Environmental Infections and Pollutants as a Cause of India’s Diabetes Explosion

Sir,

It is incontrovertible that there has been a major increase in the frequency of diabetes mellitus, particularly Type 2 over the last 40 years, quite apart from the recent affliction of our teenage population by obesity comparable to the Diabesity phenomenon recognised by Francine Kaufmann in US children. At our own centre, when I was a medical student, my chief the late Prof K S Mathur documented a low frequency of diabetes mellitus of around 2.5% in the urban population and 0.5% in the rural segments of Agra Division in the 1960s. By contrast the urban frequency in several recent surveys have reported a frequency of 12-18% from several centres, including our own, and a frequency of around 2.5% in the rural areas. Varying figures (Mohan et al. 2004; PODIS study5 reported recently may be partly due to differing sampling criteria etc but all agree that the prevalence is rising! Most workers have blamed the rising prevalence in India to westernization and urbanization. We ourselves have been adopting lifestyle modification prevention measures both in APIDS (Agra Diabetes Preventive Intervention Study in the Offspring of Biparental Diabetics) as well as while targeting trigger groups such as school children and teachers, in the Project Mang school life style education study (with Prof Anoop Mishra at Delhi and Dr Rajiv Gupta at Jaipur).3

The rapid diabetes explosion in India has also been attributed to a genetic predisposition of the Indian population, as compared to Western populations, but since the Indian gene pool has not significantly altered over the last half century this has always struck me as prima facie unlikely. Feast and famine, thrifty gene hypotheses such as the Barker concept have blamed the juxtaposition of intrauterine malnutrition with post uterine exposure to high calories. However despite improvements in antenatal care the diabetes and metabolic syndrome surge does not appear to have been dented. Prof Yagnik4 has called attention to a faulty folic acid/B12 balance in maternal supplementation and also referred to the YY (Yudkin-Yagnik) comparison in body fat content and distribution between Indian and Western subjects. However the diabetes surge in India appears to predate antenatal maternal folate supplementation and it is unknown whether folate exposure can be incriminated in other populations with rising Diabetes, both type 1 and type 2.

It is therefore pertinent to explore environmental causes for the diabetes explosion. A rapid increase in the population along with the deterioration of the environment is a phenomenon observed in many parts of Asia. I may point out that in our own centre: S N Medical College, Agra acute pancreatitis was an infrequent entity in the sixties/while today every clinician at Agra encounters cases every week, both of ultrasound proved fulminant pancreatitis as well as minor episodes of abdominal pain with elevated amylase/lipase levels.

Calcific pancreatitis had long been a well described entity described in Kerala by Prof Gee Varghese and in Orissa by Prof B B Tripathi but it was rare in North India. In the 60s the only cases encountered at our hospital were in nursing staff hailing from Kerala! Today calcific pancreatitis is observed at Agra, Uttar Pradesh, North India in several individuals who have never ventured out of their home state, although it is still less common than acute and subacute noncalcific pancreatitis.

Is this higher frequency of pancreatitis related to the increasing frequency of diabetes? Although plausible this needs documentation by long term follow up. Most cases of diabetes do not have a clinical history suggestive of pancreatitis, but this does not rule out the possibility of antecedent subclinical pancreatic disease.

Further is this increasing frequency of pancreatitis related to a new pancreatic tropic virus damaging the pancreas or setting the stage and predisposing to bacterial pancreatitis? Or does this reflect increasing exposure to an old organism due to worsening of drinking water supply as the population pressure strains civic resources. There is a dearth of virological studies in the clinical cases of acute pancreatitis, but the usually observed polymorph leucocytosis and high fever observed in these suggests either bacterial invasion or fulminant tissue necrosis.

Pancreatoxicity can be due to increased infections with environmental degradation, but we must also consider the possibility of toxin exposure.

A recent article in the New Scientist6 reviews evidence suggesting Persistent Organic Pollutants(POP) as a cause of diabetes. These include dioxins, DDT and PCB. It is noteworthy that 12 most hazardous POPs are being phased out globally under the Stockholm convention (Aldrin, Dieldrin, DDT, Endrin, Mirex, Heptachlor, Toxaphene, Hexa chlorobenzene, PCB, Dioxins and Furans). A11 of these had been phased out or regulated in the USA during 1984 to 1990, but in countries such as India many are still marketed. Further the Indian body content of DDT is very high thanks to the malaria eradication campaigns. In addition the use of non food grade plastics and recycled plastics for food packaging including milk, tea and even infant feed bottles is very widespread.

The July 1976 chemical explosion at Seveso, Italy littered the surrounding 18 square kilometrews area with dioxins, followed by increasing frequency there of cancer, cardiovascular disease and diabetes.6

Duk-Hee Lee at the Kyungpook National University, Daegu, Korea observed that elevated Gamma Glutamyl Transferase GGT levels were correlated with Diabetes. This enzyme has a role in removing pollutants including POP from inside cells. Analysing over 2000 subjects from the US National Health and Nutrition Examination Survey (NHANES study) she found a strong correlation of diabetes with the combined level of six different POPs, regardless of obesity, finding a 38 fold higher risk. The risk of diabetes was extremely low in subjects with undetectable POP levels. However in the presence of high POP levels, obesity as measured by the BMI (Body Mass Index) increased the diabetes risk. This enhanced POP diabetes risk with obesity may explain the rising US diabetes frequency despite reduction in POP levels in America. Are POPs stored in adipocytes affecting adipocytokine release? Is the hazard from visceral/ectopic fat related to POP storage? All these facets merit exploration. Meanwhile this is still more reason to avoid accumulating body fat! Further in America while some POP exposure has declined the effect of unknown chemicals such as...
plastic bottle drinking water is still unknown. Newer POPs are being used such as brominated flame retardants!

In any association it is moot to examine which is cause and which is effect? Is the POP association with diabetes due to impaired POP handling in Diabetes? Brown, 2008 refers to a Journal of Toxicology, 2003, 66,211 study in which there is no relation between diabetes and POP elimination rate. Griffin et al at Cambridge have shown metabolic disturbances similar to diabetes after exposure to low level mixtures of POPs.

What are the Implications for the Clinician in India?

Safeguarding the environment may reduce or blunt the Diabetes explosion in India, both in relation to pancreatropic infections and in relation to Persistent Organic Pollutants. This involves on the one hand protected water and food supply and sanitation, and on the other eschewing the unnecessary use of chemicals and plastics. Biological pest control, organic pesticide free farm produce, and the use of time honoured materials such as glass and stainless steel rather than the ubiquitous plastics therefore need attention particularly in diabetes prone families. While waiting for universal good sanitation, individual protection measures such as boiled water, kitchen organic gardens and glass/steel utensils can be employed. Further virological studies in all recent onset pancreatitis may be useful in implicating possible causative viruses and even in developing a vaccine. Environmental infections and toxins need consideration in any preventive strategy apart from the life style improvement and maternal nutrition that are currently being advocated.

References


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