The Effect of Angiotensin II Receptor Blocker on Peritoneal Membrane Transports in Continuous Ambulatory Peritoneal Dialysis Patients

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Objective: *The objective of this study was to examine the effects of angiotensin II receptor blocker (ARB), used as an antihypertensive medication, on peritoneal membrane transporters in continuous ambulatory peritoneal dialysis (CAPD) patients.*
Material and Method: *Prospective and cross-over experimental study of peritoneal membrane transporters was conducted in 7 CAPD patients with hypertension. All previous antihypertensive drugs had been replaced by candesartan at the dose of 8-16 mg/day to control blood pressure below 140/90 mmHg. Hydralazine, which has no effect on peritoneal membrane transports, was added if the target blood pressure was not achieved. All patients had received candesartan for 12 weeks, then, were retreated with the previous antihypertensive drugs for another 6-week period. The modified peritoneal function tests assessing peritoneal membrane transports were performed at 1) baseline, 2) 6 weeks, 3) 12 weeks following candesartan treatment, and 4) 6 weeks after candesartan withdrawal.*
Results: *The blood pressure target was achieved in all patients and was not different among the 4 periods. The albumin clearance and 4-hour albumin loss were significantly decreased following candesartan treatment ($p < 0.05$). Both values returned to the high baseline levels after 6 weeks of candesartan withdrawal. There were no significant changes in net ultrafiltration and various small and large solute transports. No adverse effects, including hyperkalemia or increased erythropoietin dosage, had been observed.*
Conclusion: *In hypertensive CAPD patients, candesartan could provide nutritional benefit by attenuating peritoneal loss of albumin and provides an effective antihypertensive action. Furthermore, candesartan does not impair other solute transports and net ultrafiltration.*

Keywords: *Angiotensin II receptor blocker, CAPD, Peritoneal membrane transports*

J Med Assoc Thai 2006; 89 (Suppl 2): S188-95
Full text. e-Journal: *http://www.medassocthai.org/journal*

Cardiovascular disorder is one of the most common causes of morbidity and mortality in end stage renal disease (ESRD) patients treated with renal replacement therapy including continuous ambulatory peritoneal dialysis (CAPD). Administration of various cardiovascular drugs has been reported, through various mechanisms, to alter solute and water transports[1].

Increasing evidences have shown that renin-angiotensin-aldosterone blockade with angiotensin converting enzyme inhibitors (ACEI) and angiotensin II receptor blockers (ARB) could provide significant cardiovascular benefit in ESRD patients[2,3]. In the placebo-controlled studies of CHARM, ARB significantly reduced cardiovascular mortality and morbidity in patients suffering from heart failure[4]. Moreover, in the

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CHARM "added" trial, the ARB offered additional protection from cardiovascular death when supplementing the drug to the initial routine therapy containing ACEI[5]. In CAPD, both *in vivo* and *in vitro* studies involving the effects of ACEI on peritoneal membrane transporters have yielded different results[6-12]. There are scarce data regarding the effects of ARB on peritoneal solute and water transports in CAPD patients.

The present study was carried out in CAPD patients to examine the effects of ARB, used as an antihypertensive drug, on the peritoneal membrane transports.

Material and Method

Patients

This was a prospective, cross-over experimental study (Fig. 1). The effects of ARB on peritoneal membrane transports were studied in 7 ESRD patients maintenance with CAPD for at least 6 months at King Chulalongkorn Memorial Hospital, Bangkok, Thailand. The present study was approved by the Ethics Research Committee, Chulalongkorn University Hospital, Bangkok Thailand. Each CAPD patient participating in the present study gave informed consent. Inclusion criterion was CAPD patients who had hypertension documented by blood pressure above 140/90 mmHg. Exclusion criteria comprised CAPD patients who had 1) uncontrolled blood pressure of higher than 180/110 mmHg or hypertension requiring more than 3 different kinds of antihypertensive drug, 2) tunnel infection or CAPD-related peritonitis within 1 month prior to or during the present study period, 3) human immunodeficiency virus infection, 4) chronic liver diseases, and 5) active systemic infection.

