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Effects of Magnesium Sulfate on Supraceliac Aortic Unclamping in Experimental Dogs

Intravascular administration of magnesium (Mg) causes vasodilation and increases renal blood flow. The aim of this study was to investigate the renal effect of Mg following unclamping of the supraceliac aorta. Mongrels were divided into two groups, control (group C, n=7) and Mg group (group Mg, n=7). In group Mg, 30 mg/kg $MgSO_4$ was injected as a bolus immediately prior to unclamping the supraceliac aorta and thereafter as an infusion (10 mg/kg/hr). The group C received an equivalent volume of saline solution. Systemic hemodynamics, renal artery blood flow, renal cortical blood flow (RCBF), renal vascular resistance, and renal function were compared. Following the aortic unclamping, cardiac output and RCBF were less attenuated, and the systemic and renal vascular resistance was elevated to a lesser degree in the group Mg compared to the group C. There was no significant difference in the plasma renin activity, serum creatinine and Cystatin-C between the two groups. The present study shows that Mg infusion improves systemic hemodynamics and RCBF after aortic unclamping. However, we did not observe any improvement in renal function when Mg was administered after supraceliac aortic unclamping.

Key Words : *Aortic Aneurysm; Magnesium; Renal Circulation; Perfusion, Regional; Reperfusion Injury*

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INTRODUCTION

Aortic cross clamping for abdominal aortic surgery may cause renal dysfunction after surgery. Ischemia-reperfusion injury is the major mechanism for renal dysfunction during and following aortic surgery (1). Abdominal aortic cross clamping induces renal vasoconstriction with a subsequent fall in renal artery blood flow (RABF) and renal function. Calcium channel blocker can potentially prevent the vasoconstriction induced by the effect of endothelin on the renal vascular bed (2). Magnesium (Mg) serves as a calcium channel blocker in nature and modulates vascular smooth muscle contraction. This action is a result of the effect of ionized Mg on the energy-dependent cation pump (3-5).

Recently Mg has been explored for a number of potential indications in anesthetic practice (6-10). The major rationale for the use of Mg for cardiovascular homeostasis in the anesthetic management derives from its ability to inhibit the release of catecholamines, reducing the sensitivity of α-adrenergic receptors to catecholamines, and a direct vasodilator effect (7, 11). Intravascular administration of Mg causes vasodilation and increases RABF. We hypothesize that Mg might effectively augment renal perfusion, and prevent renal dysfunction following unclamping of the aorta. The purpose of our study was to investigate the effect of magnesium sulfate ($MgSO_4$) on renal perfusion, renal function, and central hemodynamics after supraceliac aortic unclamping in an animal model.

MATERIALS AND METHODS

This investigation was performed under a protocol approved by the Institutional Animal Investigation Committee. A total of 14 mongrels of either sex, weighing 20-25 kg, were entered into the study. They were divided into two groups, control group (group C, n=7) and Mg group (group Mg, n=7). Anesthesia was induced by 20 mg/kg thiopental sodium and 0.1 mg/kg vecuronium bromide. After tracheal intubation, mechanical ventilation (Servo ventilator 900C, Siemens, Sweden) was done with 50% oxygen with air and 1-1.5% isoflurane at a tidal volume of 10-15 mL/kg with a respiratory rate of 10-12/min. Tidal volume and respiratory rate were adjusted to maintain the end-tidal carbon dioxide concentration between 40±5 mmHg (Capnomac, Datex-Ohmeda, Finland). Body temperature was maintained between 36°C and 38°C with

an electric heating pad. Electrocardiogram (lead II) was continuously monitored.

After inducing anesthesia, left brachial artery was dissected and a 21-gauge catheter (Angiocatheter, Becton Dickinson, Franklin Lakes, NJ, U.S.A.) was placed to monitor blood pressure continuously and to draw the blood samples. A 5-French balloon-directed thermal dilution pulmonary artery catheter (SP510H, Ohmeda, Singapore) was introduced through the right external jugular vein. The cardiac output (CO) was measured by thermodilution technique as an average of three consecutive cold bolus (0℃) injections.

