Incidence of Brucellosis in Arthritis and Chronic Low Back Pain in High Risk Group

Sir,

Lumbago is one of the most common causes of disability in patients under 45 years of age. Low back pain is exceedingly common, experienced at some time by up to 80% of the population.1

Brucella spondylitis is reported in 6-58% cases of brucellosis in various series. The spine is involved in acute as well as in chronic stage of the disease.2 The disease exists worldwide, especially in the Mediterranean basin, the Middle East, India and Central and South America where it is an important public health problem. World Health Organization figures put the number of new cases of brucellosis of more than 500000 per year. Cases of brucellosis have been reported in Bikaner division from time to time and recently there has been an epidemic of pyrexia with polyarthritis due to brucellosis.3

A total of 100 cases age ranging from 11-48 years, males 58, female 42, belonging to high risk group for brucellosis who presented with the complaints of low backache and/or arthritis in medical and orthopedics OPD were thoroughly studied. Brucellosis was established in 8 cases depending upon positive Rose Bengal plate agglutination test and brucella antibody titer more than 1:320.4 All the positive cases had mixed Brucella abortus and Brucella melitensis infection.

Age of positive cases ranged from 16 years to 40 years, all were males and belonged to poor socioeconomical status. Most of the positive cases were either shepherds (4 cases) or farmers (3 cases) and most frequently encountered animal species in our study was goat. Six cases were having habit of taking raw milk while one had habit of raw meat ingestion and one patient had direct contact with animals while working in a veterinary hospital.

The most common presenting symptoms were low back pain, (7 cases), joint pain (6 cases), fever and joint swelling (5 cases) and other constitutional symptoms like sweating, anorexia and weight loss were present in four cases. Physical examination revealed hepatomegaly (6 cases), splenomegaly (one case), peripheral arthritis (5 cases-polyarthritis 4 cases and monoarthritis one case), spinal tenderness (4 cases, most common site-L4). Other common signs detected were pallor in four cases, lymphadenopathy and paraspinous muscle spasm in two cases each.

Laboratory investigation showed mean Hb 9.43 gm% (ranging between 8.5-10.7 gm %), thus all patients were having anemia. The total leucocyte count was within normal limits in almost all the cases except in one case where we found leucocytosis with lymphocytosis. ESR was found to be ranging between 20-110 mm in first hour with mean value of 53.12, CRP was elevated in three cases out of eight. Two patients of brucellosis had detectable abnormality on X-ray of involved area of spine. In one patient vertebral body sclerosis was seen and in other one vertebral body destruction was seen.

All the patients were given treatment in the form of rifampicin 600-900 mg orally once daily and doxycycline 100 mg orally twice daily for 6 weeks and response to treatment was judged clinically by a drop in temperature, improvement in general condition resolution of arthritis, osteoarticular pain and backache. All patients responded excellent to the treatment with relief of symptoms like joint pain, joint swelling, fever etc. within seven days of the treatment.

Thus, our study concluded that patients presenting as either arthritis and/or low back pain with significant risk factors for brucellosis which entails a close direct/indirect contact with cattle, sheep, goat etc. or with a history of ingestion of raw milk or milk products or meat of these animals, the diagnosis of brucellosis should be kept in mind always, as brucellosis is a curable cause of arthritis and low back pain. For prevention of this disease in high-risk group, people should be educated not to drink raw milk, wear protective clothing/gloves and concerned authorities should carry out regular animal vaccination. As we found significant incidence of brucellosis in such cases there seems to be a need of national control programme in conjunction with the medical and veterinary department to prevent spread of the disease.

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References
Plea for a Color Code for Oral Anticoagulants

The number of patients who are being prescribed oral anticoagulants on an outpatient basis has shown a steady increase all over the globe. In India too prescriptions for oral anticoagulants have risen sharply especially over the last decade along with better laboratory control facilities. The elderly with ischemic cardiovascular accident or myocardial infarction are being given the benefit of anticoagulation and presently constitute a significant number of patients on anticoagulation.

Although the oral anticoagulants have proved their clinical usefulness in the past, they are drugs with a narrow therapeutic index. Therefore meticulous dose adjustments coupled with regular checks on the prothrombin time (PT) are essential to maintain safe levels of anticoagulation. With patients being prescribed alternate strength dosage regimens more frequently or even more complicated ones where the dose is skipped for two days or more at times, management of patients can be a challenging task. Oral anticoagulant use in India needs careful assessment regarding dosage patterns among other vital factors to ensure the desired safety of their use.

Warfarin and nicoumalone (Acitrom) which are the oral anticoagulants available in the Indian market are not color coded. Strengths of 1 mg and 5 mg of warfarin; and 1, 2 and 4 mg of nicoumalone are available in white color only. Although this might sound innocuous at the outset, its implications can be serious enough to warrant a second look.

