Tuberculous Meningitis: A Report of an Unusual Case

Sir,

I read with interest the article by I. Al Traif and his colleagues (*Saudi Med J* 1992; **13**(5): 451–454). Although the authors mentioned several atypical clinical features and initial laboratory data of this unusual case report some points need worth consideration.

First, in the elderly age-group tuberculous (TB) meningitis may present with indolent course with lethargy, fever and mental obtundation as well as with other neurological features. Second, the injection of streptomycin given for the treatment of brucellosis for some days might have masked the patient's symptoms and modified the actual clinical course.

I agree that the clinical features and the initial laboratory findings may cause diagnostic difficulty but an initial polymorphonuclear pleocytosis in CSF is a recognized finding in TB meningitis[1] as well as the finding of a positive sputum for acid fast bacilli and a normal chest film as in endobronchial tuberculosis.[2] I do not agree with the authors regarding the dose of injected streptomycin (1 g daily) in this elderly patient; this is too high (ideally it should be 0.5 g daily or according to the serum level) since the risk of toxicity of streptomycin increases markedly with the dose and with the age of the patient.[3] In addition I do not find any justification for continuing the potentially hepatotoxic pyrazinamide (PZA) for 18 months (though its value in TB meningitis is doubtless). A 12-month course with a regimen containing rifampicin and isoniazid isonicotinic acid hydrazide (INH) supplemented initially for the first 2 months by PZA and streptomycin would have been sufficient since the value of short-course chemotherapy with the above combination of drugs in TB meningitis is well established.[4,5]

However I thank the authors for their endurance in managing the patient.

Dr Mohammad Musleh Uddin
Chest Specialist
Yanbu General Hospital
PO Box 978
Yanbu Albahar
Saudi Arabia
Saudi Medical Journal 1994; **15**(4): 329

References

1. Grossman M, Jawetz E. Tubercular meningitis. In: Crupp MA, Chatton MJ eds. *Current medical diagnosis and treatment*. Lange Medical Publications, Singapore, 1983: 842–843.
2. Ip MSM, Yo SY, Lam WK, Mok CK. Endobronchial tuberculosis revisited. *Chest* 1986; **87**: 727.
3. Girling DJ. Adverse effects of antituberculous drugs. *Bull IUAT* 1984; **59**: 152–162 (Reprinted from *Drugs* 1982; **23**: 56–74).
4. Girling DJ, *et al.* Extrapulmonary tuberculosis. *Br Med Bull* 1988; **44**: 738–756.
5. Humphries M. The management of tuberculous meningitis. *Thorax* 1992; **47**: 577–581.

Sir,

We would like to thank Dr Uddin for his comments regarding our article: Tuberculous Meningitis: Report of an Unusual Case (*Saudi Med J* 1992; **13**(5): 451–454).

We agree with his points regarding:

1. the presentation of elderly age group with indolent course and non-specific complaints and findings;
2. the prior streptomycin therapy for brucellosis that may have masked and modified the clinical course of this case;
3. that polymorphonuclear pleocytosis seen in the cerebrospinal fluid (CSF) and the finding of positive sputum for acid fast bacilli with a normal chest X-ray are recognized, but unusual, findings in tuberculous meningitis and endobronchial tuberculosis respectively. Furthermore, these CSF findings account for 14.5% among 120 cases according to one report.[1] In addition, the cell count in this study ranged from 4 to 650/mm. To our knowledge, our patient cell count of 4000 in the CSF with 100% polymorphs is unheard of and has not been previously reported in tuberculous meningitis.

We concur with Dr Udin regarding anti-tuberculous therapy; however, because of the known advantages of pyrazinamide therapy for tuberculous meningitis as this drug is bactericidal with preferential blood–brain barrier passage, and the slow clinical and radiologic response in this case, this led us to treat him more aggressively for a longer period than usually recommended for routine cases of tuberculous meningitis. Furthermore, pyrazinamide is not more hepatotoxic than INH or rifampicin or the combination of the two.

Regarding streptomycin dosage, this was based on frequent biochemical monitoring and audiogram tests to detect early nephro- and autotoxicity respectively.

Because of the occurrence of many simultaneous and unusual features, we elected to report this case to alert the medical community to some atypical aspects of tuberculous meningitis.

Ibrahim Altraif
Amir Shekh Ali
Yousef Khan
Miltiadis Stefadouros
Liver Transplant Unit and Department of Medicine
King Fahad National Guard Hospital
PO Box 22490
Riyadh 11426
Saudi Arabia
Saudi Medical Journal 1994; **15**(4): 329

Reference

1. Upadhyay GC, Tripathi BN, Sukla RK, Singh KN. Tuberculous meningitis in children: A clinico-laboratory correlation of CSF findings for early diagnosis. *Indian J Pediar* 1984; **51**: 633–636.

Aortoduodenal Fistula: Secondary to Spinal Tuberculosis

Sir,

I read with interest the short communication by Dr A. Al Shahri (*Saudi Med J* 1993; **2**: 168) reporting spinal tuberculosis as a rare aetiology of aortoduodenal fistula. The author indicated rightly that his case is the first reported in the English literature. Though I enjoyed reading this case report, perhaps it is difficult to be convinced about the diagnosis, which was mainly based—according to the report—on the gross intra-operative findings. Usually, the initial presentation of aortoenteric fistula is that of intermittent 'herald' bleeding with self-limiting episodes of melena or haematemesis. These will recur over a period of days or hours prior to the final exsanguinating haemorrhage.[1] Such a case will be documented in a scientific way if an arteriogram, CT or even upper gastrointestinal endoscopy were done in the absence of the postmortem examination and/or tissue diagnosis.

I understand that time did not allow the performance of these investigations, nevertheless, it may not be easy to accept the strong recommendation of immediate laparotomy in such a case.

