Pathogenesis and management of complications of chronic peritoneal dialysis

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Introduction

In a recent editorial Dr Blake and I speculated on some of the reasons behind the recent decline in peritoneal dialysis (PD) use in North America [1]. Among other reasons, we speculated that many nephrologists, who are familiar only with haemodialysis, feel uncomfortable in managing PD patients, especially those with complications. The PD patient, who has some residual renal function, can be managed easily, but once the patient becomes anuric and develops complications, his/her management becomes difficult, time consuming, and requires a high degree of expertise. Therefore, it is vital to educate physicians, who wish to become expert in the field of PD. With similar thoughts in mind, the organizers of the BANTAO Conference decided to dedicate a round table discussion to four major complications of peritoneal dialysis.

Prevention and management of peritonitis and exit-site infections

Although its frequency has decreased, peritonitis still remains a major complication of chronic PD. The introduction of the various disconnect systems have eliminated mainly the mild peritonitides, i.e. those due to skin contaminants such as *Staphylococcus epidermidis*, whereas the serious peritonitides like those due to *Staphylococcus aureus*, pseudomonas and fungi still remain a major problem. Regarding treatment of peritonitis, an advisory committee of the International Society for Peritoneal Dialysis has updated and published its recommendations for such treatment [2].

With regards to *S. aureus* peritonitis I believe that there has been a major development in the field especially with regard to the elimination of *S. aureus* exit-site infection, and the subsequent peritonitis that often follows. Use of Mupirocin either at the nares, or at the catheter exit-site not only decreases *S. aureus* exit-site infections but also *S. aureus* peritonitis [3]. The evidence for this is strong enough that I believe we should use Mupirocin at the exit site, starting from the day of catheter implantation and that this should be standard practice in all peritoneal dialysis units. In this supplement, Dr V. Vargemezis reviews this experience as well as recent recommendations of the ISPD advisory committee on the treatment of peritonitis.

Malnutrition and the role of inflammation in its pathogenesis

Since the introduction of PD, it has become evident that patients on CAPD lose large amounts of protein in the effluent and a high percentage of these patients develop either mild or severe malnutrition [4].

Initial attempts in managing these malnourished CAPD patients were directed at replacing the protein losses by the intraperitoneal administration of amino acids [5]. Subsequently, when we recognized the importance of adequate dialysis in maintaining the patients’ appetite, we began to emphasize an increase in the dose of dialysis as a main measure to improve nutrition in these patients [6].

However, these two interventions have not always been able to restore the nutrition status of the patients. Whereas I believe that both intraperitoneal infusion of amino acids and an adequate dialysis dose have a place in the management of malnutrition in CAPD patients, we must consider that additional factors may be responsible for the lack of response in some patients.

Recently Bergström et al. [7] and other investigators have emphasized the role of chronic inflammation in the development, not only of hypoalbuminaemia and malnutrition, but also of atherosclerosis. In those patients in whom inflammation is the main cause of malnutrition, it seems that no matter how much protein...
and how large a dose of dialysis the patient receives, the nutritional status will not improve unless one finds and corrects the cause of inflammation.

Fortunately there is a simple method of measuring the inflammatory state, C-reactive protein (CRP), that shows an inverse correlation between the serum albumin and the patient’s nutritional status [8]. With this test, we now can identify patients, who will respond to an increased dose of dialysis or nutritional interventions (i.e. those with a normal CRP), whereas in patients, who have a chronic inflammation (i.e. an elevated CRP), we should direct treatment towards controlling the inflammatory state.

In this supplement, Dr N. Dombros discusses the pathogenesis and management of malnutrition in CPD patients.

Ultrafiltration failure

As we gained experience with patients on long-term PD, the syndrome of ultrafiltration failure emerged as an important complication of CAPD; indeed a significant percentage of patients fail treatment because of ultrafiltration failure and difficulties with fluid management. We are concerned when we find a decrease in ultrafiltration capacity because fluid-overloaded patients tend to become hypertensive and this may increase the number of cardiovascular deaths. Furthermore, a number of these patients may develop sclerosing encapsulating peritonitis, which often is a fatal complication. Therefore, prevention, early detection and proper management of ultrafiltration failure are critical to the long-term survival of patients on CAPD. Because of the importance of this complication, a special advisory committee of the International Society for Peritoneal Dialysis has made recommendations on the diagnosis, treatment and prevention of ultrafiltration failure which were published in a recent supplement of *Peritoneal Dialysis International* [9].

As increased use of hypertonic solutions may contribute to the development of ultrafiltration failure, it seems reasonable to decrease the need for such fluids by controlling oral fluid intake.

Dr F. Akcicek and his team have emphasized the importance of salt restriction in the control of thirst and subsequent increased fluid intake and thus avoiding frequent use of hypertonic exchanges and he has described this approach elsewhere [10]. In this supplement, Dr F. Akcicek describes his experience with ultrafiltration failure and reviews the special advisory committee’s recommendations.

High and low bone turnover diseases

Our understanding of renal osteodystrophy in patients on CAPD has gone through various stages. Initially, when the late George Degenis reviewed our experience with this condition at the Toronto Western Hospital, he found a progression of osteitis fibrosa [11]. Subsequently, however, Gokal *et al.* showed that renal osteodystrophy was well controlled in patients on CAPD [12]. At that time we attributed the difference between the two centers to the higher use of calcium carbonate in Gokal’s unit.

In a subsequent development, new dialysis solutions with low (in comparison to the standard solutions but actually normal in comparison to serum) dialysate calcium were introduced to enable nephrologists to give higher doses of calcium carbonate to control serum phosphorus while preventing hypercalcemia. Unfortunately, despite all our efforts, the use of calcium carbonate does not give adequate control of serum phosphorus in CAPD patients in whom the single most important factor responsible for the progression of hyperparathyroidism seem to be high serum phosphorus levels [13]. Forty-seven per cent of our PD patients have hyperparathyroidism [13] and 3.3% of these patients eventually require parathyroidectomy [14].

Using the combined experience of Seattle and Toronto, Sherrard *et al.* described the entity of ‘adynamic bone disease’ which they found was more frequent among PD patients than among those on haemodialysis [15]. This entity is characterized by a low PTH level and a tendency to hypercalcemia, even after small doses of calcium carbonate. Recent investigators have found a higher incidence of fractures among their patients with adynamic bone disease [16].

In the management of CAPD patients it is important to prevent hyperparathyroidism and adynamic bone disease; in this supplement, Dr N. Dimkovic describes these two conditions and discusses their prevention and management in PD patients.

References

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