In our country many patients are unaware of the strength of the dosage that they are prescribed and instead take the drug according to the tablet only i.e one, one and half etc. Most chemists do not have a ready stock of all strengths available with them and instead offer higher or lower strengths with the tablet to be taken in half or two tablets in place of one of the higher strength.

Lack of a color code and alternating strength regimens in a country where literacy levels are low provide a heady mix for wrong dosage consumption in patients leading to suboptimal or overtherapeutic international normalized ratios (INRs) and hence inappropriate management of the thrombotic states.

Physicians all over the country should unite and press on the pharmaceutical concerns involved in the manufacture of oral anticoagulant drugs to introduce a nationally acceptable color code for anticoagulants of different strengths to ensure that wrong dosages are not inadvertently not taken by patients.

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Prevalence Figures of Gastrointestinal Disorders in the Elderly — An Under or Overestimation?

Prevalence figures in the elderly for various gastrointestinal disorders are not available from India. A definitive diagnosis of the underlying disease cannot be made in most instances, partly due to large dropouts during follow-up due to old age itself, an impediment on revisits secondary to decreased mobility and above all procedural delays in investigative procedures. All these can influence the prevalence rates of old age disease in epidemiological surveys.

A retrospective study was undertaken from hospital records in patients above the age of 60 to determine the influence on prevalence figures of gastrointestinal disorders when there was either there was a loss to follow-up after registration or failure to confirm the diagnosis.

There were 3559 patients above the age of 60 yrs who had attended the GI unit over an eight year period. Most of the patients were between 60 and 69 years (73.7%); 22.7% of patients were between 70 and 79 years and 3.5% were above the age of 80. The male: female ratio remained constant in a ratio of 2:1 in all the three decades. A definitive diagnosis was possible in 50%. Failure to confirm the diagnosis due to loss of follow-up was 67% in patients with symptoms referable to gallbladder (e.g. gallbladder colic, cholecystitis), 44% in those with suspected liver disease (e.g. malignancy liver, hepatomegaly with ascites, hepatitis) and 39% those with dyspepsia with or without alarm symptom. These patients did not report for endoscopy and / or ultrasound despite fixation for these procedures within a mean period of 5.5 days of registration. The proportion men and women who were lost to follow-up remained the same as at entry of the study.

Amongst the patients who did come for follow-up, diagnosis was not possible despite in-depth evaluation in 78% of patients with small bowel disease (e.g. those with small bowel diarrhea, intestinal colic and anemia of unknown origin) and in 68% of patients with suspected intra-abdominal malignancy (e.g. anorexia, weight loss, abdominal masses, unexplained ascites). This was partly related to relative contraindication to procedures like colonoscopy, angiogram or laparoscopy secondary to cardiorespiratory embarrassment in a significant proportion of patients (67%). These patients were followed up with only a provisional diagnosis until last follow-up. This resulted in an over-estimate of prevalence figures from the case records.

To prevent both an under or probable diagnosis of gastrointestinal disorders in the elderly, there is an impending need for improvising quick and simple investigative procedures for confirming the clinical diagnosis e.g. by admitting the patient and completing the basic laboratory investigations, ultrasonogram and endoscopy within 24 to 48 hrs of registration. This would improve the quality of care to the elderly who are handicapped for frequent revisits due
to restrictive mobility and dependency on other family members.

The cost-effective health care systems that are designed to achieve the above stated end will naturally benefit not only the quality of epidemiological data from the hospitals, but more importantly improve the patient care. A prospective study is necessary to confirm this proposition not only in the elderly but also in the young.

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Venlafaxine Induced Hepatitis

Sir,

Venlafaxine, a derivative of Phenethylamine is a second generation- antidepressant and a non-selective inhibitor of the reuptake of serotonin, norepinephrine and dopamine and is used in the treatment of major depressive disorder. It is generally well tolerated. The common side effects include nausea, somnolence, dry mouth, dizziness, nervousness and impotence. It also has a potential adverse effect of inducing hypertention in some patients when given in doses excess of 300 mg per day.