Method

During the present study, the CAPD schedule treatment, four 2-liter exchanges daily, was unchanged in all patients. All previous antihypertensive medications in the patients were withdrawn and replaced with candesartan [Takeda, Thailand], an ARB, at the dose of 8-16 mg/day to control blood pressure below 140/90 mmHg (Fig. 1). If this target level of blood pressure was not achieved, hydralazine, which has no effects on peritoneal membrane transports[13], was added to optimize the blood pressure. Candesartan or candesartan plus hydralazine were continued for a period of 12 weeks. The patients were then withdrawn from candesartan and retreated with the previous antihypertensive medications for another 6-week duration. At baseline, 6 weeks following candesartan treatment (6-week candesartan period), 12 weeks following candesartan treatment (12-week candesartan period), and 6 weeks after candesartan withdrawal, the modified peritoneal function tests were carried out to assess peritoneal membrane transports. Blood samples of the 4 periods were examined to determine hematology and biochemistry data. The modified peritoneal function test was per-

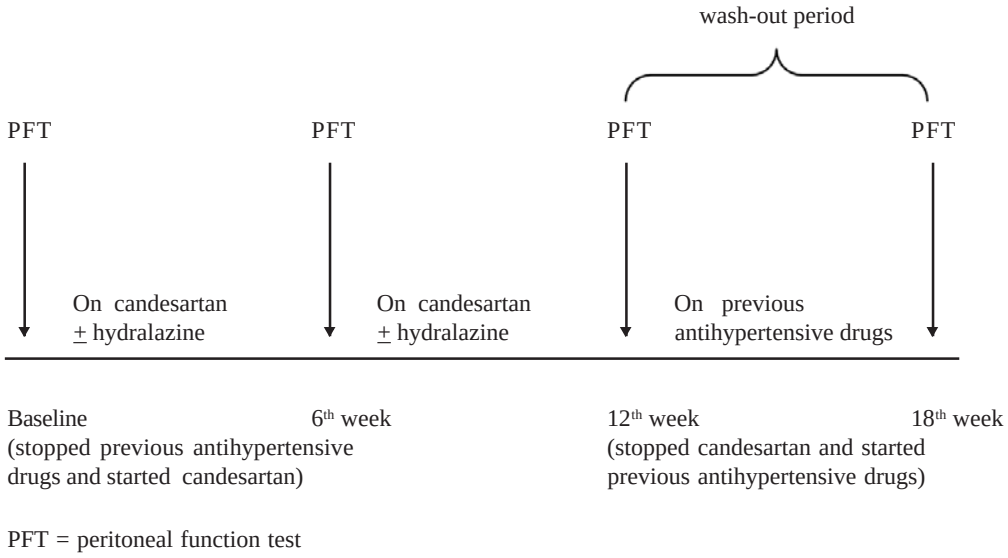


Fig. 1 Study protocol

formed as previously described by Pannekeet, et al[14]. In brief, the peritoneal cavity was rinsed with two liters of fresh 1.5% dialysis solution before installation of the test solution into the abdomen. The rinsed solution was completely drained over 20 min in the sitting position, mixed the drainage dialysate by inverting a bag three times, and dialysate samples were then collected. A blood sample was obtained at the end of the drainage. Two liters of 1.5% dialysis solution was infused in portions of 400 mL per 2 min over a period of 10 min. For better mixing of the residual peritoneal volume and the new infused solution, the patients were leaned supinely during the infusion period and were rolled side by side after infusing each 400 mL. At the completion of infusion (0-dwell time), exactly 10 min after the start of infusion, 200 mL of solution was drained into the bag, mixed by inverting the bag three times, 10 mL aliquot of dialysate was taken and the remaining 190 mL was re-infused. The patient ambled during the dwell period. After a 4-hour dwell time, the dialysate was drained over 20 min while the patient was sitting. The total volume was measured and a sample was taken. The total time of the exchange was 270 min. A blood sample was obtained at the end of drainage. A sample of dialysate was taken from the post test rinsed bag to be infused, and two liters of fresh solution were infused over 10 min with the same technique as for the test solution exchange, immediately drained over 20 min. in the sitting position.

The blood and dialysate samples were assessed by routine standard measurements for the values of urea, creatinine, urate, glucose, and potassium. The levels of albumin were determined by Bromo Cresol Green (BCG) method. To avoid the influence of globulin, the measuring process of albumin was performed within 2-3 minutes after BCG was added. The values of β_2-microglobulin (β_2-M) were quantified by COBAS CORE β_2M EIA (Roche Diagnostics GmbH, Mannheim, Germany).

