



Letter to the Editor

The effect of glutamine on oxidative damage in an experimental abdominal compartment syndrome model in rats

Dear Editor,

We read with great interest the article by Tihan D et al., regarding an experimental study about The effect of glutamine on oxidative damage in an experimental abdominal compartment syndrome model in rats.^[1]

We would like to congratulate the authors for their grate work which is very innovative.

However, it would be very informative if the authors could provide details regarding the sacrifice time of their groups. How did the author chose sacrifice time (time frame)? As we know in literature date, Abdominal compartment sendrome is emergency disease which effects many organ in the abdomen. The author chose 3th day after compartment sendome.It would be to long for seeing early damages in abdominal organ. The data is very clear from large studies that there is a correlation between elevated intra-abdominal pressure and worse outcome in terms of organ failure, ICU length of stay and mortality. Malbrain et al in 2005 demonstrated this conclusively in a multicenter trial showing even "mild" elevations of IAP (>12 mmHg) lead to worse outcomes probably due to the prolonged organ ischemia that occurs.^[2] Sugrue et al.^[3] in 1999 showed elevated IAP (over 18 mm Hg) was an independent predictor of renal failure, ranking up with hypotension, age and sepsis. Vidal^[4] found 64% of patients in a mixed ICU population had IAH, which was an independent risk factor for organ dysfunction and death. Pupelis prospectively collected IAP and outcome data on pancreatitis patients and also found significant differences in outcomes. Those patients with IAP less than 18 mmHg had no mortality, 19% incidence of MODS/SIRS and mean ICU length of stay of 9 days whereas patients with IAP greater than 18 mmHg had 36% mortality, 64% incidence of MODS/SIRS and mean ICU length of stay of 21 days.^[5,6] They conclude "The critical IAP values... with the best sensitivity specificity, were 23 mmHg for postoperative ventilatory delayed weaning (p<.05), 24 mm Hg for renal dysfunction (p<.05), and 25 mmHg for death (p<.01).

However, In this manuscript, the author chose 20 mmHg abdominal pressure within two hours. Was there any literature about this pressure and sacrifice time in rats? Also, it would be very useful if the

authors could provide additional information regarding the statistical analysis of the data. In particular, there is no description of how the sample size of each group of laboratory animals was defined. Was there any prior pilot study that described the variance?

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References

1. Tihan DN, Erbil Y, Seven R, Arkaya S, Türkoğlu U, Hepgül G, et al. The effect of glutamine on oxidative damage in an experimental abdominal compartment syndrome model in rats. *Ulus Travma Acil Cerrahi Derg* 2011;17:1-8.
2. Malbrain ML, Chiumello D, Pelosi P, Bihari D, Innes R, Ranieri VM, et al. Incidence and prognosis of intraabdominal hypertension in a mixed population of critically ill patients: a multiple-center epidemiological study. *Crit Care Med* 2005;33:315-22.
3. Sugrue M, Jones F, Deane SA, Bishop G, Bauman A, Hillman K. Intra-abdominal hypertension is an independent cause of postoperative renal impairment. *Arch Surg* 1999;134:1082-5.
4. Vidal MG, Ruiz Weisser J, Gonzalez F, Toro MA, Loudet C, Balasini C, et al. Incidence and clinical effects of intra-abdominal hypertension in critically ill patients. *Crit Care Med* 2008;36:1823-31.
5. Pupelis G, Austrums E, Snippe K, Berzins M. Clinical significance of increased intraabdominal pressure in severe acute pancreatitis. *Acta Chir Belg* 2002;102:71-4.
6. Pupelis G, Zeiza K, Plaudis H, Suhova A. Conservative approach in the management of severe acute pancreatitis: eight-year experience in a single institution. *HPB (Oxford)* 2008;10:347-55.
7. Biancofiore G, Bindi ML, Boldrini A, Consani G, Bisà M, Esposito M, et al. Intraabdominal pressure in liver transplant recipients: incidence and clinical significance. *Transplant Proc* 2004;36:547-9.

Author's response

Dear Editor,

I'm very pleased to get such important opinions of reades and other authors, and also I would like thier kind intrests. I want to answer to these comments, question by question.

First of all, I'm agree that the abdominal compartment syndrome (ACS) is an emergency and should be

treated as soon as possible. However there are some predictable situations which may cause abdominal hypertension such as prolonged major laparoscopic procedures or early-stage pancreatitis. What we want to search is may the use of glutamine be effective in these entities on clinical practice. For example, diet rich in glutamine can be added to the nutritional supplement of a patient who is on the preparation stage of a major laparoscopic surgery. Additionally, if ACS occur, glutamine can also be added rapidly to the total parenteral nutrition supply.

So, we decided to administer glutamine one week before insufflation, and to sacrifice rats 3 days after intraabdominal hypertension procedure. Yet, many different experimental model can be planned.

Obviously, prolonged mild elevations of intraabdominal pressure (IAP) (grade I and II regarding to Burch classification) also can cause multi-organ damage due to ischemia.^[1,2] We decided to insufflate rats by 20 mmHg according to another experimental abdominal hypertension model performed by Kaçmaz et al.^[3] Furthermore, this experiment can modified.

Other sorts of rats are also used in many other models, for example male Brown Norway rats, or male Sprague-Dawley rats.^[4,5] We choose Wistar-Albino rats, because this species are easy to find and frequently preferred.^[2,3]

I tried to answer all questions. I hope it would be useful. I want to thank again to you and the author of the letter.

Sincerely,

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References

1. Burch JM, Moore EE, Moore FA, Franciose R. The abdominal compartment syndrome. *Surg Clin North Am* 1996;76:833-42.
2. Akbulut G, Polat C, Aktepe F, Yilmaz S, Kahraman A, Serteser M, et al. The oxidative effect of prolonged CO2 pneumoperitoneum on renal tissue of rats. *Surg Endosc* 2004;18:1384-8.
3. Kaçmaz A, Polat A, User Y, Tilki M, Ozkan S, Sener G. Octreotide improves reperfusion-induced oxidative injury in acute abdominal hypertension in rats. *J Gastrointest Surg* 2004;8:113-9.
4. Duenschede F, Erbes K, Riegler N, Ewald P, Kircher A, Westermann S, et al. Protective effects of ischemic preconditioning and application of lipoic acid prior to 90 min of hepatic ischemia in a rat model. *World J Gastroenterol* 2007;13:3692-8.
5. Tuncer MC, Ozturk H, Buyukbayram H, Ozturk H. Interaction of L-arginine-methyl ester and Sonic hedgehog in liver ischemia-reperfusion injury in the rats. *World J Gastroenterol* 2007;13:3841-6.