Dr Hasan Ali Al Zahrani
Medical Director and Associate Professor of Surgery
Consultant of Vascular Surgery
Al Noor Specialist Hospital
Makkah Al Mukarramah
Saudi Arabia
Saudi Medical Journal 1994; **13**(4): 329-330

Reference

1. Kleinman LH, Towne JB, Bernhard VM. A diagnostic and therapeutic approach to aorto-enteric fistulas: Clinical experience with twenty patients. *Surgery* 1979; **86**: 868-873.

Transient Blindness in Pregnancy-induced Hypertension

Sir,

It was very interesting to read two cases reported by Dr Golam M. Zakaria (*Saudi Med J* 1993; **14**(2): 167) which were very similar to the cases reported by us earlier (*Saudi Med J* 1991; **12**: 441). Both cases fulfilled the criteria of eclampsia and the second case had seizures following the complaint of blurring vision, which could well be due to aura of the epilepsy. The importance of early management has been stressed well by the author.

Dr Hassan N. Ranganath
Senior Consultant Neurologist
King Abdulaziz Hospital and Oncology Centre
PO Box 31467
Jeddah 21497
Saudi Arabia
Saudi Medical Journal 1994; **15**(4): 330

Incidence of Human T-cell Lymphotropic Virus Type-I (HTLV-I) Among Healthy Volunteer Blood Donors in Eastern Saudi Arabia: A Preliminary Study

Sir,

HTLV-I has been linked with adult T-cell leukaemia/lymphoma (ATLL), tropical spastic paraparesis (TSP), HTLV-I associated myopathy (HAM), and polymyositis.[1,2] The HTLV-I antibodies are found with high frequency (48%) in persons affected with these disorders, and an association between HTLV-I antibodies and Norwegian scabies has been recently demonstrated.[3] However, the HTLV-I antibody prevalence varies between (<2 and >18%) among apparently healthy subjects in Southeastern Japan, the Caribbean, Tropical and Central Africa, Southeastern United States, Northeastern South America, and parts of South Italy and Israel.[1,4] It is now well-documented that HTLV-I is transmitted mainly through transfusion of infected blood and cellular blood products, intravenous drug abuse, perinatally, and sexually.[3,5,6] And, though HTLV-I is a human retrovirus, yet HTLV-I is remotely related to human immunodeficiency virus (HIV) with no cross-reactivity between them, while HTLV-II is more closely related to HTLV-I, and both viruses are cross-reactive.[2,7,8]

The purpose of this preliminary study (October 1992 through June 1993) was to look for the HTLV-I antibodies among the healthy blood donors in the Eastern Province of Saudi Arabia before any decision could be taken about including such marker in the blood screening programme. In this study we screened 910 blood serum specimens collected from apparently healthy blood donors of different nationalities. We used the Abbott's HTLV-I EIA (ELISA) kits, North Chicago, IL-60064, USA. Our results gave the incidence of (0.1%) as only one specimen was reactive, while the remaining 909 specimens were negative, even their absorbance values were very far below the cut-off value. We reached the conclusion that HTLV-I is not prevalent in the Eastern Province of Saudi Arabia, as even the only repeatedly reactive specimen, was from a donor of African origin.

However, further studies are needed and more research has to be planned to determine whether an additional anti-HTLV-I blood screening test could be of public health interest, or whether there is no need to introduce such a test? Nevertheless, it is well understood why the HTLV-I screening was introduced in Japan for the first time in 1987,[9] and in the USA in 1988,[10] while UK health authorities are undecided whether to include this test in their blood screening programmes.[10,11]

The author is aware of the limited number of subjects in this preliminary study, and is planning to undertake a new trial with more materials and a more sophisticated method of testing. Whatever the screening policy adopted for screening this virus marker or other virus(es) in denoted blood, it is to be borne in mind, that the life-sustaining benefits of blood transfusion are not achieved without risk, since the decision to transfuse must be critically evaluated and the clinical indications for each transfusion should be unequivocal and clearly justified.

The author would like to express his thanks to Mrs Priscilla Garcia Jimenez, Mrs Claire Mababa Galima, and Ms Aida Gimena the medical technologists, Virology Department, Dammam Regional Laboratories and Blood Bank, Dammam for their technical help.

Dr Sami El-Sayied Fathalla
Consultant Clinical Virologist
Dammam Regional Laboratories and Blood Bank
PO Box 4103
Dammam 31491
Saudi Arabia
Saudi Medical Journal 1994; **15**(14): 330-331

References

1. Wong-Staal F, Gallo R. Human T-lymphotropic retroviruses. *Nature* 1985; **316**: 395-403.
2. Dalgleish AG, Weiss RA. Human retroviruses. In: Zuckerman AJ, Bantavala JC, Pattison JR, eds. *Principles and practise of clinical virology* 2nd edn. Chichester, UK: John Wiley, 1990: 573-578.
3. Mollisan LC, Lo STH, Marning G. HTLV-I and scabies in Australian Aborigines. *Lancet* 1993; **341**: 1281-1282.
4. Blayney DW, Blattner WA, Robert-Guroff M, *et al.* The HTLV-I in Southeastern United States. *JAMA* 1983; **250**: 1048-1052.
5. Sandler SG. HTLV-I and II: New risks for recipients of blood transfusion. *JAMA* 1986; **256**: 2245-2246.
6. Anonymous. Virus diseases; HTLV-I. *Wkly Epidemiol Rec* 1989; **49**: 382-383.
7. Murphy EL. HTLV-II related disease. *Lancet* 1993; **341**: 888.
8. Wiktor SZ, Alexander SS, Shaw GM, *et al.* Distinguishing between HTLV-I and HTLV-II by Western blot. *Lancet* 1990; **335**: 1533.