The transports of low molecular weight (LMW) solutes, including creatinine and urate, were expressed as mass transfer area coefficients (MTAC). The MTACs of creatinine and urate were calculated according to the model of Waniewski, with a correction factor for plasma water[15].

Because the concentrations of urea in dialysate were equal to plasma in 4 patients, the MTAC of urea could not be calculated. Thus, dialysate/plasma ratio was utilized in assessing the peritoneal transport of urea[16].

Peritoneal clearances of β_2-microglobulin were calculated after four-hour dwelling using the equation: Cl (ml/min) = DxV/Pxt, where Cl is the clearance, D is the dialysate concentration, V is the dialysate volume, P is the plasma concentration, and t is the dwell time[16].

Ultrafiltration (UF) was assessed by direct measurement of the difference between the drained and infused dialysate volume.

Statistical analysis

All the data presented in the present study are expressed as mean $\pm$ SE of 7 patients. Statistical analysis was determined by repeated measured ANOVA (analysis of variance). Statistical significance was defined when p-value was < 0.05.

Results

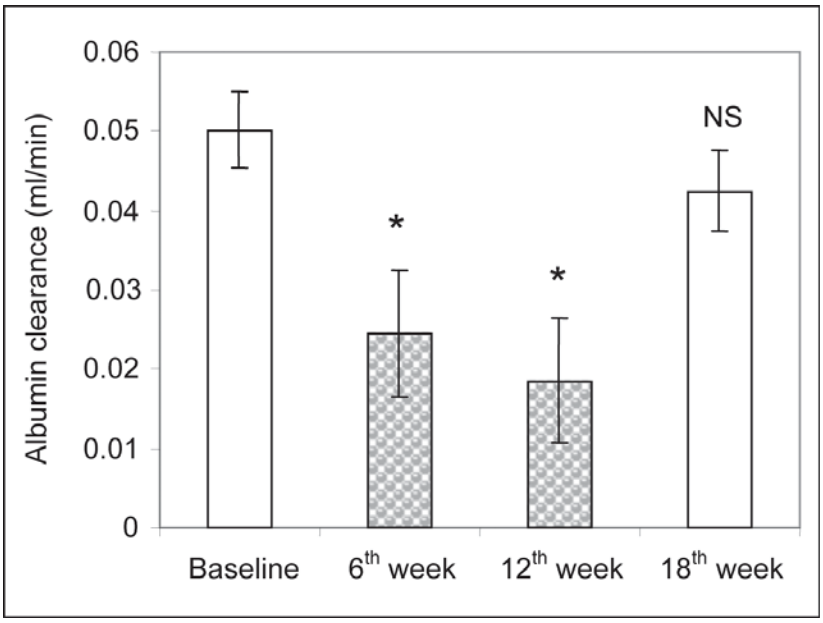
Basic patient characteristics

Seven stable CAPD patients (5 males, 2 females; mean age 62.0 $\pm$ 3.6 years, ranged 45-73 years; mean body weight 62.6 $\pm$ 4.5 kg., ranged 48.0-75.2 kg) were recruited in the present study. The causes of ESRD were diabetic nephropathy (43%), hypertension (29%), and unknown (28%). The mean duration of peritoneal dialysis treatment prior to the present study was 42.6 $\pm$ 11.3 months. Peritoneal function tests revealed "low average" results in all patients. Baseline serum biochemistry data comprised: blood urea nitrogen = 46 $\pm$ 5.6 mg/dL, creatinine = 11.1 $\pm$ 1.3 mg/dL, and albumin = 3.7 $\pm$ 0.2 g/dL. Hematocrit was 37.5 $\pm$ 1.5%. All patients were anuric with the mean 24-hour urine volume of 16.9 $\pm$ 8.2 mL. Mean creatinine clearance, determined by 24-hour collection, was 0.6 $\pm$ 0.4 mL/minute. No statistically significant differences were noted in the parametric data between males and females.

