African Journal of Biology and Medical Research
ISSN: 2689-534X
Volume 3, Issue 1, 2020 (pp. 9-14)
www.abjournals.org

BRAIN KIDNEY INTERACTIONS IN POST TRAUMATIC BRAIN DEATH

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ABSTRACT: Brain death is the cause of a major inflammatory systemic response syndrome (SIRS) affecting all the organs and partly responsible for their dysfunction. One of these consequences is kidney failure (AKI: Acute Kidney Injury). Between September 2008 and September 2013, renal function was analyzed in patients admitted for severe head injury who progressed to brain death. Impairment of renal function has been demonstrated in these subjects upon admission, suggesting a direct interaction between the brain and the kidney. An early deterioration of renal function in the subject in a brain death state is often highlighted in our study, its occurrence results from the addition of hemodynamic, neurohormonal and inflammatory factors.

KEYWORDS: Brain Death, Kidney Failure, Interaction, Acute, Injury, Systemic Inflammatory Response Syndrome (SIRS), Post Traumatic Brain Death

INTRODUCTION

Brain death is one of the consequences of severe head trauma and the cause of a major inflammatory systemic response syndrome (SIRS) affecting all the organs and partly responsible for their dysfunction. One of these consequences is kidney failure (AKI: Acute Kidney Injury).

It can cause post-transplant complications if it is not taken care of quickly. But more than that, direct interactions seem to exist between the brain and the kidney and begin to be highlighted.

LITERATURE/THEORETICAL UNDERPINNING

Encephalopathy are clinical complications of acute kidney injury and acute brain injury can also induce a variety of altered renal function. Clinically, studies have reported a link between brain death and increased inflammatory response in the kidneys used for organ donation, leading to donor graft dysfunction[1].

The brain–kidney interconnection is more complicated than can be explained by examining either organ independently, and it likely involves inflammatory, unknown hormonal, or cytokine-mediated organ dysfunction, genetic, and blood pressure-related physiology. Therefore they must also be understood in the greater context of critical care, molecular/cellular biology, medicine, and physiology[2].

METHODOLOGY

All patients hospitalized for severe head trauma between September 2008 and September 2013, who progressed to brain death were prospectively included in our study and matched with dead severe head injuries, but of non-neurological cause. The renal function (serum creatinine and urea level) was analyzed and compared (MDRD: Modified diet in renal disease) between the two series, the GFR (glomerular filtration rate) is calculated according to the formula CKD-EPI (Chronic Kidney Disease Epidemiology).

The advantage of using this criterion is that the glomerular filtration rate (GFR) is the best quantitative marker of renal function because it is directly correlated to the quantity of functional nephrons. In clinical practice, it is recommended to estimate the GFR using formulas derived from serum creatinine.

The HAS and international guidelines recommend the use of the CKD-EPI formula, which has demonstrated the best performance across the entire GFR spectrum, as first line.

Statistical analysis has been performed with biostatgv free tool.

RESULTS/FINDINGS

An impairment of renal function is demonstrated in 56 patients, Creatininaemia = 104.84 ± 49.94 µmol / l in the cases (EME +) and 74.16 ± 20.86 µmol / l in the controls (EME -) with a significant difference between the two populations (p = 0.004), a stage 3 MRC is also highlighted upon admission in 25% of cases (EME +).

The results of our study are presented in the following table **(Table 1)**.

Discussion

This result confirms the existence of an alteration in early renal function during brain death and raises the possibility of an interaction between the brain and the kidney. The brain death is associated with an intense systemic inflammatory response, more precisely the expression of adhesion molecules ICAM-1 and VCAM-1 is high on kidney biopsies from subjects in brain state, their level is correlated with delayed impairment of renal function. In our series we were able to highlight this drop in glomerular filtration in brain death subjects using MRC classification [3] **(Table 2)**.

The increase in kidney transplants from brain dead subjects has led several teams to question the effects of brain death on the viability of transplanted organs [4]. Sanchez-Fructuoso et al [5] reported significantly lower viability of transplanted kidneys from subjects with brain death. Other studies have found a correlation between kidney from a subject in brain death, delayed recovery of renal function and increased acute rejection [6].

