Extended Abstract

Transplant Reports: Open Access

2019

Vol.5 Issue.3

Impacts of Renal Denervation and Vagus Nerve Stimulation on Acute Renal Failure Induced by Renal Ischemia Reperfusion injury in Rat Model

Dr. Mohamed H ElSayed

Assistant Professor of Physiology, Faculty of Medicine, Ain Shams University, Egypt

Abstract

Ischemia-Reperfusion Injury (IRI) is characterized by temporary cessation followed by restoration of blood supply and re-oxygenation of a certain organ. In the kidney, IRI contributes to Acute Kidney Injury (AKI) with rapid kidney damage and high morbidity and mortality rates. A surgical or drug-induced blockage of renal sympathetic nerve prevents, partially, the development of IR-induced AKI. Modulation of the Cholinergic Anti-Inflammatory Pathway (CAP) by Vagal Nerve Stimulation (VNS) has also a delayed but effective impact in renal IRI. However, the combined effect of renal sympathectomy and VNS had not been well investigated.

Introduction

Acute Kidney Injury (AKI) is commonly associated with high mortality and morbidity and may lead to Chronic Kidney Disease (CKD) and End-Stage Renal Disease (ESRD) [1-3]. AKI is a major consequence of ischemia-reperfusion injury due to decreased arterial and venous blood flow which results in cellular death due to the depletion of energy stores and toxic metabolite accumulation. However, restoration of blood flow and re- oxygenation of the ischemic tissue may paradoxically exacerbate the injury . Actually, re-oxygenation to a previously hypoxic tissue is the basic mechanism for the formation of reactive oxygen species [6]. Consequently, multiple enzyme systems including proteases, nitric oxide synthases, phospholipases, and endonuclease are induced and stimulated causing cytoskeleton disruption, membrane damage, DNA degradation, and eventually cell death, AKI is a clinical syndrome characterized by abrupt and sustained decline in glomerular filtration rate resulting in the accumulation of urea and other chemicals in the blood. The neuro-immunological interaction maintains homeostasis and ameliorates responses to stressful conditions and injury. Fast nerve conduction can essentially modulate inflammation [8]. One of the nervous reflexes that modulate inflammation is the Cholinergic Anti-Inflammatory Pathway (CAP). Such pathway modifies innate and adaptive immunity mainly with the spleen and modulation of this reflex by Vagus Nerve Stimulation (VNS) is effective in different inflammatory diseases. Therefore, cholinergic signals derived by VNS provide continuous nervous regulation of cytokine synthesis, which limits the magnitude of the inflammatory immune response and mediates protective effect against AKI induced by IRI.

Keywords

Acute kidney injury; Ischemia-reperfusion injury; Renal denervation; Vagus nerve stimulation.

This work is partly presented at Joint Event on International Conference on Organ Donation & Transplantation Science, April- 09-10, 2019 at Sydney, Australia

Extended Abstract | Transplant Reports: Open Access | 2019 | Vol.5 Issue.3

Blood and Tissue Sampling

Blood samples were collected from retro-orbital plexus with the help of capillary tube into serum gel tubes which were centrifuged (6000 rpm for 15 min) to separate serum. The serum was then pipetted into clean storage tubes and stored at -20°C for later determination of serum urea, creatinine, and MDA. Whereas, left kidneys were stored at -80°C for later determination of renal tissue GPX and renal tissue nitrate and renal tissue TNFα as well. On the other hand, right kidneys were divided into 2 halves; one of them was stored in 10% formalin for later light microscopic examination while the other half was stored in glutaraldehyde for E/M examination.

Histological and Immunohistochemical Studies

One half from right kidney was processed for histological examination by light microscope, the kidney specimens were fixed in 10% neutral buffered formalin and paraffin blocks were prepared. Serial 4-6 µm-thick sections were cut and stained by Hematoxylin and Eosin (H & E), Masson trichrome (MT) stain and BCL2 immune reaction. Another half from right kidney was divided into 2 parts, one (1/4 kidney) processed for transmission electron microscopic examination, small kidney specimens (1 mm3) were fixed in a 4% glutaraldehyde solution. Ultrathin sections were cut and examined using a 1200 EX Jeol, Japan, transmission electron microscope at the Electron Microscopic Unit-Faculty of Science, Ain Shams University. The second part (1/4 kidney) was placed on the ice quickly and homogenized with lysis buffer.

References

1. Coca SG, Singanamala S, Parikh CR (2012) Chronic kidney disease after acute kidney injury: A systematic review and meta-analysis. Kidney Int 81: 442-448.

2. Schiffl H, Lang SM, Fischer R (2012) Long-term outcomes of survivors of ICU acute kidney injury requiring renal replacement therapy: A 10-year prospective cohort study. Clin Kidney J 5: 297-302.

3. Rifkin DE, Coca SG, Kalantar-Zadeh K (2012) Does AKI truly lead to CKD? J Am Soc Nephrol 23: 979-984.

4. Kellum JA, Unruh ML, Murugan R (2011) Acute kidney injury. BMJ Clin Evid.

E-mail: doctorpioneer@yahoo.com

This work is partly presented at Joint Event on International Conference on Organ Donation & Transplantation Science, April- 09-10, 2019 at Sydney, Australia