Making India a Rabies-Free Country

Sir,

India has the highest incidence of rabies world-wide. About 35,000 people die annually in our country and this is when the first successful vaccine was discovered on July 6, 1885 by Louis Pasteur. About one million people receive post-exposure vaccination. Upto 50% receive the sheep brain vaccine that is less effective, requires more doses and carried the dreaded risk of neuroparalytic complications despite WHO recommendation for its substitution in 1987. Rabies immunoglobulin remains beyond the economic reach of most patients. Newspapers carry stories of deaths due to rabies and of valiant efforts by animal activists. Rabies is not a notifiable disease and our surveillance mechanisms are poor.

A study carried out in four cities of India has reported the annual incidence of animal bites to be 2.1 per 1000 population. The number of animal bites in our capital city of Delhi had increased from 23852 in 1995 to 29905 in 1998 of which over 95% were dog bites. Children constitute 30 to 50% of those receiving post-exposure vaccination or from dying from the disease. Facilities for laboratory confirmation by fluorescent antibody test, production of better vaccines and for proper management of hydrophobia are highly limited. About 99% of admitted hydrophobia cases do not observe the elementary concept of wound washing. The knowledge among health care providers on this subject is also poor.

The nuisance of stray dogs seems to have assumed epidemic proportions everywhere. Municipal bodies across India avoid catching these stray dogs due to animal rights activists, for instance the number of stray dogs culled in Delhi had decreased from 50,000 to 2500 a year. This impasse has to be resolved. I fail to understand why the society continues to tolerate the threat of stray and unvaccinated dogs, which are the major reservoir of this disease in India? This also holds true as regards the universal inaccessibility of modern vaccines and immunoglobulin at the least.

The recent judgements have clearly upheld the rights of common citizens to appropriate health care through government health care facilities and decent quality of life. I wonder what would be the government’s plea if a future animal bite patient were to file public interest litigation upon denial of a modern vaccine and immunoglobulin as infringement of the right to life. This could also apply to doctors treating such patients. Also I would opine that any citizen should be free to go about his work on the roads without the perennial fear of the right to life. Can we still continue to be silent spectators to these preventable deaths when means of reservoir control and effective vaccines exist? I hope that doctors, executives, policy makers and judiciary take a note of this and work concertedly to make India a rabies-free country.

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Posterior Circulation Stroke Following Manipulation of Neck by a Barber

Sir,

Neck manipulation as a therapeutic strategy for head and neck pain is common and is usually perceived as benign and risk-free. However, it can lead to serious neurological complications. We report a case of posterior circulation stroke, developing in a young male, following manipulation of neck by a barber.

A 37 years old right handed, non-smoker, non-diabetic, non-hypertensive man developed sudden, sharp, severe, shooting pain from nape of neck to the shoulder region on left side following head massage and neck manipulation by his barber, during which the patient’s neck was suddenly jerked from left to right and then right to left by keeping one hand on forehead and other on the chin on opposite side. Pain was accompanied by vertigo and 1-2 episodes of vomiting, following which the patient fell unconscious and was admitted to a local hospital. He regained consciousness after 15 hours, and was noticed to have slurring of speech with inability to sit and stand without support. Neurological examination, ten days later revealed cerebellar dysarthria, bilateral gaze evoked nystagmus, broad-based gait and bilateral cerebellar signs (left more than right). Plantars were bilateral flexor. Rest of the motor and sensory system examination, general and systemic examination including fundus oculi were normal. There was no bruit over the carotids or vertebral artery.

Hematological, biochemical, radiological investigations, urinalysis, standard 12-lead electrocardiogram and echocardiography were normal. Magnetic resonance imaging (MRI) of brain revealed evidence of multiple poorly defined T2 hyperintensities involving white matter of cerebral cortex and deep white matter of cerebellar hemisphere bilaterally (R > L), superior vermis, infero-lateral pons and midbrain (Fig. 1), which were hyperintense on PD WI also and were isointense to hypointense on T1 WI (Fig. 2), suggestive of multiple infarcts in the posterior cerebral circulation. Magnetic
resonance angiography (MRA) revealed absent left vertebral artery, narrow posterior cerebral arteries, posterior communicating arteries and A-1 segment of right anterior cerebral artery (Fig. 3). Vertebral angiography could not be done as the patient did not give consent as he had started improving by that time. Patient was treated with aspirin 150 mg/day and follow-up examination, two months after discharge, showed a significant improvement in neurological signs. Six months after the episode he had only mild slurring of speech. Repeat MRA cervical region revealed that there was still no recanalisation of the left vertebral artery, thus a congenital absence of the left vertebral artery could not be excluded.

The neurological signs and symptoms in our patient occurred acutely after sudden head movements, so one would postulate a vascular lesion resulting from indirect neck trauma.

Trauma as a cause of stroke is considered uncommon, however in a recent report non-penetrating arterial trauma was the commonest likely predisposing factor in patients less than 45 years of age, with cervical arterial dissections being the underlying etiology in as many as 20% patients. The anatomical pattern of the vertebral artery at the level of foramen magnum makes the artery more susceptible to injury. Stretching and compression of the vertebral artery occurs on rotation and overextension of head. Other factors like marked variation in the size of two vertebral arteries, defect of odontoid process leading to excessive motion at atlanto-axial joint, and cervical osteophytes may be due to restoration of blood supply to the brainstem as 80% of patients show a reduction in the degree of stenosis on follow-up.

So in a young patient of posterior circulation stroke, without any identifiable risk factor, the physician should enquire about recent neck manipulation, as the association between non-penetrating injury and brain infarction may be covert.

