measurement of central obesity is waist circumference, which is a marker of risk for both diabetes and cardiovascular disease. Recently, studies of Indian patients in India have shown much lower thresholds for waist circumference and increased risk of glucose in tolerance in south Asians compared with Europeans.[8]

Increasing fatness is becoming common among Westernised south Asians owing to cultural and environmental influences, in particular Western influences on the traditional (and healthier) south Asian diet, and to low levels of physical activity.[9] Great concern is caused by the increasing levels of obesity among south Asian children (Taylor S, 2003, personal communication). In the face of a rapid increase in the prevalence of diabetes among south Asians, what can be done to reduce their risk of developing the condition?

Firstly, the particular educational needs of south Asian communities need to be addressed. Knowledge of the risks of increasing fatness is poor among many south Asian communities, and it is clear that culturally appropriate interventions are required that involve a whole community.[10] An example of this is "Project Dil" in Leicester, which has undertaken focus groups to identify the south Asian communities' needs, engaged primary care to set up training programmes for patients and healthcare professionals, and developed peer educators to spread the message.[11] This has accessed large numbers of south Asian people, and its success has resulted in continued mainstream funding. It is clear, however, that far more research is required to provide an evidence base for health promotion in south Asians, with rigorous evaluation of culturally tailored community initiatives. In east London, where the problems of diabetes and coronary heart disease in south Asians are intense, we are attempting to develop and evaluate community based weight management programmes for our local Bangladeshi community.

Secondly, healthcare professionals need to understand better the higher risks of coronary heart disease and diabetes and lower thresholds for intervention required in south Asian people. Clinicians must be aware of the very high risk of developing diabetes in people with strong family histories of diabetes, obesity, or cardiovascular disease. The recently published "Diabetes Prevention Programme" offers some hope that intervention before the development of diabetes in high risk groups may prevent or delay the onset of diabetes.[12] This study undertook lifestyle intervention, treatment with metformin, or routine care in 3234 patients with impaired glucose tolerance. The lifestyle intervention was intense, with one to one monthly sessions with a case manager to help support behavioural change. Importantly, almost half of the participants were from high risk ethnic groups. Over five years, the risk of diabetes was reduced substantially (58%) in the lifestyle intervention group and 31% in the metformin group, with a modest average weight loss of 5.6 kg.

Thirdly, although prevention of diabetes should be of central concern, growing evidence is now available to enable us to intervene intensively to reduce cardiovascular risk in patients with diabetes. It should be recognised that south Asian patients with diabetes, by virtue of their higher risk for complications, should be subject to lower thresholds for intervention in high blood pressure, dyslipidaemias, or glycaemia compared with Europeans.

South Asian people are at high risk of developing type 2 diabetes. Local and national multi agency action is urgently required to raise the profile of diabetes, obesity, and related health issues affecting south Asians. It is disappointing that, while the first standard of the recently published national service framework

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for diabetes indicated that more needed to be done to prevent diabetes, the opportunity to tackle this challenge was not grasped in the delivery document of the framework. The cornerstone of any intervention should be improved education of the community in a healthy lifestyle and improved diet, increased physical activity, and thereby prevention of obesity. Effective programmes need to be established, tailored for each population group and rigorously evaluated. The current situation of "too little and (often) too late" cannot be allowed to continue. Only in this way will the epidemic of diabetes witnessed in south Asians be arrested and reversed.

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Aspirin for diabetic retinopathy

The evidence of a beneficial effect is from basic science, not clinical trials

The earliest clinically recognisable lesions in diabetic retinopathy are capillary occlusions, which can be shown on fluorescein angiograms, a standard investigation in patients with retinopathy. The response to capillary blockage is dilation of neighbouring ones, leading to breakdown of the blood-retina barrier and leakage. Later large vessels become affected, and the result is retinal ischaemia, which is responsible for the secretion of vasoactive cytokines, leading to formation of the sight threatening proliferative lesions.

Since capillary occlusion is a pivotal lesion, causes and cures for it have been sought for a long time. Aggregation of platelets is increased in diabetes, and this has been proposed as the underlying abnormality.[1] An early paper by Pope et al showed platelet thrombi in retinal capillaries of diabetic patients.[2] Furthermore, observational evidence showed that patients treated with acetyl salicylic acid had less retinopathy than expected in a general diabetic population.[3] More recently in a detailed postmortem study on nine eyes Boeri et al found large thrombi more often in retinas from diabetic patients than in control retinas and double the number of vascular segments immunostaining for platelet glycoprotein IIIa.[4] They suggested as a result of their findings that aspirin be considered as an intervention in early diabetic retinopathy. This work was supported by the finding of Kern and Engerman, who in diabetic dogs found that treatment with aspirin reduced the number of acellular capillaries and retinal haemorrhages after five years of diabetes.[5]

Important advances have occurred in the understanding of diabetic retinopathy in the past 10 years. Platelets by themselves are no longer thought to be of prime importance in blocking retinal capillaries; rather, changes in the endothelial cells and white cells are responsible for capillary occlusion, as shown in the review of their own extensive work by Schroder et al.[6] Adamis's group noted the underlying inflammatory component of early diabetic retinopathy, and in a series of elegant experiments in the diabetic rat model showed that high dose aspirin (2 mg/kg/day) reduced leucocyte adhesion in diabetic retinal capillaries, arterioles, and venules.[7] Aspirin also reduced expression of integrins on the surface of leucocytes and the adhesion molecules, ICAM-1, in the capillary wall. Further effects included reduction of nitric oxide synthetase (eNOS) levels and reduced production of the vasoactive cytokine, tumour necrosis factor α, found to be raised in diabetic retinopathy. Thus there seems to be ample reason for using of aspirin in diabetic retinopathy.

Only two large studies of aspirin for retinopathy have been reported. The joint French-UK aspirin and dipyridamole trial was a randomised controlled study of 475 patients followed for five years. Patients with early retinopathy were included (at least five microaneurysms on fluorescein angiograms or areas of nonperfusion in the macular area).[8] The dosage of aspirin used was 330 mg, three times daily, given alone or in combination with dipyrimadole (75 mg three times daily). After five years fewer new microaneurysms formed in patients taking aspirin (the reduction was 1-2 new microaneurysms per patient per year). The findings were statistically significant but were not thought to be clinically important, and it was thought that there was only slight beneficial effect and no con-

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