What is new in peritoneal dialysis in the years 2003-2004

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Abstract

This review paper describes new data on the role of aquaporins in peritoneal function, peritoneal membrane permeability, peritoneal dialysis (PD) adequacy and peritoneal membrane histology, new aspects of the use of PD solutions alternative to glucose ones and indications for use of peritoneal catheters with different configurations.

Results of main studies, published predominantly in 2003-2004, are presented and discussed. Clinically important news, although preliminary in some cases, are also included.

Key words: aquaporins, peritoneal membrane, dialysis solutions, catheters.

Introduction

The following subjects will be discussed:

1. The role of aquaporins (APQs) in peritoneal function,
2. Peritoneal membrane permeability, peritoneal dialysis (PD) adequacy and peritoneal membrane histology,
3. New aspects of the use of PD solutions alternative to glucose ones,
4. Indications for use of peritoneal catheters with different configurations.

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The role of AQPs in peritoneal function

When expression of cellular water channels – AQPs – was found in kidney tissue, exploration of peritoneum for AQPs was also initiated. In human peritoneum mRNA for AQP1, AQP3 and AQP4 was shown [1]. The localization of AQP1 protein in peritoneal mesothelial cells was confirmed by double immunohistochemical staining of the mesothelial lining of human peritoneal membrane. Immunohistologic studies revealed different localization of AQP1 and AQP3 in human peritoneal mesothelial cells, with apical localization of AQP1 and basolateral localization of AQP3 [2]. In mice, the presence of mRNA for AQP1, AQP3, AQP4, AQP7 and AQP8 was reported in peritoneum [3]. In the rat peritoneum, mRNA for AQP1 and AQP4 was shown [4].

AQPs, which are present in human peritoneum, were also found in other human organs. AQP1 was detected in kidneys (proximal tubule, descending thin limb of the loop of Henle, the descending vasa recta), chorid plexus, eye, gall bladder, lung, red blood cell, endothelium of non-fenestrated capillaries. AQP3 is present in kidney collecting ducts (basolateral membrane), lung, gastrointestinal tract, chorid plexus and eye. AQP4 was found in brain, lung and kidney [2,5].

Important progression of investigations in cell cultures was achieved with detection that glucose itself or hyperosmotic conditions, caused by high glucose concentrations (4%), increase expression of mRNA and synthesis of AQP1 protein in endothelial cells of human umbiliculus vein [6] and in rat peritoneal mesothelial cells [7]. These data suggest that during PD a number of AQP1 may increase due to action of hyperosmotic solutions containing glucose, if cells responsible for AQPs formation are capable to answer on stimulus like hyperosmotic glucose solution. Chronic exposition of peritoneum on dialysis solution with low pH (3.5) causes a decrease of mRNA for AQP1 and AQP4 in the rat model of PD [4].

Not all AQPs are involved in transperitoneal ultrafiltration. Studies on AQP1-null mice or AQP4-null mice have shown that AQP4 plays no significant role in peritoneal water permeability,

whereas in AQP1-null mice the rate of increase in the volume of a hypertonic dialysate was reduced to about 40% of the level seen in control mice [3]. Results of these studies also indicate that a presence of AQP1 is not the only condition causing transperitoneal ultrafiltration under hypertonic circumstances. Investigations on rats with renal-vascular hypertension revealed that greater ultrafiltration is accompanied by higher expression of AQP1 and AQP4 [8]. Expression of AQPs may be decreased by administration of angiotensin converting enzyme inhibitors (ACEI) or drugs which are antagonists for angiotensin II receptor (AIIA). Action of ACEI or AIIA results in decreased transperitoneal ultrafiltration [8].

Reduced expression of AQPs as a cause of deteriorated ultrafiltration should be suspected when patients with decreased ultrafiltration demonstrate impaired sodium sieving, but peritoneal transport of small solutes is not increased [2].

Attempts to demonstrate a decreased number of AQP1 in patients with impaired peritoneal ultrafiltration capacity were, however, not successful. Changes in AQP1 expression were not shown during peritonitis in human [9] and in the rat model of peritonitis. A presence of AQP1 was also revealed in a long-term patient with impaired transcellular water transport [10]. A question arises, what is really important – a number or a function of AQPs. It cannot be excluded that glycation of proteins of AQPs is a reason for loss of their physiological functions [2].

