Fish oils and glomerulonephritis

Sir,
I agree that fish oils administered at high dosage will reduce access thrombosis [1] and that fish oils lower serum triglycerides [2] and so protect against atherosclerosis. However, the evidence that fish oils really benefit patients with glomerulonephritis deserves closer scrutiny. In spite of impressive lymphocyte suppression in mice or in vitro, results accumulated over two decades show only a modest benefit of fish oils on eicosanoid production and cytokine release and on the clinical status of persons with rheumatoid arthritis, and other Th-1 lymphocyte-mediated pathologies [3].

We were reliably informed that fish oils could reduce the decline of creatinine clearance, and yet not reduce proteinuria significantly [4]. At the ISN IgA Nephropathy Conference in Leiden in 1998 we noted that fish oils did not reduce urine thromboxanes, as anticipated. Now Grimble [5] reports that in UK subjects, fish oils will increase serum TNFα in half, and lower it in the other 50%

Nephrologists should stop extrapolating results from dietary experiments in mice to what might happen in humans, and carry out more detailed investigations of their own patients who participate in trials.

21 Common Road
North Leigh
Oxford OX29 6RD, UK


Steroid therapy in chronic interstitial renal fibrosis: the case of Chinese-herb nephropathy

Sir,
Chinese-herb nephropathy (CHN) is a progressive interstitial renal fibrosis, initially reported to occur after exposure to herbal medicine containing aristolochic acids [1,2]. In some CHN patients, corticosteroid therapy (Cs) was successfully attempted to slow the rate of progression of the disease. The first pilot study involved 14 CHN patients treated with Cs, with 12 of them included in the 1-year follow-up study already published [3], whose clinical evolution was compared with 23 other control CHN patients not given corticosteroids (nCs). After 1 year of observation, only two of the 12 Cs CHN patients required renal replacement therapy (RRT) compared with 16 of the 23 nCs CHN control cases (P=0.0045). Interestingly, in a recent study dealing with the relationship between the rate of progression of the renal disease in our CHN patients and the cumulative dose intake of Chinese herbs, we found that Cs treatment had actually slowed the progression to end-stage renal disease (ESRD) in some CHN patients [4]. As this report raised some questions [5], we decided to investigate further the role of Cs therapy in the progression rate of CHN; first, by actualizing at 8 years the follow-up of the pilot study cohort (study 1); and secondly, by focusing on Cs therapy in the analysis of renal disease progression rate in the patient groups of the study of dose relationship (study 2).

According to the pilot study, Cs therapy (treatment is detailed in [3]) was actually proposed to all CHN patients referred to us with moderate but progressive renal failure. This means that Cs therapy was proposed neither to patients with preterminal renal failure [plasma creatinine (Pcreat) > 4 mg/dl] nor to patients with stable renal function. Fourteen CHN patients were included in the Cs group of the pilot study (study 1). They were also taken into account in study 2, as well as nine additional CHN patients having received Cs after completion of the pilot study. Two of these nine patients were given Cs despite the fact that Pcreat was > 4 mg/dl (4.1 and 5.8 mg/dl, respectively). For the nCs CHN control group of patients, there was a significant difference between study 1 and study 2. For study 1, according to the pilot study protocol, nCs CHN patients with preterminal renal failure or stable renal disease, similar to the Cs group, were excluded from the control group, while 11 patients cared for in other nephrology centres were included in the 23 nCs CHN control group. For study 2, on the other hand, all nCs CHN patients from our centre were included whatever the degree of renal failure, while patients from other centres were excluded. This means that studies 1 and 2 had only 12 patients of the control group in common.

For study 1, the results of the follow-up of the 37 CHN patients (14 Cs, 23 nCs) are shown in Table 1. After 1 year, 12 out of 14 Cs patients escaped RRT, in comparison with seven out of 23 nCs patients (P=0.0019). Three years later, the evolution of the same cohort was re-analysed: six of the 14 Cs CHN patients were still receiving conservative treatment, compared with two of the 23 nCs CHN patients (P=0.0345) [6]. At 8 years, the corresponding figures were three out of 14 and one out of 23, respectively (not significant, P=0.1419) (Table 1).

For study 2, a total of 22 CHN patients treated with Cs were finally identified in our database [4]. Fifteen of them belonged to the ESRD group (n=44), while the remaining seven were in the chronic renal failure (CRF) group (n=27). Within these two groups, no statistical difference was found between the mean (±SEM) cumulative dose of the so-called Stephania tetrandra—actually replaced by Aristolochia fangchi (symbolized by ST-AF)—ingested by Cs and nCs patients [ESRD group: 215 ± 24 vs 180 ± 15 g, respectively (P=0.20); CRF group: 125 ± 19 vs 143 ± 21 g, respectively (P=0.64)]. In contrast, among Cs-treated patients, significantly higher amounts of ST-AF had been ingested by patients who developed ESRD further (Table 2).

In this ESRD group, levels of Pcreat at the initiation of Cs were significantly higher and a treatment with Cs was initiated more rapidly compared with CRF patients,