A median laparotomy was performed and supraceliac abdominal aorta was exposed for occlusion and release. A thermal diffusion microprobe was implanted in the left renal cortex to measure real-time continuous renal cortical blood flow (RCBF) simultaneously. For microprobe implantation, the renal capsule was locally incised and a microprobe was inserted into the outer renal cortex (depth approximately 15 mm) at an angle of 45° to the renal surface (12). Minor bleeding after probe insertion always ceased spontaneously. Renal poles were not used for implantation. To prevent the microprobe displacement it was fixed to the renal capsule with a 5-0 Prolene suture. The microprobe was then connected to QFlow™500 perfusion measurement system (Thermal technologies Inc., Cambridge, MA, U.S.A.) to measure RCBF. We identified the appropriate location of the thermal diffusion microprobe by k-value. The k-value or tissue conductivity should not exceed 6.23 m·W/cm/℃ in this system (13). A k-value exceeding 6.23 m·W/cm/℃ indicates inappropriate microprobe positioning or other artifacts. The perfusion results obtained by perfusion measurement system (measurement interval 1/sec) were calculated as means of 1 min recordings. The left renal artery was dissected and an ultrasonic flow probe (HT107 medical volume flowmeter, Transonic systems Inc., Ithaca, NY, U.S.A.) was placed around the left renal artery to measure RABF. Right renal vein was dissected and 24-gauge catheters (Angiocatheter, Becton Dickinson) were placed to draw the blood samples.

On completion of the surgical preparation, the fluid was continuously administered with balanced salt solution to achieve a pulmonary artery wedge pressure (PAWP) of 8-12 mmHg. Adequate hydration was maintained throughout the experiment with balanced salt solution and colloid in a 3:1 ratio.

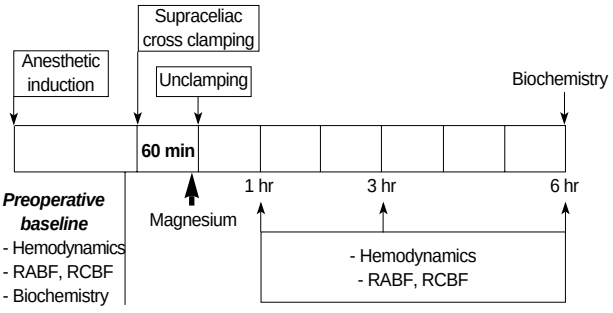

Fig. 1. Schematic diagram of the experimental protocol and data collection. RABF, renal artery blood flow; RCBF, renal cortical blood flow. Biochemistry include plasma renin activity, serum creatinine, Cystatin-C, and serum magnesium and calcium concentrations.

After a sufficient stabilization period (30-60 min following surgical preparation), blood samples from the right renal vein for estimation of plasma renin activity were drawn very slowly to avoid vena cava blood contamination and repeated at 6 hr after supraceliac aortic unclamping. Mg, calcium, creatinine, and Cystatin-C (a marker of glomerular filtration rate) concentrations were drawn from the left brachial artery. At the time of blood sampling, standard hemodynamic variables, RABF, and RCBF were measured. Renal vascular resistances (RVR) were calculated as following equation, RVR = [mean arterial pressure (MAP)-central venous pressure (CVP)] × 80/RABF.

Systemic anticoagulation was achieved with intravenous 100 U/kg heparin sodium 10 min before supraceliac aortic cross clamping. The exposed supraceliac abdominal aorta was then occluded with an atraumatic clip for a period of 60 min, and all data were measured immediately before aortic unclamping (baseline). To prevent the catastrophic increase of blood pressure during the aortic cross clamping, 0.5-5 μg/kg/min sodium nitroprusside (Nitropress Inj, Abott Laboratories, North Chicago, IL, U.S.A.), a ultra-short acting vasodilator, was continuously infused and was stopped immediately before aortic unclamping.

In group Mg, $MgSO_4$ (Magunesin®, Daewon Pharmaceutical Co., Seoul, Korea), was injected as a bolus 30 mg/kg over 3 min immediately before aortic unclamping and was continuously infused 10 mg/kg/hr with a syringe pump (STC 524, Terumo, Tokyo, Japan). The group C received an equivalent volume of saline solution.