Peritoneal membrane permeability, PD adequacy and peritoneal membrane histology

Long-term exposure of peritoneal membrane to bioincompatible dialysis solutions and episodes of dialysis-related peritonitis lead to functional and structural changes in peritoneum of PD patients.

Functional changes of peritoneal membrane are relatively easy to check with repeated performance of peritoneal tests, like peritoneal equilibration test – PET [11], standard permeability analysis – SPA [12], personal dialysis capacity – PDC [13,14]. In 2003, reference values of SPA were established [15], and PET was modified by the use of a radiopharmaceutical: ^{99}Tc – diethylenetriaminepentaacetate – ^{99}Tc – DTPA, which was intravenously injected at the end of peritoneal instillation of 2 L of 2.5% glucose – containing PD solution as a bolus [16]. Comparing to standard PET, nuclear PET may have the added advantages of simplicity and a possibility of measuring total clearance (PD + renal) in the same sitting. All afore-mentioned tests determine transperitoneal movement of small solutes. SPA and PDC additionally determine peritoneal ultrafiltration capacity, function of water channels and peritoneal transport of large molecules. However, standard PET remains the most popular method of functional evaluation of peritoneal membrane.

Numerous papers indicate changes in peritoneal permeability in the course of PD treatment. An increase in peritoneal permeability is predominantly described. It occurs usually after at least 2 years of PD duration [17-21]. Increments in peritoneal permeability were also observed in the rat model of PD [22]. Reports on decreasing permeability of peritoneum during PD treatment also appear in the scientific literature [19,23-27].

Recent studies indicate that the normal anatomic peritoneum (the mesothelium and associated surface coatings, stagnant layers and the connective tissue immediately adherent to the mesothelium) is relatively unimportant as a physical transport barrier and does not provide a major limitation to small solutes permeability and osmotic ultrafiltration in PD [28]. In cases of alterations of the peritoneum over years, this conclusion may not apply.

Conventional dialysis solutions, containing high glucose lood, lead to formation of advanced glycation end-products (AGE) in peritoneum. In diabetic patients non-enzymatic glycation of proteins begins already in pre-dialytic period. Accumulation of AGE in peritoneum leads to increased permeability of peritoneal membrane. Diabetes mellitus, according to CANUSA studies, is more frequently related to high peritoneal permeability than other diseases causing renal insufficiency [29]. Diabetic patients show higher peritoneal transport, because AGE accumulation in peritoneum and in dialysate effluent is significantly greater in diabetics than in non-diabetics. Differences in peritoneal transport between insulin-dependent patients and insulin-nondependent ones were not demonstrated [30]. However, not all authors confirm more frequent occurrence of high transport in diabetics compared to non-diabetics [31,32]. Dimkovic et al. [33] showed even a greater number of low transporters among Asian Indian patients with diabetes than in non Indian ones. Recent study, using PDC [13], demonstrated that peritoneal function, including peritoneal membrane transport and peritoneal permeability to protein, was significantly higher in diabetics than in non-diabetics [34]. Therefore, hypoproteinemia in PD diabetic patients might be associated with high permeability of peritoneal membrane [34].

In 2003, PD adequacy parameters were related to histologic changes of peritoneal membrane, shown in a peritoneal biopsy at initiation of PD treatment and after a mean of 4 years on continuous ambulatory PD [35]. The main histologic changes were loss of mesothelial cells and decrease in normal mesothelial surface, thickening of the submesothelial collagenous zone, and presence of vascular hyalinosis. Only a trend was observed toward more severe lesions in patients treated with PD for about 4 years than in those starting PD. These not significant structural changes were not followed by functional changes during the first 4 years on PD. However, this study is limited by the small number (n = 18) of patients included [35].

After numerous experimental investigations, clinical trials with glycosaminoglycans have been already started to slow the progressive reduction in the dialytic efficiency of the peritoneal membrane. Intraperitoneal use of hyaluronan for 6-hour dwell in PD patients did not reveal significant changes neither in ultrafiltration nor peritoneal transport, but there was no adverse events related to hyaluronan administration [36]. Further studies with oral treatment of long-term PD patients with the glycosaminoglycan sulodexide showed that this drug improves some functional peritoneal membrane parameters (induces an increase in D/P urea and D/P creatinine and a decrease in peritoneal albumin loss), but it is inclear whether this therapy may be a strategy effective in stopping peritoneal dialytic failure [37].