Blood pressure data

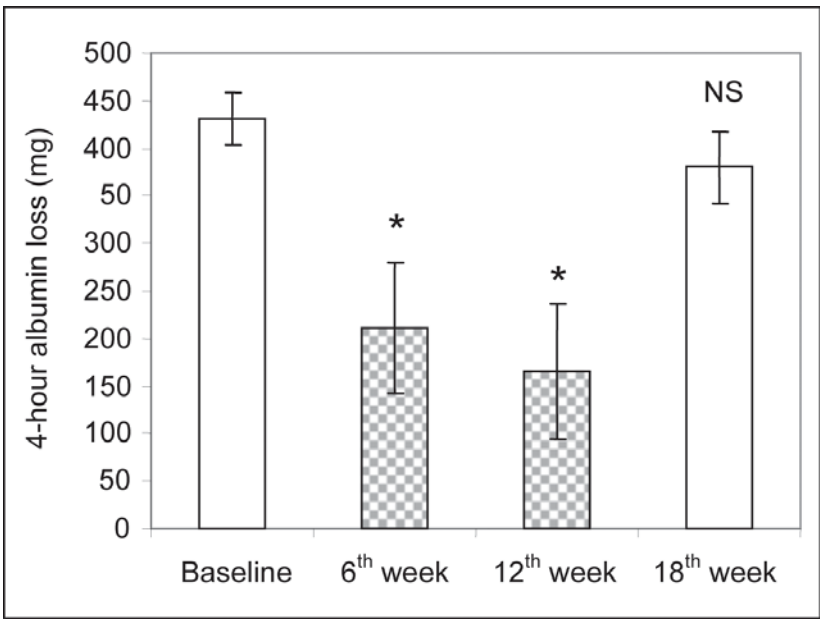
Previous antihypertensive drugs used in the participating patients were calcium-channel blocker (100%), alpha blocker (43%), and beta blocker (57%). During the experimental period, the use of candesartan at the dose of 8 and 16 mg/day achieved the target blood pressure below 140/90 mmHg in 2 and 5 patients, respectively. Hydralazine at the dose of 50 mg/day had been supplemented in one patient who was treated with 16 mg/day of candesartan.

The value of mean arterial pressure (MAP) during the 4 periods was 108 $\pm$ 7 mmHg at baseline, 107 $\pm$ 10 mmHg at 6-week candesartan, 113 $\pm$ 5 mmHg at 12-week candesartan, and 109 $\pm$ 7 mmHg at 6-week candesartan withdrawal. No significant differences were observed among the 4 values of MAP.



baseline = before start candesartan, 6th week = 6 weeks after candesartan use, 12th week = 12 weeks after candesartan use, 18th week = 6 weeks after candesartan withdrawal, *p < 0.05, when compared with baseline and 18th week, NS = non significant when compared with baseline

Fig. 2 Albumin clearance



baseline = before start candesartan, 6th week = 6 weeks after candesartan use, 12th week = 12 weeks after candesartan use, 18th week = 6 weeks after candesartan withdrawal, * p < 0.05, when compared with baseline and 18th week, NS = non significant when compared with baseline

Fig. 3 4-hour albumin loss

Table 1. The mean of MAP, peritoneal transports, net ultrafiltration , serum potassium level, and dose of recombinant human erythropoietin

	Baseline	6th week	12th week	18th week
MAP (mmHg)	107.9 ± 6.5	106.7 ±10.1	113.3 ±4.8	108.6 ± 6.6
Net ultrafiltration (mL)	142.9 ± 60.9	197.1 ± 137.4	154.3 ± 69.2	162.9 ± 57.1
D/P urea	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1
MTAC creatinine	7.4 ± 1.1	8.0 ± 1.4	7.4 ± 0.8	7.5 ± 0.8
MTAC urate	6.3 ± 0.6	7.2 ± 1.0	6.4 ± 0.8	7.5 ± 1.1
β_2-microglobulin clearance (x 10^{-3} ml/min)	705.4 ± 76.0	602.4 ± 88.5	685.6 ± 89.4	662.5 ± 70.0
Glucose absorption rate (%)	41.5 ± 4.1	43.8 ± 2.4	43.5 ± 3.8	39.0 ± 1.8
Serum albumin (g/dL)	3.7 ± 0.2	3.6 ± 0.2	3.6 ± 0.1	3.8 ± 0.1
Serum potassium level (mEq/L)	3.8 ± 0.2	3.8 ± 0.3	4.0 ± 0.3	4.2 ± 0.4
EPO dose (unit/week)	5,014 ± 1,714	5,014 ± 1,714	4,985 ± 1,670	4,628 ± 1,640

Abbreviations: MAP = mean arterial pressure, D/P urea = dialysate urea to plasma urea ratio, EPO = recombinant human erythropoietin

Peritoneal-membrane transport data

As detailed in Table 1, among the 4 experimental periods, there were no significant differences in the following parameters: net ultrafiltration, D/P urea, MTAC urate, β_2-microglobulin clearance, and glucose absorption rate.