We are reporting a case of a 30 year old healthy man, with no previous significant medical or psychiatric history or history of alcohol or substance abuse was diagnosed to have depressive episode, moderate type with somatic symptoms according to ICD-10 Diagnostic Criteria. He was started on Venlafaxine 37.5mg by a psychiatrist. Over a period of 4 months, Venlafaxine was gradually increased to 150 mg per day and patient continued to take the same medication for about 6 months, with a mild improvement in the depressive symptoms. Patient reported to us with a 15 days history of generalized weakness, nausea and vomiting. Physical examination was normal except for tenderness over the right hypochondriacal region. Laboratory investigation revealed deranged liver function tests ( Total bilirubin=1.1(0.1-1.0 mg/dl), conjugated bilirubin=0.5 (0-0.04mg/dl), aspartate aminotransferase = 167 (N= upto 37U/L), alanine aminotransferase = 81 (N=Upto 65U/L) and gamma glutamyl transferase level = 677 (N = 11-50U/L)). To exclude other causes of hepatotoxicity, investigations including hepatitis B and C viral markers, HIV and abdominal ultrasound were done and found to be normal. Venlafaxine was gradually tapered and stopped and patient was started on an antidepressant belonging to a different chemical class (Dothiepen). A week later patient improved clinically and repeat liver function test showed a significant improvement. (Gamma glutamyl transferase level decreased to 148 (N = 11-50U/L)).

We believe that there was a causal association between Venlafaxine and hepatitis because other causes of hepatitis were ruled out and discontinuation of Venlafaxine therapy led to the clinical recovery with improvement of liver function tests. There are few case reports of Venlafaxine induced hepatitis. Horsmans et al reported a case where a patient developed acute hepatitis after Venlafaxine administration. Liver biopsy done in the patient, revealed well-demarcated zone 3 confluent necrosis, inflammation and clumps of perivenular Kuffer cells containing lipid-rich ceroid pigment; however the portal tracts were not affected.1 After excluding other causes of hepatotoxicity, they reported a causal association between Venlafaxine and hepatitis, because the histologic findings supported drug-related hepatotoxicity. The liver function tests became normal after gradually withdrawing Venlafaxine. Other authors have also reported cases who presented with a similar history of Venlafaxine-induced hepatitis and with prominent cholestasis, who improved on withdrawing Venlafaxine. 2,3 However, the exact mechanism of Venlafaxine-induced hepatitis is not known. We suggest that liver function tests should be done regularly in patients receiving Venlafaxine and clinicians should be aware of the possibility of hepatitis in patients presenting with gastrointestinal symptoms.

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REFERENCE

Migration Stress and Premature Coronary Artery Disease : An Illustrative Pedigree

Prevalence of coronary artery disease (CAD) has been found to be higher among immigrant Indian population compared to natives in several countries. Stress of migration, change of lifestyle and heredofamilial factors have been blamed in the etiopathogenesis of premature CAD in this group of people.1

We recently came across a family in which several members had undergone the stress of migration to two different countries (India ↔ Mauritius ↔ UK) and developed premature vascular disease.

A 67 years lady attended the cardiology OPD for dyspnoea and retrosternal distress for last six months. She was detected to be hypertensive 16 years back, six months after the death of her husband who had died of an acute coronary episode. Later on she developed left hemiplegia at the age of sixty
She started having stable angina pectoris for the last six months which responded partially to beta blockers, aspirin and nitrates. She was a chronic tobacco chewer for last 35 years and continued to take tobacco till date in spite of repeated medical advice to leave it. Her roots were from Bihar (India). She had migrated to Mauritius after her marriage about 50 years back (Pedigree - 1).

Detailed family history of the index case (Fig. 1) revealed that her father had suffered stroke at the age of 50 years. Nearly all her brothers and sisters were hypertensive and diabetic; two other sisters also had CAD and one of them had undergone coronary artery bypass. She had three sons and a daughter. The eldest son developed hypertension and CAD at age 44 years. Second son is obese (BMI - 30) and detected to be hypertensive at age thirty two. Her youngest son became hypertensive at 40 years and had sudden cardiac death at age forty two.

Chest X-ray of the index case revealed cardiomegaly which was confirmed by echocardiographic evidence of concentric LVH. ECG showed ST-T changes consistent with ischemia.

Her only daughter married to an India, became widow at age 30, had migrated to UK after the death of her husband. Though asymptomatic she had low HDL cholesterol (27 mg/dl) and central obesity (WHR = 1.0). The fourth generation adolescent boy aged 19 years who had migrated to UK from India following the death of his father had a BMI of 24.0 and central obesity (WHR = 0.92). His lipid profile exhibited cholesterol 200 mg/dl, HDL 30 mg/dl, LDL 110 mg/dl and triglyceride 100 mg/dl. Lp (a) was normal (10 mg/dl).

Detailed family history of husband of the index case revealed that his father was a diabetic and developed sudden cardiac death at age 58 years (pedigree 2) (Fig. 2). He had six brothers who were chronic smokers and obese. All except one developed acute coronary syndrome prompting aggressive revascularisation.

Asian Indian immigrants have been singular exceptions in having CAD rates higher than those of dominant culture. Major conventional risk factors such as high levels of serum cholesterol or LDL cholesterol, hypertension and smoking alone fail to explain the excess risk of CAD in this ethnic group.