New therapeutic strategies aiming to protect the peritoneal membrane from the consequences of long-term PD include [38,39]:

1. administration of L-arginine analogues,
2. modulating angiogenesis using agents that inhibit endothelial cell growth, adhesion and cell migration, or that interfere with vascular growth factors VEGF and βFGF, or their receptors,
3. gene therapy

   a) peritoneal mesothelial cells or peritoneal leukocytes can be modified to express antiinflammatory cytokines, as IL-1 receptor antagonist, the soluble receptor to TNF-α and IL-10,

   b) membrane integrity could be preserved enhancing the expression of fibrinolytic factors (tissue plasminogen activator) and anti-fibrotic molecules that counteract VEGF action and inhibit factor Kappa B and transforming growth factor β.

New aspects of the use of PD solutions alternative to glucose ones

Malnutrition is common among PD patients. Numerous factors lead to depletion of body tissue and nutrients, among them reduced nutrient intake, reflecting disturbed appetite, was recently proved in PD patients using electronic appetite rating system [40].

Amino acid-based dialysis solution (AA-DS) was designed to replace transperitoneal losses of amino acids and proteins during PD, thereby improving PD patients' nutrition. After many correction, AA-DS exerts beneficial nutritional effects, including improved nitrogen balance, increased concentration of plasma proteins, improved anthropometric measurements and improved plasma amino acid pattern [41,42]. However, Brulez et al. [43] have observed already in 1999 that absorption of L-methionine from AA-DS induces an increase in the plasma level of homocysteine by about 40% after use of AA-DS for 2 months and, therefore, increases the potential risk of cardiovascular illness. Perhaps AA-DS formulation can be still optimised. Recent data also demonstrate that AA-DS increases serum homocysteine level irrespective of patient sex, age, underlying disease, or diabetic status, and suggest that L-methionine content in AA-DS should be lower than 85 mg/dL, but optimal concentration was not established as yet [44].

It was shown in 2001 that leptinemia of patients switched to icodextrin dialysis solution is lower compared to that of a control group continuing to receive treatment with a glucose – based solution only [45]. Further studies of the same authors revealed that icodextrin administration leads to an increase in leptin peritoneal clearance, presumably as a consequence of increased ultrafiltration [46]. However, the previously established decrease in leptinemia during long-term icodextrin treatment cannot be simply an effect of an increased icodextrin peritoneal clearance. A role in the decrease in leptinemia during icodextrin treatment could also be played by reduced leptin synthesis following the decrease in glucose load and/or hyperinsulinemia and body fat mass in the long run [46].

Episodes of icodextrin-associated sterile peritonitis in patients maintained on chronic PD have been repeatedly described. In many cases this syndrome is caused by contamination of icodextrin with a gram-positive bacteria-derived peptidoglycan, a nonendotoxin pyrogen capable of provoking the inflammatory response in peritoneum. It is established that only a < 10 ng/ml peptidoglycan level fluid should be used, but some patients may become hypersensitive to the peptidoglycan contaminant in icodextrin solution. Such patients may only be able to tolerate fluid containing no peptidoglycan [47,48]. In contrast to bacterial peritonitis, there is no increase in CD14 (receptor for lipopolysaccharide) expression on the peripheral and peritoneal macrophages on the day of presentation and during the follow-up period of icodextrin-associated peritonitis [49].

Indications for use of peritoneal catheters with different configurations

With development of PD treatment, different types of peritoneal catheters are commercially available. Precise indications for their choice for individual patient are not well established. Chinese group of clinical investogators have published in 2003 the results of a prospective randomized controlled trial in PD patients who received a conventional straight double-cuffed catheter, a swan-neck straight catheter, or a swan-neck curled tip catheter [50]. These three different types of PD catheters did not have markedly different outcomes. However, there was a trend toward lower risk of exit site infections in swan-neck catheters, and significantly fewer swan-neck catheters were removed because exit site infections. The main benefit of swan-neck catheters was found in nasal non carriers of Staph. aureus, but swan-neck curled tip catheters had a high migration rate. Swan-neck straight catheters are therefore recommended by authors as the first-line catheters of choice, particularly in populations with a low rate of Staph. aureus nasal carriage.

Due to frequently observed migration of intraabdominal part of Tenckhoff catheter, techniques for correction of its position in the peritoneal cavity are continuously elaborated. Recently, the double guidewire method was introduced by Taiwan group [51]. The first guidewire is used to correct the direction of the catheter tip and the second guidewire is used to anchor the catheter so that an ideal course of the catheter can be maintained during removal of the first guidewire.

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