Brain death appears to affect kidney function through a combination of factors such as :

- Hemodynamic effects [7].
- Neurohormonal activation [8].

• An inflammatory response [9].

• Endothelial activation [10].

This impairment of renal function meets the definition of Acute Kidney Injury (AKI) [11] which is a clinical-biological syndrome encompassing the entire spectrum of acute renal failure, from minor changes to the need for an extra-renal purification, and of which the etiologies are multiple. AKI defined as an increase in plasma creatinine ≥ 26.5 μmol / l in the last 48 hours or a recent increase (occurring in the last 7 days) in plasma creatinine ≥ 1.5 times the base creatinine or a diuresis of less than 0.5 ml / kg / h for at least 6 h. The pathophysiology of AKI remains unclear, it is becoming increasingly evident that AKI should be considered, not as a pathology, but rather as a clinical-biological syndrome with multiple etiologies.

It is moreover likely that when an AKI appears, it results from renal attacks. Consequently, acting on the risk factors of exposure and susceptibility takes on its full significance and must make it possible to limit the risk of progression to an established AKI or to limit its severity if it occurs.

In 2018, an Iceland study [12] evoked the similarity between the factors altering the microcirculation at the cerebral level and at the renal level with manifestations relating to the same pathophysiology, showing that even before the stage of chronic renal failure, the decrease in DFG was associated with brain damage. Albuminuria, which has also been measured, is an early sign of vascular damage to the microcirculation and the perforating cerebral arterioles have the same structure as the afferent renal arterioles. The other similarities between the two organs are hemodynamic, they are both subjected to a very large blood flow and high perfusion pressures can alter the vascular endothelium. These data, although they do not make it possible to envisage any specific therapy, nevertheless encourage early correction of all of the cardiovascular risk factors.

Implication to Research and Practice

Our study confirms the occurrence of impaired renal function upon admission in 25% of our brain death patients.

This alteration in renal function manifests itself from the first hours of treatment and cannot therefore be attributed to initial resuscitation.

So certain measures are to be taken in patients at risk are as follows [13]:

- Stop the exposure of any nephrotoxic agent as far as possible.

- Optimizing blood volume, oxygenation and renal perfusion pressure

- Monitor patients hemodynamically.

- Closely monitor serum creatinine and diuresis.

- Avoid hyperglycemia

- Avoid injection of radiological contrast medium as far as possible or carry out a volume expansion before the examination using isotonic saline or a solution of sodium bicarbonates.

In our case this situation imposes to anticipate this acute kidney injury and quickly have some actions for avoid kidney destruction.

CONCLUSION

An early deterioration of renal function in brain death subject is often highlighted, its occurrence results from the addition of hemodynamic, neurohormonal and inflammatory factors, this notion, confirmed in the literature, could allow the anticipation of this situation by optimizing the potential donor early and by preparing the transplant teams for the management of a difficult post-transplant, but in general this deterioration of renal function in a subject in a brain-dead state should not reject the sample of the graft, this due to the current shortage of organs [13].

Future Research

This new situation introduces the concept of organ resuscitation [14] where the anesthesiologist plays a proactive role as soon as a brain death is announced or predictive factors are present [15].

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ISSN: 2689-534X
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APPENDIX

TABLES

Table 1: Results

	Cases	Control	p
Total	95	95	
Age (mean±sd) in years	25,88±15,08	38,71±21,53	<0.01
Urea (mean±sd) in g/l	0.42±0,29	0.29±0,10	<0,01
Créatininemia (mean±sd) in µmol/l	104.84 ± 49.94	74.16 ± 20.86	<0,04

Table 2: Stages GFR in our Serie

			Brain Death		Total
			No	Yes	
Stage	1,00	GFR>90 ml/mn/1.73m^2	22	10	32
	2,00	60<GFR<89	14	5	19
	3,00	30<GFR<59	0	5	5
Total			**36**	**20**	**56**