Following 6-week candesartan treatment, there were significant decreases in the values of peritoneal albumin clearance ($p < 0.05$) and 4-hour albumin loss ($p < 0.05$) (Fig. 2, 3). The reduced values of both parameters remained to be observed following 12-week candesartan therapy ($p < 0.05$). After 12-week candesartan withdrawal, the values of both parameters returned to the baseline levels (Fig. 2, 3).

Despite the reduction in the peritoneal loss of albumin during candesartan treatment, serum albumin levels were not significantly different among the 4 periods (Table 1).

Adverse effects

During treatment with candesartan, no adverse effects including hyperkalemia or increasing the dosage of erythropoietin had been found (Table 1).

Discussion

The results in the present study have demonstrated that in hypertensive CAPD patients 1) candesartan at the dose of 8-16 mg/day could effectively control blood pressure below 140/90 mmHg, 2) candesartan could reduce peritoneal albumin clearance and 4-hour albumin loss, 3) candesartan did not alter other peritoneal membrane transports, and 4) candesartan did not cause serious adverse effects including hyperkalemia and increased erythropoietin dosage.

Increasing evidence has established the antihypertensive effect and cardiovascular protective role of ACEI and ARB in ESRD patients receiving renal replacement therapy[4,5]. In CAPD patients, various antihypertensive agents could alter peritoneal membrane transports[1]. Sodium nitroprusside, calcium channel blocker, diazoxide, and minoxidil increases diffusion while beta blocker affect convection[1].

Studies demonstrate that the effects of ACEI on peritoneal membrane transports remain inconclusive[6-12]. In Sprague Dawley (SD) rats, Lal et al revealed that captopril administered intraperitoneally at the dose of 75 mg/exchange could cause hypotension with increased urea clearance and dialysate protein loss but enhanced glucose absorption[6]. No UF changes were observed. Captopril at lower doses unalters peritoneal transports. However, in a recent study by Kumano et al, captopril treated intraperitoneally in SD rats, enhanced peritoneal clearances of urea, glucose, protein, and peritoneal net fluid absorption rate in a dose-dependent fashion[10]. Coronel et al found that oral captopril 50 mg/day orally administered to 12 hypertensive CAPD patients with diabetes for 1 month reduced peritoneal albumin loss without significant change in systemic blood pressure[7]. In 9 CAPD patients, following 2 weeks of oral enalapril 20 mg twice daily, Favazza et al demonstrated a significant decrease in mean arterial pressure in association with increases in creatinine and β_2-microglobulin clearances but a decrease in glucose absorption[8]. On the contrary, Ripley et al illustrated that, in 16 CAPD patients, both oral enalapril and intraperitoneal enalaprilat administrations for one week exerted antihypertensive effect but caused no changes in peritoneal transport characteristics[9].

There are sparse data regarding the effect of ARB on peritoneal membrane transports especially in CAPD patients. In Wistar Kyoto rats, intraperitoneal valsartan, 10 mg/kg/day, suppressed expression of aquaporins 1 and 4, accompanied by loss of ultrafiltration volume[17]. The effect of valsartan on peritoneal membrane transports was not examined in the mentioned study. Recently, Ishida et al demonstrated that benazepril, an ACEI, and valsartan, an ARB, could increase peritoneal solute clearances in hypertensive dogs with mild renal insufficiency[11]. Duman et al showed that lisinopril, an ACEI, as well as CS866, an ARB, could attenuate the impairment in peritoneal solute and ultrafiltration transports occurring in uremic rats with peritoneal sclerosis[12].

In the present study, oral candesartan, 8-16 mg/day, provided salutary antihypertensive effect while it did not alter peritoneal solute and water transports (Table 1). The peritoneal transports results in CAPD patients shown in the present study, thus, were different from previous animal studies[11,12,17]. The underlying mechanism of this disparity is still unknown and needs further study.

