

Abstract Title: Furosemide- induced diuresis with matched hydration compared to standard hydration for contrast- induced nephropathy prevention: preliminary results of the MYTHOS trial

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Topic: Prevention of Contrast Induced Nephropathy (CIN)

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Abstract: **Background:** Contrast-induced nephropathy (CIN) is a frequent cause of hospital-acquired acute kidney injury and is associated with prolongation of hospital stay and increased short and long-term mortality. We investigated the effect of a new CIN preventive strategy based on a dedicated device (RenalGuard; PLC Medical System Inc., USA). The system is capable of delivering i.v. saline solution to a patient in an amount matched to the volume of urine produced after an i.v. bolus of furosemide. The aim of the study was to evaluate if furosemide-induced high-volume diuresis with concurrent maintenance of intravascular volume may prevent CIN.

Methods: To date, 105 chronic kidney disease (CKD) patients (eGFR<60 ml/min/1.73 m²) undergoing elective or urgent percutaneous coronary interventions (PCI) were enrolled in the MYTHOS trial. They were randomized to matched hydration (RenalGuard group, n=48) or to i.v. isotonic saline hydration (control group; n=57) at a rate of 1 ml/kg/hour for 12 hours before and after PCI. Matched fluid replacement was started approximately 90 minutes pre-PCI, maintained during catheterization, and for up to 4 hours afterwards. Patients were given an initial i.v. bolus of 250 ml of normal saline over 30 minutes and then an i.v. bolus of furosemide (0.5 mg/kg). The RenalGuard measures the patient's urine volume and then automatically adjusts the infusion pump rate to precisely replace the measured urine output in real time. When a significant (>300 ml/hour) urine output rate was obtained, patients underwent PCI. CIN was defined as a ≥ 0.5 mg/dl or $\geq 25\%$ rise in serum creatinine over baseline. Iomeron was used in all patients.

Results: Overall, the mean eGFR was 39 ± 10 ml/min/1.73 m². The mean contrast volume was 197 ± 115 ml in RenalGuard group and 214 ± 115 ml in controls (P=ns). In RenalGuard group, patients had a urine output mean increase of 827 ± 354 ml/hr. CIN occurred in 2 patients (4.2%) of the RenalGuard group and in 8 (14.0%) in the control group (P=0.08). There were 3 (6.3%) in-hospital adverse clinical events in the RenalGuard group and 8 (17.5%) in the control group.

Conclusions: These preliminary results indicate that furosemide-induced high urine output with maintenance of intravascular volume through matched hydration can be safely obtained with the RenalGuard system and seems to reduce the risk of CIN in CKD patients undergoing PCI. A trend toward a reduction of in-hospital adverse clinical events was also observed in RenalGuard-treated patients.