

Blunt Renal Artery Trauma: How Should it be Treated, or rather, Should it be Treated?

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One hundred and fifty years after its first description, the treatment of renal artery occlusion secondary to trauma is still controversial. Although some reviews report a high success rate with revascularization [2], more recent series suggest that restoring normal renal function is unlikely when the main renal artery is injured [3,4]. The success of any arterial reconstruction is related to the duration and degree of ischemia, presence or absence of collateral arteries, and the complexity of the repair. Warm kidney ischemia of more than 2 hours causes irreversible tissue damage, but collateral circulation via the renal capsular, peripelvic and periureteric vessels as well as a patent renal vein may extend the kidney viability beyond the 2 hour time limit [5]. Moreover, the success of renal artery reconstruction is difficult to interpret as outcomes have not been consistently defined and follow-up tends to be poor in these patients.

Isolated renal artery injury is the result of either penetrating or blunt trauma. The decision regarding management is dictated mainly by the patient's hemodynamic status and the extent of the damage to the renal artery. There is general agreement that minor renal artery injury should be treated conservatively. On the other hand, in patients with a single kidney, repair of the renal artery should be attempted unless

the patient's condition is deteriorating. In patients needing damage-control maneuvers, the renal artery should be repaired by simple suturing if possible, avoiding any complex repairs. If the renal artery is injured at the hilum area, nephrectomy is the treatment of choice.

In most patients with two normal kidneys, the combination of multiple injuries and prolonged "warm ischemic time" dictates either immediate nephrectomy or no intervention at all.

Blunt trauma to the artery is the leading cause of injuries in the civilian population. The most common mechanism is spontaneous deceleration, leading to intimal tear, dissection, pseudoaneurysm or transection, either complete or incomplete. The proximal part of the artery, beginning 2–4 cm distal to the renal artery orifices, is the most vulnerable arterial part since the renal artery is "anchored" to the aorta proximally and to the kidney distally, making this mobile part prone to injury.

Historically, surgical repair of renal artery injuries has had dismal results. In a group of 228 patients with single renal artery injury, 34 underwent surgical revascularization but only 8 (23%) had satisfying technical results and 6 (18%) developed postoperative hypertension [6]. In another group of 12 patients, 5 had surgical revascularization and 7 were treated conservatively. Of the five patients who underwent surgery, with median warm ischemic time of 5 hours, four operations were technically successful yet all patients lost their kidney within 90 days and one died. Of the seven patients who were treated conservatively, three had delayed nephrectomy due to uncontrolled hypertension and four were normotensive [7].

Feliciano [8], a leading authority in trauma, stated in 2004: "Based on the historical and recent data it is difficult to recommend revascularization of one renal artery in a patient with a functioning contralateral kidney. This is especially true if the patient has other serious injuries and the time to revascularization would exceed 6 hours from the injury."

In recent years endovascular therapy has been proposed as the ideal method of treating blunt renal artery trauma. Yet the current literature supporting such treatment is based on selected case reports, with most of the therapies lacking scientific evidence and disregarding the potentially severe and even lethal complications of such treatment [9,10]. The three patients presented in this issue of IMAJ are a good example of this attitude [12].

At the present time it seems there is a place for endovascular management of traumatic renal artery injuries in a selected group of patients in whom restoration of renal artery blood flow can be achieved in less than 3 hours in a viable kidney. When the renal artery is thrombosed, the warm ischemic time exceeds 3 hours and there is no evidence of collateral blood flow, we believe that conservative management will probably produce better results.

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References

1. Von Rechlinghausen F. Haemorrhagische Niereninfarkte. *Arch Path Anat Physiol* 1861; 20: 20.
2. Lock JS, Carraway RP, Hudson HC, et al. Proper management of renal artery injury from blunt trauma. *South Med J* 1985; 78: 406-10.
3. Haas CA, Spirnak JP. Traumatic renal artery occlu-

