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H. pylori : Association with Megaloblastic Anaemia

Sir,

The article by Desai and Gupte¹ entitled “*Helicobacter pylori* link to pernicious anaemia” is very interesting and timely. Megaloblastic anaemia is a common problem both in outdoor as well as in indoor patients, where they are admitted for anaemia related complications. We have also noticed that the usual causes of nutritional or systemic disorders are absent in a great majority of these patients and at time it is extremely difficult to pinpoint a probable etiology. Gastrointestinal symptoms are usually neglected unless they point towards definitive malabsorption features. We report below a young patient with megaloblastic anaemia in whom a diagnostic work-up was done as the anaemia had developed over a few weeks without a definitive etiology both systemic as well as nutritional.

A 23 years old male presented with generalised weakness for 4 weeks, blurring of vision, nausea and vomiting for 15 days. There was no history of addiction, especially alcohol intake/prolonged drug intake such as antiepileptic drugs. He came from a good family background and nutritional intake was absolutely normal. On examination patient was conscious, oriented to time, place and person, BP was 114/70 mmHg, pulse rate 126/min regular, respiratory rate 20/min and he was severely pale. CVS – loud S1 and soft systolic murmur at apex was present. Respiratory system, abdominal and nervous system examination were normal. Investigations revealed Hb of 4.0 gm%, TLC 3400/mm³, DLC – P₇₀L₂₈M₁E₁, platelet count 134000/mm³, reticulocyte count 3.2%, PCV 11.9%, RBC 1.27x10⁶/mm³, MCV 104.7 fl, MCH 31.5 pg/cell and MCHC was 33.2 g/l. Peripheral smear showed anisocytosis, microcytosis, macrocytosis, poikilocytosis and neutrophil hypersegmentation. Kidney function test, serum electrolytes, blood sugar and urine examination were normal. In liver function tests serum bilirubin (T) was 1.6 mg/dl (direct 0.03 mg/dl), SGPT

23 U/l, SGOT 64 U/l, alkaline phosphatase 36 KAU/l and total protein was 6 gm/dl (albumin 4 gm/dl). X-ray chest PA view and ultrasound abdomen were within normal limits. Stool for occult blood was positive. Iron study was normal and serum vitamin B12 level was 146 (normal 200-800 pg/ml). Fundus examination showed anemic retinopathy. Upper GI endoscopy revealed gastric erosions and rapid urease test was positive for *H. pylori*. Gastric and duodenal biopsies were taken to rule out atrophic gastritis and sprue. Report revealed chronic gastritis with activity and Giemsa stain was positive for *H. pylori*. Duodenal biopsy was suggestive of non-specific inflammation. Anti-gastric parietal cell antibodies was 49.0 U/ml (normal <10 U/ml). IFA could not be done due to cost factor. Patient was put on *H. pylori* kit (two Cap. Omeprazole 20 mg, two Tab. Amoxicillin 750 mg and two Tab. Tinidazole 500 mg) 1 OD for 10 days and inj. Vitcofol once a week for 4 weeks. Patient showed dramatic response to the treatment.

Our case illustrates that GI symptoms should be sought carefully and minutely in all patients with megaloblastic anaemia especially when profile indicates absence of usual causes. Upper GI endoscopy combined with investigations for *H. pylori* both in serum and biopsy become mandatory as indicated by these authors¹ as well as Kaptan *et al*,² who suggested *H. pylori* as novel causative agent in vitamin B12 deficiency. Similar observations have also been reported elsewhere.³ We agree with the view that the work-up as suggested in this article will establish diagnosis of pernicious anaemia / atrophic gastritis in a cost effective manner.

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Renal Consequences of Metabolic Syndrome

Sir,

Obesity has become a major public health problem worldwide, as a result of abundant food, sedentary lifestyle and Thrifty gene phenotype. The study by Gupta *et al* in a large group of randomly selected adults has shown a continuous positive relationship of all markers of obesity (body-mass index, waist size and waist hip ratio) with major coronary risk factors-hypertension, diabetes and metabolic syndrome.¹ Recent studies have shown that Metabolic Syndromes

(MetS) has harmful effects on the kidney also. Chagnac has demonstrated the renal pathological changes in obese Zucker rats.² These rats develop hyper-filtration, proteinuria, glomerulomegaly and focal segmental glomerulosclerosis. Studies in obese humans have confirmed these findings.^{3,4} Higher the body mass index, more the prevalence of end-stage renal disease after adjustment of blood pressure and presence of diabetes.⁵ In another study of 10,000 non-diabetic USA subjects with a normal baseline glomerular filtration rate, who were followed for nine years, the adjusted risk of developing chronic kidney disease (CKD) was 43% higher in subjects with the MetS.⁶ The pathophysiological factors contributing to renal disease include insulin resistance, adipocytokines, endothelial resistance, renin-angiotensin-aldosterone-system activation and oxidative stress.

The problem often starts in childhood. The popular myth 'A chubby child is a healthy child' is proving to be a bane for public health. Majority of obese children grow up to become obese adults. They are exposed to the metabolic consequences of obesity for a far more prolonged period, and hence are more likely to develop the renal complications. Mehta *et al* in a study of affluent Delhi schoolgirls has shown that the prevalence of obesity and overweight is 5.3% and 15.2% respectively.⁷ More than half of these obese children have central obesity. It is likely that in the coming years they may pose a huge burden on the already fragile renal health delivery system including dialysis facilities.

The main utility of the concept of MetS is that it is a powerful public education tool. The cluster of clinical

features, its complications and treatment, which essentially consists of life-style changes, can be easily explained and impressed upon the minds of the lay public. Hence it should become a part of the school health and other public education programmes, so that the epidemic of MetS can be nipped in the bud and its serious consequences in adulthood averted. So far there are no Indian studies on the renal consequences of MetS. We hope that future studies in India will be more comprehensive and also include the renal effects of MetS.

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