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CLINICAL INVESTIGATION

Effect of Intravascular Iodinated Contrast Media on Natural Course of End-Stage Renal Disease Progression in Hemodialysis Patients: A Prospective Study

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Abstract We evaluated the impact of intravascular iodinated contrast medium on residual diuresis in hemodialyzed patients. Two groups of clinically stable hemodialyzed patients with residual diuresis minimally 500 ml of urine per day were studied. The patients from the first group were given iso-osmolal contrast agent iodixanol (Visipaque, GE Healthcare, United Kingdom) in concentration of iodine 320 mg/ml with osmolality 290 mOsm/kg of water during the endovascular procedure. The second control group was followed without contrast medium administered. Residual diuresis and residual renal excretory capacity expressed as 24-h calculated creatinine clearance were evaluated in the both groups after 6 months. The evaluated group included 42 patients who were given 99.3 ml of iodixanol in average (range, 60–180 ml). The control group included 45 patients. There was no statistically significant difference found between both groups in daily volume of urine ($P = 0.855$) and calculated clearance of creatinine ($P = 0.573$). We can conclude that residual diuresis is not significantly influenced by intravascular administration of iso-osmolal iodinated contrast agent (iodixanol) in range of volume from 60 to 180 ml in comparison to natural course of urinary output and residual renal function during end-stage renal disease. This result can help the nephrologist to decide which imaging method/contrast medium to use in dialyzed patients in current practice.

Keywords Hemodialysis · Iodixanol · Residual diuresis · Residual renal function

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Introduction

The number of patients with end-stage renal disease treated by maintenance hemodialysis is growing as a result of advances in medicine and longer survival of general population. There is also an increasing number of diagnostic and even therapeutic endovascular procedures requiring administration of iodinated contrast agent (ICA) in these patients with disease.

Contrast-induced nephropathy (CIN) is a term applied to acute kidney injury associated with intravascular administration of ICAs. Preexisting nephropathy is considered to be the main risk factor for development of CIN [1, 2]. In

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addition, recent data suggest that a combination of factors, including renal insufficiency and exposure to gadolinium-based contrast agents, may play a role in the development of potentially disabling or even fatal nephrogenic systemic fibrosis [3–8].

Residual renal function (RRF) and residual diuresis are considered to be an important predictor of outcome in patient population on peritoneal dialysis, but it is also vitally important in hemodialysis (HD) patients [9, 10].

Data about the impact of intravascular application of ICAs on natural course of the function of remaining kidney function in end-stage renal disease patients treated by maintenance HD are scarce. Only few authors have studied the consequences of intravascular ICA in dialysis patients. There are only a few studies on the impact of intravascular ICA on residual diuresis in patients in peritoneal dialysis [11–13] or on general tolerance but not RRF of intravascular ICA in HD patients [14–16]. To our knowledge, there is no available study that evaluates the long-term effect of intravascular ICA on residual renal elimination capacity in maintenance HD patients.

The purpose of our study was to follow up chronically HD patients with residual diuresis of more than 500 ml of urine per day after intravascular administration of at least 60 ml of iso-osmolal ICA during an endovascular interventional procedure and to compare the natural course of kidney function loss in these patients with HD controls.

Materials and Methods

The study lasted 6 months; two groups of regularly hemodialyzed patients were observed on a monthly basis. All patients were dialyzed three times a week for a period longer than 6 months. Only clinically stable patients with no serious concomitant disease and who survived for a 6-month follow-up period with an unchanged dialysis strategy were evaluated. At the start of the observation period, all the patients had residual diuresis of more than 500 ml/day (this value corresponds to the threshold oligoric status, 20 ml/h), and they were dialyzed for at least 3 months.

The evaluated group (group A) prospectively included patients who were provided intravascular nonionic dimer iodixanol (Visipaque, GE Healthcare, UK) in iodine concentration of 320 mg/ml and osmolality 290 mOsm/kg H_2O. In all patients, the administered amount was more than 60 ml. The average quantity of iodixanol was 99.3 ml (range, 60–180 ml). When calculated to kilogram of optimal body weight, the average dose was 1.56 ml of iodixanol per kilogram of body weight (range, 0.93–2.93 ml/kg) The endovascular interventions were indicated on a clinical basis and included transluminal angioplasty of stenosed HD vascular access (n = 33), urgent recanalization of thrombosed vascular dialysis access (n = 6), or angiography and transluminal angioplasty for critical limb ischemia (n = 3).

According to the center's clinical policy, all patients had HD scheduled after the radiologic intervention procedure on the same day (with 3–5 h delay). Patients were hydrated in their usual way before and after this particular HD, with ultrafiltration to their target "dry" weight, which was stable in the long term. No specific nephroprotective measures were used. For the study, clinical and laboratory records were reviewed for the 3 months before and 3 months after the endovascular intervention with iodine contrast media application.