Hemodynamic measurements, including heart rate (HR), MAP, central venous pressure (CVP), pulmonary artery pressure (PAP), PAWP, CO, systemic vascular resistance (SVR), and pulmonary vascular resistance (PVR), were repeated at 1, 3, and 6 hr after aortic unclamping (Fig. 1). Blood samples of brachial artery were drawn at 6 hr after aortic unclamping. SVR and PVR were calculated using standard formula [SVR = (MAP-CVP) × 80/CO and PVR = (PAP-PAWP) × 80/CO].

Left kidney was excised and fixed in 10% buffered formalin for histological analysis. The paraffin-embedded specimens were cut perpendicular to the puncture channels in the segment where the microprobe had been located. After staining with hematoxylin and eosin microscopic examination was performed by an independent pathologist for the following parameters; insertion channel of microprobe, morphometry of glomerulus, afferent and efferent arterioles, and neutrophil infiltration. Upon extraction of the thermal diffusion microprobes no signs of hemorrhage into the puncture channel were detected. At the end of each experiment, the animals were euthanized by anesthesia overdose and intra-arterial injection of concentrated potassium chloride solution.

All data are presented as mean±standard deviation (SD). Preoperative parameters were compared to those of the cross clamping and unclamping. Statistical analysis was done by Mann-Whitney test for difference of hemodynamic parameters among test and control groups. Comparisons of variables at different times were tested by the Friedman test followed by Wilcoxon signed-rank test. The five percent level of probability was accepted as significant. Data was analyzed using the SPSS statistical package program version 11.0 (SPSS for Windows, SPSS Inc., Chicago, IL, U.S.A.).

RESULTS

Systemic hemodynamic parameters according to supraceliac aortic cross clamping and unclamping in the two groups are presented in Table 1. There were no statistical differences between the two groups in HR, CVP and PAWP throughout the experiment. MAP increased after aortic cross clamping in both groups, but returned to baseline values after unclamping. CO was reduced significantly compared to baseline value at 6 hr after aortic unclamping in group C (p=0.027). SVR increased at aortic cross clamping in both groups. In group C, SVR increased significantly in comparison to group Mg at 6 hr after aortic unclamping (p=0.016). PVR increased at 3 and 6 hr after aortic unclamping in group C and at 1, 3, and 6 hr after aortic unclamping in group Mg. There were no statistical differences in PVR between the two groups.

Baseline values of RVR in group C and group Mg were 59.6±14.6 dyn·sec/cm^5 and 67.3±24.5 dyn·sec/cm^5, respectively (Fig. 2). After aortic cross clamping, the RVR increased in group C and group Mg (p=0.003 and <0.001, respectively). In group C, the RVR increased compared to baseline value at 1, 3, and 6 hr after aortic unclamping. In group Mg, however, the RVR returned to the baseline value throughout experiment after aortic unclamping. The RABF decreased in both groups for the duration of the experiment. There were no statistical differences in RABF between the two groups.

Baseline values of RCBF in group C and group Mg were 57.0±11.9 mL/min/100 g and 66.3±24.3 mL/min/100 g, respectively (Fig. 3). After aortic cross clamping, the RCBF

Table 1. Systemic hemodynamic parameters in anesthetized dogs after supraceliac aortic cross clamping and unclamping

Parameters	Group	Baseline	ACC	Unclamp-1	Unclamp-3	Unclamp-6
HR	Control	134.7±15.8	126.1±17.7	144.0±10.7	140.7±12.0	144.9±6.5
(bpm)	Magnesium	145.3±11.4	124.6±17.2	145.1±11.5	151.6±13.3	150.3±13.2
MAP	Control	96.7±12.6	130.7±12.9*	100.1±21.2	100.1±10.6	88.9±8.7
(mmHg)	Magnesium	106.1±18.0	127.4±4.3*	102.7±6.9	100.4±12.1	91.1±5.6
CVP	Control	8.0±2.4	9.7±1.4	7.0±2.5	7.2±2.1	7.2±2.4
(mmHg)	Magnesium	7.2±1.4	8.5±1.6	6.8±1.4	6.7±1.0	6.8±1.8
PAWP	Control	9.9±0.7	11.6±2.7	9.6±1.6	9.7±1.1	8.3±1.4
(mmHg)	Magnesium	9.1±1.3	11.1±0.7	9.1±1.3	10.0±2.6	8.9±1.6
CO	Control	3.0±0.9	2.9±0.4	2.5±1.3	2.2±0.9	1.8±0.9*
(L/min)	Magnesium	3.0±0.4	2.8±1.2	2.5±0.8	2.5±0.6	2.4±0.9
SVR	Control	2521±641	3417±547*	3496±937	4316±1409*	5021±1543*
(dyn·sec/cm^5)	Magnesium	2695±784	4001±1668*	3417±1143	3249±913	3197±994†
PVR	Control	239±74	229±38	300±116	371±82*	584±214*
(dyn·sec/cm^5)	Magnesium	240±89	253±123	338±109*	363±130*	392±125*