Of interest, loss of albumin across the peritoneal membrane has a significant effect on serum albumin concentrations in CAPD patients on long-term peritoneal dialysis[18,19]. Serum albumin levels are strongly correlated with 8-hour peritoneal mass transfer, albumin clearance, and 8-hour effluent concentrations of protein. As such, if peritoneal albumin loss is reduced, there would be improvement in serum albumin levels, nutritional status, and morbidity as well as mortality of the CAPD patients.

In the present study, oral candesartan could significantly reduce albumin clearance and 4-hour albumin loss during the 12 week duration of treatment (Fig. 2, 3). The effect of candesartan in decreasing peritoneal albumin loss observed in the present study is comparable to ACEI reported by Coronel et al[7]. The values of serum albumin levels, however, were not determined in the latter study. Also, the mechanism of ACEI in attenuating peritoneal albumin loss had not been explored. In the present study, no significant change in serum albumin concentrations was noted following 12 weeks of candesartan treatment (Table 1). The duration of treatment might not be long enough to observe alteration in serum albumin levels.

The mechanism underlying the beneficial effect of candesartan on peritoneal albumin loss is still not yet established. This would be functional changes rather than permanent structural alterations. This is because the amount of peritoneal albumin loss was increased to the baseline levels after candesartan withdrawal (Fig. 2, 3).

Candesartan did not alter serum potassium levels and the dose of recombinant human erythropoietin in treatment with CAPD is in agreement with previous studies in other modalities of renal replacement therapy[20-22].

In conclusion, candesartan treatment in hypertensive CAPD patients could provide salutary antihypertensive effect and could reduce peritoneal loss of albumin, but it unalters other solute transports and net UF. The cardiovascular and nutritional benefit of candesartan would improve the survival and quality of life in CAPD patients.

Acknowledgement

The authors wish to thank Ms. Tipwan Tongthamrongrat for her typographical assistance.

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ผลของยาแองจิโอเทนซินทูรีเซฟเตอร์บล็อคเกอร์ต่อการขนส่งผ่านผนังเยื่อบุช่องท้องในการล้างไตทางหน้าท้องชนิดถาวร

วีระศักดิ์ เจียรสุจิตวิมล, สมชาย เอี่ยมอ่อง, เถลิงศักดิ์ กาญจนบุษย์, อาภา วรรธนะวหะ, พงศ์ศักดิ์ พันธุ์สิน

วัตถุประสงค์: *ศึกษาผลของยาแองจิโอเทนซินทูรีเซฟเตอร์บล็อคเกอร์ ซึ่งเป็นยารักษาความดันโลหิตต่อการขนส่งผ่านผนังเยื่อบุช่องท้องในการล้างไตทางหน้าท้องชนิดถาวร*

วัสดุและวิธีการ: *ทำการศึกษาในผู้ป่วย 7 รายโดยงดยาความดันโลหิตเดิม และให้ยาแคนดีซาร์ทาน ขนาด 8-16 มก./วัน เพื่อควบคุมความดันโลหิตต่ำกว่า 140/90 มม.ปรอท อาจให้ยาไฮดราลาซีนเสริม ภายหลัง 12 สัปดาห์ ผู้ป่วยจะได้รับยาความดันโลหิตเดิมเป็นเวลา 6 สัปดาห์ ทำการศึกษาหน้าที่เยื่อบุช่องท้อง ณ จุดเริ่มต้น ที่เวลา 6 และ 12 สัปดาห์หลังรับประทานยาแคนดีซาร์ทานและที่ 6 สัปดาห์หลังหยุดยาแคนดีซาร์ทาน*

ผลการศึกษา: *ระดับความดันโลหิตในทุกระยะไม่แตกต่างกันและบรรลุถึงเป้าหมายที่กำหนด ภายหลังการให้แคนดีซาร์ทานพบว่าการขจัดอัลบูมินลดลงและเพิ่มกลับขึ้นสู่ระดับเดิมหลังการหยุดแคนดีซาร์ทาน ไม่มีความแตกต่างในการขนส่งสารอื่น ๆ ไม่พบผลข้างเคียงจากการให้ยา*

สรุป: *ในผู้ป่วยล้างไตทางหน้าท้องชนิดถาวรที่มีความดันโลหิตสูงพบว่า แคนดีซาร์ทานมีประสิทธิภาพในการลดความดันโลหิตและลดการสูญเสียอัลบูมินผ่านทางเยื่อบุช่องท้องโดยไม่มีผลต่อการขนส่งของสารอื่น ๆ*