The control (group B) included matched clinically stable hemodialyzed patients in terms of residual diuresis (at least 500 ml of urine per day) but without any ICA administered. Monthly clinical and laboratory records were assessed for a period of 6 consecutive months.

HD procedures in all patients were performed according to standardized clinical practice without any change during the whole study period (dialysis monitor Fresenius 4008S and 4008B, polysulphone high-flux dialyzers 1.6–1.8 m^2, bicarbonate dialysis solution 500 ml/min, dialysate temperature 36°C, mean blood flow approximately 300 ml/min, target spKt/V at least 1.4, mean ultrafiltration 2500 ml with constant ultrafiltration rate). Adequacy of ultrafiltration was controlled with a Crit-Line device, confirming blood volume decrease maximally 10% during HD.

The patients were followed according to the standardized clinical and laboratory protocol, registering daily urinary output once a month, just before a monthly laboratory checkup. Our study included only values that were based on routine monitoring during regular HD in the first week of the month with a stable interdialysis interval. The patients were carefully and repeatedly instructed to provide correct urine collection according European Best Practice Guidelines for Haemodialysis [17]. The patients collected the urine in a collecting measuring bottle. The urine collected within last 24 h before monitored dialysis was brought to the dialysis session. Blood samples were taken immediately before HD from an inserted HD needle and immediately assessed. On the basis of availability of urine sampling and urine and serum creatinine values, we calculated creatinine clearance according to a generally known formula. This clearance was normalized to 1.73 m^2 body surface area and expressed in units of ml/min/1.73 m^2.

Calculated values of creatinine clearance (expressed as calculated creatinine clearance based on urine sampling for 24 h during the last interdialysis day) did not exactly represent real glomerular filtration. Because both the method of evaluation and the interdialysis intervals were unchanged, our results reflect residual renal excretory capacity

(RREC), and the values from each month follow the long-term dynamics of function changes in the remaining still-working glomerules.

Our aim was not to evaluate the immediate effect of contrast medium to RRF during the several days after application. Rather, we wanted to evaluate its long-term clinical effect. This is why we compared the volume of daily diuresis and RREC 3 months before and 3 months after ICA administration. The average values obtained 3 months before and 3 months after ICA administration were used for evaluation to eliminate the variability of measurements over time. The values measured during HD, immediately after ICA administration, were not registered.

We calculated mean values; median, minimal, and maximal values; and interquartile range (i.e., range from 25th percentile to the 75th percentile). We used the two-sample *t*-test and nonparametric Kolmogorov-Smirnov test to evaluate differences between groups and the nonparametric Wilcoxon test to evaluate changes. Fisher's exact test was used for evaluating differences between sexes. Level of significance was $\alpha = 0.05$.

Results

At the beginning of the prospective study, 95 patients were recruited (46 in evaluation group A, and 49 patients in control group B). During the 6-month follow-up period, four patients from group A (8.6%) and four patients (8.2%) from group B died. For this reason, only 87 patients were evaluated (42 in group A and 45 in group B).

Group A prospectively included 42 patients (21 men and 21 women) with an average age of 64.4 years (range, 41–84 years) from 2004 to 2007. The kidney disease that caused chronic renal failure was diabetic glomerulosclerosis in 18 cases, tubulointerstitial nephritis in 9 cases, chronic glomerulonephritis in 6 cases, vascular nephrosclerosis in 3 cases, polycystic kidney disease in 2 cases, and lipoid nephritis and analgesic drug-induced nephropathy in 1 case each.

The control group included 45 patients (27 men, 18 women) with an average age of 64.4 years (range, 29–87 years) during the same period. The cause of chronic renal failure was diabetic glomerulosclerosis in 13 cases, tubulointerstitial nephritis in 17 cases, vascular nephrosclerosis in 8 cases, polycystic kidney disease in 4 cases, chronic glomerulonephritis in 2 cases, and lupoid nephritis in 1 case

Both groups A and B were found to be comparable for sex ($P = 0.285$) and age ($P = 0.999$). The course of daily diuresis for contrast media group A and control group B during 6 months is shown in Table 1. Data of RREC, expressed as calculated creatinine clearance based on urine sampling for 24 h during the last interdialysis day, are provided in Table 2. The medians of daily diuresis and RREC of both group A and group B dropped between the first and last measurements (in treatment group A from 1500 to 1225 ml and from 7.44 to 6.0 ml/min/1.73 m^2; in control group B from 1400 to 900 ml and from 5.82 to 3.12 ml/min/1.73 m^2). Average 3-month daily urine volume and RREC are shown in Tables 3 and 4. The average daily urine volume and RREC decreased significantly during the evaluated period (in treatment group A from 1400 ml to 1275 ml and from 6.84 to 5.76 ml/min/1.73 m^2; in control group B from 1250 to 1000 ml and from 6.18 to 3.96 ml/min/1.73 m^2).