- sion: a review of the literature. *Tech Urol* 1998; 4: 1-11.
4. Glenski WJ, Husmann DA. Traumatic renal artery thrombosis: management and long term followup. *J Urol* 1995; 153: 316A.
 5. Lohse JR, Shore RM, Belzer FO. Acute renal artery occlusion: the role of collateral circulation. *Arch Surg* 1982; 117: 801-4.
 6. Clark DE, Jeorgitis JW, Ray FS. Renal arterial injuries caused by blunt trauma. *Surgery* 1981; 90 (1): 87-96.
 7. Hass CA, Dinchman KH, Nasralla PF, Spirnal JP. Traumatic renal artery occlusion: a 15 year review. *J Trauma* 1998; 45: 557-61.
 8. Feliciano D. Injury to the abdominal aorta and visceral arteries. In: Rich N, Mattox K, Hirshberg A, eds. *Vascular Trauma*. 2nd edn. Philadelphia: Saunders, 2004: 299-313.
 9. Bruce LM, Croce MA, Santaniello JM, Lyden SP, Fabian TC. Blunt renal artery injury: incidence, diagnosis and management. *Am Surg* 2001; 67 (6): 550-4.
 10. Dinkel PH, Danuser H, Triller J. Blunt renal trauma: minimally invasive management with microcatheter embolization – experience in nine patients. *Radiology* 2002; 223: 723-30.
 11. Abu-Gazala M, Shussman, N, Abu-Gazala S, et al. Endovascular management of blunt renal artery trauma. *IMAJ Isr Med Assoc J* 2013; 15: 278-83.

Capsule

High frequency oscillation in early acute respiratory distress syndrome

Previous trials suggesting that high frequency oscillatory ventilation (HFOV) reduced mortality among adults with the acute respiratory distress syndrome (ARDS) were limited by the use of outdated comparator ventilation strategies and small sample sizes. In a multicenter randomized controlled trial conducted at 39 intensive care units in five countries, Ferguson et al. randomly assigned adults with new-onset, moderate-to-severe ARDS to HFOV targeting lung recruitment or to a control ventilation strategy targeting lung recruitment with the use of low tidal volumes and high positive end-expiratory pressure. The primary outcome was the rate of in-hospital death from any cause. On the recommendation of the data monitoring committee, the trial was stopped after 548 of a planned 1200 patients had undergone randomization. The two study groups were well matched at baseline. The HFOV group underwent HFOV for a median of 3 days (interquartile range 2-8); in addition, 34 of 273 patients (12%) in the control

group received HFOV for refractory hypoxemia. In-hospital mortality was 47% in the HFOV group, as compared with 35% in the control group (relative risk of death with HFOV 1.33; 95% confidence interval 1.09-1.64, $P = 0.005$). This finding was independent of baseline abnormalities in oxygenation or respiratory compliance. Patients in the HFOV group received higher doses of midazolam than did patients in the control group – 199 mg/day (interquartile range 100-382) vs. 141 mg/day (interquartile range 68-240), $P < 0.001$, and more patients in the HFOV group than in the control group received neuromuscular blockers (83% vs. 68%, $P < 0.001$). In addition, more patients in the HFOV group received vasoactive drugs (91% vs. 84%, $P = 0.01$) and received them for a longer period than did patients in the control group (5 days vs. 3 days, $P = 0.01$).

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Eitan Israeli

Capsule

Genetic variation in the serotonin receptor gene affects immune responses in rheumatoid arthritis

Many genetic variants associate with the risk of developing rheumatoid arthritis (RA); however, their functional roles are largely unknown. Snir and colleagues investigated whether the RA-associated serotonin receptor 2A (*HTR2A*) haplotype affects T cell and monocyte functions. Patients with established RA (n=379) were genotyped for two single-nucleotide polymorphisms in the *HTR2A* locus, rs6314 and rs1328674, to define presence of the risk haplotype for each individual. Patients with and without the RA-associated TC haplotype were selected and T cell and monocyte function was monitored following in vitro stimulations with staphylococcal enterotoxin B and lipopolysaccharide using multiparameter flow cytometry. Within the cohort, 44 patients were heterozygous for the TC haplotype (11.6%) while none were homozygous. Upon

stimulation, T cells from TC-carrier patients produced more pro-inflammatory cytokines, namely tumor necrosis factor-alpha (TNF α), interleukin-17 and interferon gamma, and monocytes produced higher levels of TNF α compared with patients carrying the non-TC haplotype ($P < 0.05$ and 0.01, respectively). Such cytokine production could be inhibited in the presence of the selective 5-HT2 receptor agonist (2,5-dimethoxy-4-iodoamphetamine, DOI); interestingly, this effect was more pronounced in TC carriers. Our data demonstrate that association of RA with a distinct serotonin receptor haplotype has functional impact by affecting the immunological phenotype of T cells and monocytes.

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“Children seldom misquote you. In fact, they usually repeat word for word what you shouldn’t have said”

Anonymous