Though the traditional risk factors such as smoking, hypertension, diabetes mellitus, obesity, apple-shaped body and dyslipidaemia play an important role in increased incidence and prevalence of CAD but newer risk factors like lipoprotein (a) [Lp(a)], insulin resistance syndrome, hypertriglyceridemia, high small dense LDL, raised apolipoprotein B, apolipoprotein A-1, thrombogenic factors and hyperhomocysteinemia are becoming more and more important. The excess of CAD rates in immigrant Indians to Western countries is likely to be due to a combination of intrinsic susceptibility (nature), environmental factors (nurture) and stress of migration. The two pedigrees discussed above illustrate the complex interplay of genetic predisposition, environmental factors and stress of migration resulting in premature vascular disease in second and third generation in pedigree one.

Migration to a different country leads to several problems relating to communication, psychosocial and cultural factors. Quantitative research on health attitudes and lifestyle of Asian Indians living in Britain indicates that there is some dissatisfaction among these people. Many such Asians display type A personality with relatively high levels of ambitiousness, aggressiveness, impatience and competitiveness. Stress may manifest as depression or anxiety and probable high risk of CAD.

Thus excess of CAD in immigrant Indians is likely to be due to a combination of genetic and environmental factors. Genetic predisposition combined with environmental factors as a result of urbanisation, immigration and acculturation have a multiplicative effect leading to metabolic abnormalities suited for the development of premature CAD.

Fig. 1 : Showing details of pedigree of index case.

Fig. 2 : Showing details of husband’s pedigree of index case.
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Ghost of Metabolic Syndrome

Sir,

I read with great interest the article on Metabolic syndrome X.1 I have coined an acronym for Metabolic syndrome X which consists of its components - Ghost on the lines of CHAOS in previous descriptions of the disease.

G - Glucose intolerance
H - HDL - ↓
O - Obesity
S - Systematic hypertension
T - TGL - ↑

The acronym aptly describes its variable description and its impact on global health burden.

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Eales' Disease with Neurological Manifestations

Sir,

I read the case report by Kumaravelu S et al, entitled “Eales’ Disease with Neurological Manifestations” with great interest. Neurological involvement in Eales’ disease is rare and stroke-like presentation is further rarity. Infact, I had reported a case “Paraplegia in Eales’ Disease” in 1990.1 This was a young man of 25 years with acute onset, asymmetric paraplegia alongwith sensory and double sphincteric affection, who presented one year after the first ocular episode. Cord swelling cephalad to D4 was seen on myelography possibly due to cord edema. CSF revealed 10 RBC/mm³ with elevated proteins (457.5 mg%). Facility for CSF oligoclonal bands was not available. Work up for tuberculosis and collagen vascular disorders was negative. Partial but transient improvement was seen with steroids. This was the first case report of Eales’ Disease where MR imaging was performed. MRI scan of spinal cord and brain performed after two weeks of steroid therapy did not reveal any abnormality, as the cord edema resolved.

In the cases reported by Kumaravelu S et al, the imaging studies fail to provide explanation for left sided cerebellar signs and drowsiness, unless associated metabolic abnormality accounts for drowsiness. I wonder, whether the ring lesion seen on MRI was in fact a granuloma. Moreover, Kumaravelu S et al, have not evaluated the case for HIV and brucellosis. There is no mention of CSF proteins also.

The retinal periphlebitis, age and mode of onset, waxing and waning course, and sometimes multiple sites of lesions raise the suspicion of multiple sclerosis in such cases. However, in multiple sclerosis, optic atrophy is frequent, band of periphlebitis is very thin and neovascularization, retinal haemorrhages and retinitis proliferans are seldom seen.2 The neurologic disability is more severe with slight improvement in very first episode of Eales’ disease unlike remitting-relapsing course of multiple sclerosis. Further, it is rare to get a pleocytosis more than 50 cells/mm³ and proteins higher than 100 mg/100 ml in CSF in multiple sclerosis while they are elevated to a greater extent during acute phase of Eales’ disease. To conclude, neurological involvement is rare but well known in Eales’ disease. However, what is more important is the differentiation from multiple sclerosis as the prognosis differs markedly in two disorders.

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Reply from the Authors

Sir,

In our patient, the absence of cerebellar lesions was noted. However cerebellar dysfunction with ipsilateral pyramidal dysfunction can be seen with lesions right from contralateral frontal lobe to pons. Our patient had bilateral basal ganglia infarction with internal capsular involvement. This can explain the cerebellar dysfunction.

The ring enhancing lesion could be a granuloma of any etiology. He was negative for HIV serology. He was not evaluated for brucellosis. His CSF protein was 90 mg/dl.

We did not consider multiple sclerosis in our patient, as the clinical profile is not suggestive of the same.

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