The statistical significance level in group A was $P = 0.00112$ for urine volume and $P = 0.00088$ for RREC; in group B, the significance level was $P = 0.000276$ for urine volume and $P = 0.0345$ for RREC. No statistically significant difference was found between groups either for urine volume ($P = 0.855$) or RREC ($P = 0.573$).

Discussion

RRF and residual diuresis are considered to be an important predictor of outcome in patients who undergo peritoneal dialysis, but is also vitally important in HD patients [9, 10]. Every effort should be made to protect existing renal

Table 1 Daily urine volumes (V, ml)

Volume	Group A, contrast media			Group B, control		
	Median	Range	Quartile (25th–75th percentile)	Median	Range	Quartile (25th–75th percentile)
1	1500	500–4500	987–2000	1400	500–3500	1000–1750
2	1475	500–3220	987–2000	1200	500–2500	960–1500
3	1500	500–3800	937–2000	1100	500–2100	875–1500
4	1150	500–3500	850–1925	1000	500–2100	780–1575
5	1300	500–2420	700–1775	1000	500–2300	725–1400
6	1225	500–2100	737–1662	900	500–2400	700–1200

Table 2 Residual renal excretory capacity (RREC), expressed as calculated creatinine clearance based on urine sampling for 24 h during the last interdialysis day (ml/min/1.73 m^2)

RREC	Group A, contrast media			Group B, control		
	Median	Range	Quartile (25th–75th percentile)	Median	Range	Quartile (25th–75th percentile)
1	7.44	0.36–54.6	3.31–12.8	5.82	1.68–19.32	2.82–9.91
2	5.28	0.120–71.6	3.62–11.8	3.62	1.08–15.6	2.22–8.58
3	7.02	0.061–42	3.13–11.6	3.91	1.44–19.9	2.40–7.86
4	4.98	0.063–19.5	3.61–8.58	4.56	1.26–13.7	2.52–7.74
5	5.28	0.062–35.4	3.12–8.71	4.08	1.44–13.0	2.22–8.22
6	6.60	0.120–23.4	3.72–10.1	3.12	1.02–14.6	2.34–5.04

Table 3 Average daily urine volumes (ml)

Variable	Group A, contrast media			Group B, control		
	Median	Range	Quartile (25th–75th percentile)	Median	Range	Quartile (25th–75th percentile)
Volume average 3 months before	1400	550–3667	1052–1913	1250	500–2500	978–1533
Volume average 3 months after	1275	500–2267	733–1867	1000	500–2133	842–1400
Volume difference	−150	−1692–733	−367–0	−167	−1000–368	−433–17

Table 4 Average residual renal excretory capacity (RREC) (ml/min/1.73 m^2)

Variable	Group A, contrast media			Group B, control		
	Median	Range	Quartile (25th–75th percentile)	Median	Range	Quartile (25th–75th percentile)
RREC average 3 months before	6.84	0.18–42.6	4.02–13.2	6.18	1.80–14.8	2.58–9.0
RREC average 3 months after	5.76	0.61–35.4	3.54–8.41	3.96	1.86–13.1	2.76–7.08
RREC difference	−0.90	−38.3–30.1	−4.14–0.018	−1.20	−5.34–2.88	−3.06–0.60

function in HD patients, especially if daily urine volume exceeds 100 ml [18]. The presence of RRF was associated with a lower mortality and better survival in HD patients [10, 19]. The RRF in HD patients provides a beneficial effect on nutritional status [20]. The malnutrition in dialyzed patients is multifactorial (disturbances of protein and glucose metabolism, hormonal dysbalance, increased basal metabolism, anorexia). Preserved RRF helps to improve appetite and significantly contributes to nutrition, which can be assessed by levels of serum albumin or transferrin. The volume of urine produced each day allows more fluid intake, reducing the otherwise larger fluctuations in body fluid volumes between dialysis treatments that contribute to volume overload syndromes, hypertension, and cardiac hypertrophy [18, 21]. The other published studies reported lower potassium and uric acid level, better phosphate balance, lower beta-2-microglobulin levels, and higher hemoglobin and endogenous erythropoietin levels [22–24]. RRF allows for reduction in the duration of HD sessions and reduces fluid restrictions [9]. In patients who are candidates for kidney transplantation, residual diuresis retains the function of ureters. The psychological aspect of treatment is also important. Patients with residual diuresis feel much better.