Values expressed as mean±SD. *p<0.05 versus baseline, $^\dagger p$<0.05 versus control.
ACC; supraceliac aortic cross clamping, Unclamp-1, 3, and 6=1, 3, and 6 hr after supraceliac aortic unclamping. HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PAWP, pulmonary atery wedge pressure; CO, cardiac output; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance.

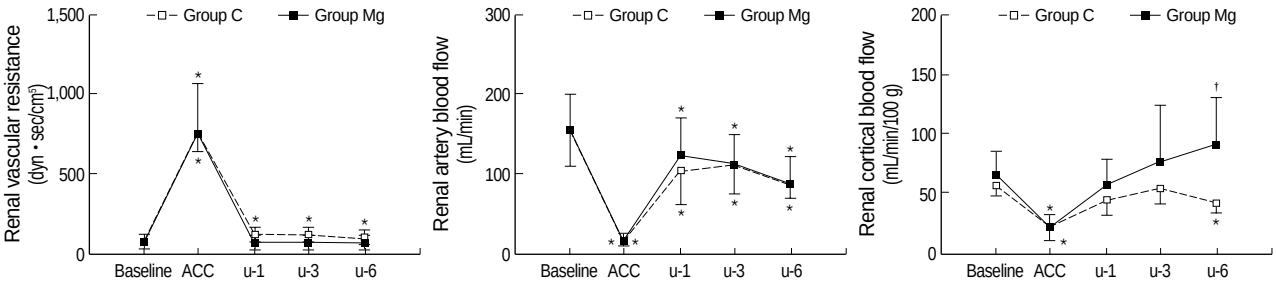

Fig. 2. Renal vascular resistance, renal artery blood flow, and renal cortical blood flow in anesthetized dogs after supraceliac aortic cross clamping and unclamping. ACC; supraceliac aortic cross clamping, U-1, 3, and 6=1, 3, and 6 hr after supraceliac aortic unclamping. *p<0.05 versus baseline, $^\dagger p$<0.05 versus control.

decreased significantly in both groups. At 6 hr after aortic unclamping, there was a significant difference in RCBF between the two groups (p=0.012).

Biochemistry data are presented in Table 2. There were no significant changes in plasma renin activity, serum creatinine, and Cystatin-C between the two groups and all values in both groups were within normal ranges in dogs. Serum Mg concentrations increased significantly from 1.66±0.25 to 2.29 ±0.24 mg/dL in the group Mg at 6 hr after aortic unclamping. There was statistically significant difference in serum Mg concentrations 6 hr after aortic unclamping between the two groups.

Extraction of probes did not result in any hemorrhage into the puncture channels. Histologically the glomeruli showed some neutrophilc and lymphocytic infiltration but the structure of afferent and efferent arterioles was well preserved in both groups (Fig. 3).

Table 2. Biochemistry parameters according to supraceliac aortic cross clamping and unclamping

Parameters	Group	Baseline	Unclamp-6
Renin activity (ng/mL)	Control	7.46±1.44	10.24±4.06
	Magnesium	7.96±1.11	10.64±4.95
Creatinine (mg/dL)	Control	0.78±0.07	0.84±0.24
	Magnesium	0.84±0.21	0.92±0.24
Cystatin-C (mg/L)	Control	0.13±0.01	0.12±0.03
	Magnesium	0.13±0.04	0.10±0.02
Magnesium (mg/dL)	Control	1.51±0.30	1.36±0.29
	Magnesium	1.66±0.25	2.29±0.24*,†
Calcium (mg/dL)	Control	0.88±0.04	0.94±0.26
	Magnesium	0.90±0.04	0.95±0.12

Values expressed as mean±SD. Unclamp-6=6 hr after supraceliac aortic unclamping.
*p<0.05 versus baseline, †p<0.05 versus control.