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Non-resolving Choroidal Tubercles May Indicate Multidrug Resistant (MDR) Tuberculosis

Sir,

Choroidal tubercles are a directly visualized infection of the choroid, found in up to 1.4% of patients with pulmonary TB and resolve in 8-12 weeks following therapy. We discuss two cases where the presence of non-resolving tubercles suggested a diagnosis of MDR (multi-drug resistant) tuberculosis.

A 31/M patient, undergoing anti-tuberculous therapy for the last six months (isoniazid, rifampicin, ethambutol and pyrazinamide for 2 months followed by isoniazid, rifampicin and pyrazinamide) for pulmonary and spinal tuberculosis, presented for refraction. Earlier findings included a right-sided pleural effusion and lumbar spinal tuberculosis. Acid-fast bacilli were seen in sputum smears and a mantoux test was positive (24 mm x 12 mm). He had normal visual acuities and anterior segments. A choroidal tubercle of ½ Disc diameters (DD) by ½ DD was seen in the left macula. This finding of an as yet unresolved tubercle suggested MDR-TB and systemic ofloxacin was added. One month later, he developed and breathlessness. A CT scan showed a loculated empyema in the right pleural space. This was drained and *Mycobacterium tuberculosis* was isolated from the aspirate. The five-drug regime was continued for 6 months leading to resolution of the tubercle and the patient becoming clinically and radiologically disease-free.

A 41/M presented with an acute onset high fever. A chest X-ray revealed right and left hilar lymphadenopathy and a CT scan of the chest revealed extensive hilar and mediastinal lymphadenopathy. A histopathological diagnosis of tuberculosis was made from a supraclavicular node biopsy. A choroidal tubercle of ½ DD by 1/2 DD was seen in the left eye. A four-drug regime (isoniazid, rifampicin, pyrazinamide, and ethambutol) was prescribed. Follow-up at one and two months revealed no change in the presence or dimensions of the tubercle. One month later, the patient complained of severe headache with recurrent fever. On examination, CT and cerebrospinal fluid (CSF) findings were suggestive of tuberculous meningitis. A fundoscopy revealed no change in the presence or size of the tubercle. This led us to a diagnosis of MDR and additionally streptomycin and ciprofloxacin were added. There was resolution of the tubercle and significant neurological improvement, by the end of three months of therapy.

MDR-TB is becoming increasingly common with prevalences of 2.5% -3.3% for primary drug resistance and 38.2%-100% for acquired drug resistance.1,2 Diagnostic difficulties remain due to poor follow up or inadequate diagnostic facilities and diagnosis is often based on clinical response. As tubercles are a directly visualized inflammation, their non-resolution may be due to a primary or acquired MDR-TB or poor compliance, theoretically suggesting similar processes elsewhere in the patient. Illingworth3 discussed the natural history of tubercles and commented on their prognostic importance. In eight patients undergoing streptomycin therapy, the tubercles did not undergo changes suggestive of healing. Five of these patients died and in three patients’ recovery was thought to be “unlikely”. In these patients, this finding led us to consider MDR-TB and additional second line therapy allowed a better outcome.

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REFERENCES


Indian Diabetes Guidelines 2002

Sir,

We congratulate the academic wing of Association of Physicians of India on their excellent performance in presenting the Indian Diabetes Guidelines 2002 (J Assoc Physicians India 2002;50:295-366). This was long overdue.

We want to comment on the gestational diabetes mellitus (GDM) section (Section 15.3.b.iii) of the article. In our opinion, the diagnostic criteria proposed by the authors lacks of the results of the prospective population-based observational study conducted by the Brazilian Gestational Diabetes Study Group, 2-h 75-g OGTT (oral glucose tolerance test) seems to
be the right choice for our population. Here, about 5,000 pregnant women underwent 75-g OGTT between 24 to 28 weeks of gestation. The 1999 WHO (World Health Organization) diagnostic criteria was able to diagnose more cases of GDM compared with the 2000 ADA (American Diabetes Association) diagnostic criteria. Women classified only by WHO criteria had lower fasting (90 vs. 103 mg/dl; P < 0.001) but higher 2-h (149 vs. 117 mg/dl; P < 0.001) plasma glucose values than women classified only by ADA criteria. They were also younger, shorter, and leaner. This phenotype is very similar to that of the pregnant women in our population. In the Brazilian study, no statistically significant differences between these two groups were observed regarding frequencies of macrosomia, preeclampsia, or perinatal death. Moreover, the diagnostic cut-off values of the WHO criteria are easier to remember making interpretation of the 75-g OGTT result in pregnant women simpler for health professionals.

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Reply from the Author

Sir,

I entirely agree with the observations made by Shankar and Sundarka. We need a simple and acceptable screening procedure and diagnostic criteria for the diagnosis of GDM. The diagnostic criteria recommended by ADA and WHO are validated and accepted, as diagnosis of GDM based on two-hour 75g OGTT by either WHO or ADA predicts adverse pregnancy outcome. Further in clinical practice two hour glucose level is preferred for the diagnosis of GDM. Moses et al adapted WHO criteria in their study where they used a single 75g OGTT and diagnosed GDM with 2 hour PPG ≥ 140 mg/dl. David Pettitt endorses the data of Moses et al in the editorial on gestational diabetes ‘who to test and how to test’. Further, assuming that the effective treatment is available, WHO criteria of 2 hour PPG ≥ 140 mg/dl identifying a large number of cases may have a greater potential for prevention. Hence for our country WHO screening procedure and diagnostic criteria is highly suitable.

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