Post Med J (1990) **66**, 155 - 157

© The Fellowship of Postgraduate Medicine, 1990

Letters to the Editor

Control of warfarin therapy

Sir,

We believe that Dr Scott, in his recent leading article, 'Control of warfarin therapy (*Postgrad Med J* 1989, **65:** 611-612), dismissed predictive schedules for warfarin therapy rather lightly. Our own schedule for example is widely used both in the UK and abroad[1,2] and was the subject of a recent encouraging validation study in patients requiring anticoagulants, including the elderly,[3] and is now printed on the reverse of the inpatient anticoagulation treatment sheets in our own hospital. Although it was less accurate in patients with serious and fluctuating medical conditions, such as unstable cardiac failure and acute alcoholism, the scheme still proved useful.[3] The suggested 'try it and see' scheme proposed by Scott, of 9-10 mg of warfarin for 3 days results in over 30% of patients being overanticoagulated, some dangerously so.[4,5] This situation is avoided when the prothrombin time (INR) is monitored and the warfarin dose adjusted after each induction dose of warfarin. After 3 doses of warfarin, the patient may (if his condition allows) be sent home on a predicted maintenance dose which will be within 1 mg of the final maintenance dose, in 75-80% of cases.[3] Thus the patient can be spared unnecessary time in hospital solely to monitor the INR and this should bring savings to the health service.

A. Fennerty
P.A. Routledge
University of Wales College of Medicine, Department of Pharmacology and Therapeutics, Heath Park, Cardiff CF4 4XN, UK.

References

1. Fennerty, A.G., Dolben, J. & Thomas, P. Flexible induction dose regimen for warfarin and prediction of maintenance dose. *Br Med J* 1984, **288:** 1268-1270.
2. Fennerty, A.G., Campbell, I.A. & Routledge, P.A. Anticoagulants in venous thromboembolism. *Br Med J* 1988, **297:** 1285-1288.
3. Cosh, D.G., Moritz, C.K., Ashrian, K.J., Dally, R.J. & Gallus, A.S. Prospective evaluation of a flexible protocol for starting treatment with warfarin and predicting its maintenance dose. *Aust N Z J Med* 1989, **19:** 191-197.
4. Routledge, P.A., Davies, D.M., Bell, S.M., Cavanagh, J.S. & Rawlins, M.D. Predicting patients' warfarin requirements. *Lancet* 1977, **ii:** 854-855.
5. Thomas, P., Fennerty, A., Backhouse, G., Bentley, D.P., Campbell, I.A. Routledge, P.A. Monitoring effects of oral anticoagulants during treatment with heparin. *Br Med J* 1984, **288:** 191.

Trichomoniasis and cystic fibrosis

Sir,

I would like to make a few points with regard to the paper of Dr Gordon J. Canny.[1] He reports the liver abscesses of the siblings with cystic fibrosis to be sterile, with pseudonomas grown later from one with transcutaneous drainage, I think there are two reasons why material from liver abscess or from other organs should be examined for *Trichomonas* species in patients with cystic fibrosis, especially in sterile cases. The first is that trichomonads have been grown on several occasions from liver abscesses, the first being 6 decades ago.[2] The second is the reported close relation between human trichomonads and cystic fibrosis.[3]

After long-standing comparative research of the clinical phenomenology of human trichomoniasis and cystic fibrosis, I became convinced of their intimate relationship which is much more than occasional. According to my 'unorthodox' trichomonal conception of the aetiology of cystic fibrosis, the hereditary factor is in the sphere of genetic regulation of susceptibility to trichomonads in conditions of familial trichomoniasis and congenital infection of future cystic fibrosis patients. That susceptibility is also inherited by a recessive model.[4] I therefore believe that the liver abscesses of both Canny's patients cannot be considered sterile without attempts to exclude trichomonads.

There are difficulties in diagnosing trichomonads in atypical sites because they are most frequently aflagellate round pseudocystic forms. The cultures are uncertain even in microscopically positive cases. Immunodiagnostic methods can fail as well because of antigenic variability of these protozoa in subepithelial locations. All three species of human trichomonads are immunogenic[5] and can cause immunodepression. Therefore irrespective of the precise relationship between trichomoniasis and cystic fibrosis, there is a need to attempt detection and eradication of these underrated protozoa in cystic fibrosis patients. Careful trials of metronidazole are justified not only for its effect on trichomonads and anaerobic bacteria but also for its unexplained regenerating effect on the host immune apparatus.[6,7]

S. Krvavac
Women's Health Institute, University Medical Center, Sarajevo, Adr. 71000 Sarajevo, Alajbegovica 1, Yugoslavia.

References

1. Canny, G.L. Hepatic abscess and cystic fibrosis. *Postgrad Med J* 1989, **65:** 506.
2. Kessel, J.F. The ingestion of erythrocytes by *Trichomonas hominis* and its occurrence in the pus of an amoebic liver abscess. *J Parasitol* 1925, **11:** 151-152.
3. Krvavac, S. Aflagellary forms of *Trichomonas* species as etiologic agent of cystic fibrosis. *XIIth World Gyn/Obst Congress*, Rio de Janeiro 1988, Abstracts, pp. 681-682.
4. Wakelin, D. Genetic control of susceptibility and resistance to parasitic infection. *Adv Parasitol* 1978, **16:** 219-308.
5. Honigberg, B.M. Trichomonads of importance in human medicine. In: Kreier, J.P. (ed.) *Parasitic Protozoa*, vol. 2. Academic Press, New York 1978, pp. 275-454.
6. Shroit, I.G., Anisimova, L.A., Khodyveva, G.D. *et al.* Influence of metronidazole on the course of experimental anaerobic streptococcal pneumonia. *J Microb Epidemiol Immunol* 1986, **4:** 21.

Postgrad Med J: first published as 10.1136/pgmj.66.772.155 on 1 February 1990. Downloaded from http://pmj.bmj.com/ on January 15, 2021 by guest. Protected by copyright.