DISCUSSION

Repair of aortic aneurysm that extend above the level of the renal arteries is associated with a higher mortality rate than infrarenal aortic surgery (14). Severe renal microcirculatory disturbances during aortic surgery may precede the perioperative renal function. Although renal dysfunction certainly increases the mortality after suprarenal aortic aneurysmal repair, the ability to alter the course of acute renal failure associated with aortic surgery is currently limited (15).

To reduce perioperative renal dysfunction, suppressing renovascular vasoconstriction, renovascular vasodilation, decreasing cellular oxygen consumption, and/or attenuating reperfusion injury are crucial (16). Maintenance of adequate renal perfusion is considered to be the most important strategy under these circumstances (17). Several adjunctive measures has been suggested before, during, and after aortic surgery to prevent postoperative renal dysfunction following aortic surgery. Commonly used agents like dopamine, mannitol, and Prostaglandin E1, have not been consistent in preserving renal function after aortic cross clamping and unclamping for aneurysm repair (18, 19). According to a previous study, the vasodilator effect of Mg might also be of use during aortic cross clamping for major vascular operations (20), but in another report intra-aortic Mg during or after aortic cross clamping had no effect on renal function (21).

$MgSO_4$ has been used as an adjuvant for anesthesia (22), and has a number of potential indications in anesthetic practice (6-10). It reduces the requirement for anesthetics and opioids and potentiates their actions (23). We hypothesized that $MgSO_4$ administration might augment renal blood flow and perfusion, and thus prevent the renal dysfunction that follows aortic cross clamping.

Adequate renal function is a result of a generous renal blood

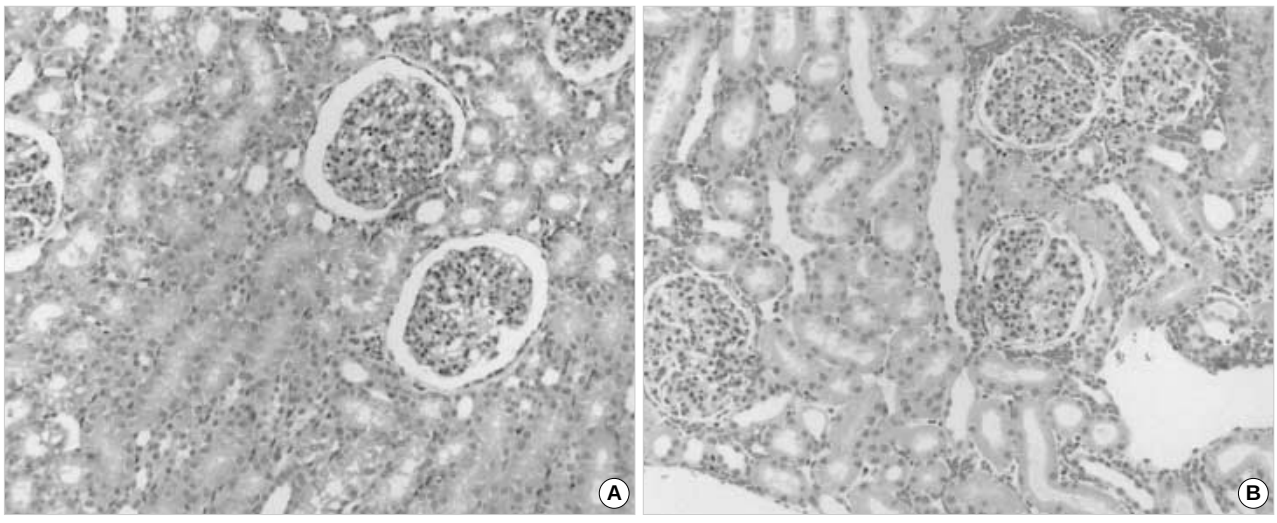

Fig. 3. Light microscopic findings followed by unclamping of supraceliac aortic cross clamping in dogs. Kidneys were fixed by immersion in 10% buffered formalin solution. The glomeruli show some neutrophilc and lymphocytic infiltration but the structure of afferent and efferent arterioles was preserved in both groups (H&E, ×200). (A) group C, (B) group Mg.