On the negative side, preserving RRF may prolong protein malnutrition and worsen hypoalbuminemia in highly proteinuric patients [25]. The "artificial kidney" is only partially replacing the native kidneys. The elimination through the dialysis membrane is only temporary and is restricted only to the time of dialysis—that is, several hours per week. Any contribution of the native kidneys is therefore of importance in clearing the blood.

Measurement of RRF is a well-established method in continual outpatient peritoneal dialysis but is not a validated and universally accepted method for more complicated measuring renal function in HD. HD patients are not in a steady state because of the intermittent nature of the dialysis therapies [17, 26]. Measurement of volume of

residual daily output of urine seems to be the simplest way for approximation of RRFs. In dialysis patients, glomerular filtration rate should be estimated as the mean urea and creatinine clearance of urea using urine collections [17]. Without collecting daily output of urine, it is possible to evaluate RRF by means of compounds such as inulin or iohexol [9, 27] or to use the formula for modification of diet in renal disease, but this formula has not been well validated in patients with a glomerular filtration rate of <10 ml/min [28]. Another recent and accurate method for estimation of glomerular filtration rate uses the serum concentration of cystatin C [29].

The ICA is quickly excreted by glomerular filtration without tubular secretion or reabsorption in patients with normal kidney functions. Thus, 50% of the administered ICA is excreted in urine in 2 h. The excretion is significantly slower in patients with impaired renal functions (50% of ICA is excreted in 16 to 84 h) [30]. The ICA is mostly excreted by liver in bile in patients with kidney failure, and the ICA is retained for long time.

Two groups of dialyzed patients can be defined according to the necessity of intravascular ICA administration. The first group consists of anuric patients. In these patients, ICA cannot do more damage to the kidneys, and only hemodynamic effect should be considered [31]. The second group includes patients with RRF. In these patients, the intravascular ICA can increase the load of remaining nephrons and extend the exposition of ICA in the tubules [2]. It is recommended that precautions similar to the prevention of contrast nephropathy be used, as in patients with impaired renal function—that is, hydration [32] and use of isotonic ICA [33].

HD, even performed immediately after intravascular administration of ICA, cannot protect the kidneys against contrast nephropathy even though HD can effectively eliminate all ICA from the blood [2, 16, 30, 34, 35]. Insufficiency of HD in this regard is explained by the fast nephrotoxic effect of ICA after its administration.

Osmolality plays significant role in inducing contrast nephropathy. High-osmolality ICA (osmolality more than 1500 mOsm/kg H_2O) has a higher incidence of contrast nephropathy than low-osmolality ICA (less than 915 mOsm/kg H_2O). Theoretically, iso-osmolal ICA (290 mOsm/kg H_2O) should be less nephrotoxic. The significant benefit of iso-osmolal ICA over low-osmolal ICA was proved in the NEPHRIC study [33]. This was not proved to be as significant in other studies [36–39].

The risk of CIN increases with increasing dose of ICA. In patients with serum creatinine above 300 μmol/l is a risk factor a dose more 100 ml of ICA (ICA in concentration of iodine 300 mg/ml) [1]. The amount of contrast medium considered to be safe for dialyzed patients is not known.

We have found only three studies reporting on patients on peritoneal dialysis that evaluate the influence of intravascular ICA on RRF. No significant acceleration of RRF decline was observed in a group of 36 patients who were well hydrated before ICA administration [12]. A second report of 10 patients on peritoneal dialysis who were administered 107.5 ml of iopromide drew the same conclusion [11]. In a third study [13], only one patient on peritoneal dialysis became permanently anuric after coronarography with average dose of 74 ml of low osmolal ICA; the remaining 23 patients with complete postangiography assessment had the same gradual rate of loss of RRF as the control group.

Our study has some drawbacks: we did not use exact calculations for creatinine clearance because these data are based on longer times of urine sampling, and this was not the practice in our dialysis units. Therefore, our data cannot be considered as fully representative in this regard. On the other hand, our aim was not to precisely assess the renal function, but rather to follow its changes over 6 months. Furthermore, distribution of the primary diseases responsible for renal failure was not equal in both groups. There were more patients with chronic glomerulonephritis in the control group.

In summary, our data do not indicate long-term negative effects of intravascular administration of iodixanol (320 mg/ml in average volume of 99.3 ml) on the natural course of urinary output in clinically stable HD patients.

Preexisting nephropathy is considered to be the main risk factor for development of CIN [1, 2]. According to our results, the risk of RRF impairment in HD patients after ICA application is relatively small. Our findings may influence the decision of whether to use iodine-enhanced imaging (computed tomography or catheter angiography), thus only minimally influencing RRF, or gadolinium-enhanced magnetic resonance imaging, with its potential risk of nephrogenic systemic fibrosis.

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