flow. The actual renal perfusion is driven by intra-renal vascular resistance. In our present study, the RVR increased throughout aortic unclamping in group C. However, the RVR rapidly returned and persisted close to baseline level throughout the aortic unclamping period in group Mg. This result is coincident with the reports that Mg causes vasodilation in vascular beds and increases renal blood flow (5, 24).

RCBF is a major determinant of renal function. RABF can be unevenly distributed and shunted from superficial to deeper cortical vessels in normal physiologic condition and may be heterogeneous in various pathologic conditions (12, 25). RCBF and medullary blood flow are differentially regulated by various factors, including vascular structure, innervation density, in sensitivity to norepinephrine, or locally acting counter-regulatory mechanisms (26). In previous animal experiment, the renal cortex and medulla had different responses to drugs, endotoxin, and hypoxic stress (27). Therefore, a continuous monitoring of RCBF would be of use for quantification and evaluation of dynamic RCBF changes during experimental pharmacological trials, aiming at renal function improvement (12). The enhanced thermal diffusion electrode which is used in our experimentation is newly developed and allows continuous quantification of parenchymal renal perfusion after local probe implantation. In our present study using thermal diffusion electrode, RCBF decreased at 6 hr after aortic unclamping in group C, whereas the RCBF returned to baseline value in group Mg. This result may suggest that the administration of $MgSO_4$ prevents the increase in RVR and improves the RCBF after the aortic unclamping. However, there were no significant differences between the groups in the changes in plasma renin activity, creatinine, and Cystatin-C levels.

Mg is the second most abundant intracellular cation in the human body. It regulates the movement of calcium into the smooth muscle cells, and therefore has an important role in the maintenance of peripheral vascular tone. The administration of $MgSO_4$ also has some systemic effects like a rapid but transient decrease in arterial pressure in hypertensive patients, but it has no appreciable changes in blood pressure in normotensive subjects (28). In the present study, the MAP after aortic unclamping did not change significantly in any of the groups when compared to baseline. However, the mechanism of the maintenance of MAP was different between the two groups. $MgSO_4$ given as a bolus immediately preceding aortic unclamping followed by a continuous infusion thereafter significantly attenuated the increase in SVR and the decrease in CO after aortic unclamping. In group C, however, the MAP was maintanined by the increase in SVR despite the decrease in CO after aortic unclamping. These hemodynamic results demonstrate that the administration of $MgSO_4$ during supraceliac aortic unclamping causes systemically dilation of vascular bed and attenuates increase in SVR and decrease in CO following aortic unclamping.

We have several limitations in this present study. At first, the Mg and saline were administered immediately before supraceliac aortic unclamping and thereafter as an infusion. In general, the adjuvants to protect renal dysfunction for aneurysm repair surgery are administered before aortic cross clamping or after the induction of anesthesia. Secondly, to protect renal dysfunction the CO and oxygen delivery should be well controlled during aortic cross clamping and unclamping. In our experimental design, however, we did not control the CO throughout the experimentation. The reason for this was that we wanted to find out the systemic and renal effect of Mg itself without control of hemodynamics. Finally, in spite of the changes in blood flow of the renal cortex and medulla have different responses, we measured only RCBF as local renal perfusion but not renal medullary blood flow. Therefore, the measurement of the renal medullary blood flow should be simutaneously performed with the RCBF measurement to understand the whole distribution of the renal blood flow.

In summary, $MgSO_4$ 30 mg/kg over 3 min immediately before aortic unclamping followed by an infusion of 10 mg/kg/hr attenuated the increase in RVR and improves RCBF after aortic unclamping in dogs. And the CO and SVR were well preserved after aortic unclamping. However, despite these beneficial effects, we could not find any improvement of renal function when $MgSO_4$ was administered after supraceliac aortic unclamping.

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