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The intra-dialytic hypertension: prevalence and risk factors

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ABSTRACT

Introduction:The intra- dialytic hypertension is a complication usually defined by an increase in mean arterial pressure above 15mmHg during or immediately after the dialysis session .Materials and methods:Retrospective study of 28 months (May 2010 to September 2012) in our hemodialysis center aimed to determine the prevalence and risk factors for intra- dialytic hypertension.Results: The intra- dialytic hypertension was noted in 7.9 % of sessions . The above 60years age, diabetes , heart disease , male sex , the temperature of the dialysate , and the shooting ratio of weight / dry weight , were identified as contributing factors its occurrence . Sodic profiles significantly influence the prevalence of this complication. His support was based primarily on the optimization of ultrafiltration in 75 % of cases. Conclusion : The intra- dialytic hypertension is a rare complication , its pathophysiology is poorly understood and its treatment is not codified mainly based on optimizing the basis weight that should be done with caution . Further studies are needed to assess the epidemiology, pathophysiology and management of this complication

Keywords: Intra-dialytic Hypertension, Resistors Vascular, Cardiac Output, Sympathetic hyperactivity, Renin Angiotensin System, Hypervolemia.

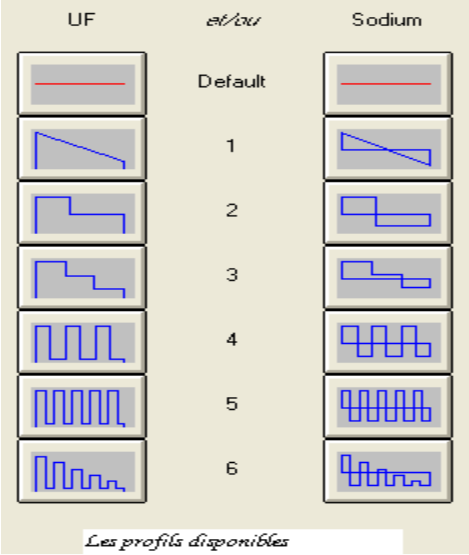
INTRODUCTION

The intra-dialytic hypertension is a rare complication, usually defined by an increase in mean arterial pressure of more than 15mmHg during or immediately after the dialysis session. However, there is no consensus on this definition. Its pathophysiology is complex and poorly understood and its management is not codified. We analyzed the prevalence and risk factors associated with this high on 16,000 sessions conducted in our hemodialysis center.

MATERIALS AND METHODS

This is a retrospective study over 28 months (May 2010 - September 2012) in our hemodialysis center, aimed to determine the prevalence and risk factors for intra-dialytic hypertension. The definition consideration was the increase in mean arterial pressure of more than 15mmHg during or immediately after the dialysis session.

The factors studied were:
Advanced age (> 60 years), diabetes, gender, presence of heart disease, length of hemodialysis, the report taking weight / dry weight, the dialysate conductivity, temperature dialysate flow of the blood pump, and the sodium and profiles ultrafiltration.



Statistical analysis was conducted by the test of two Chi, Student's t test and ANOVA.

RESULT AND DISCUSSION

Prevalence
16000 sessions conducted during the study period; the prevalence of intra-dialytic hypertension was 7.9%.

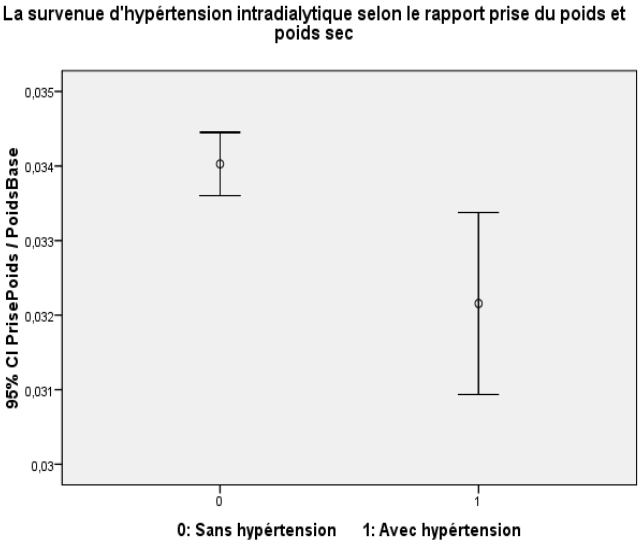
Risk factors:
Contributing factors the occurrence of intra-dialytic hypertension found statistically significant in our study were: age greater than 60 years, diabetes, presence of heart disease, male sex, the temperature of the dialysate, and the report: taking weight / dry weight.

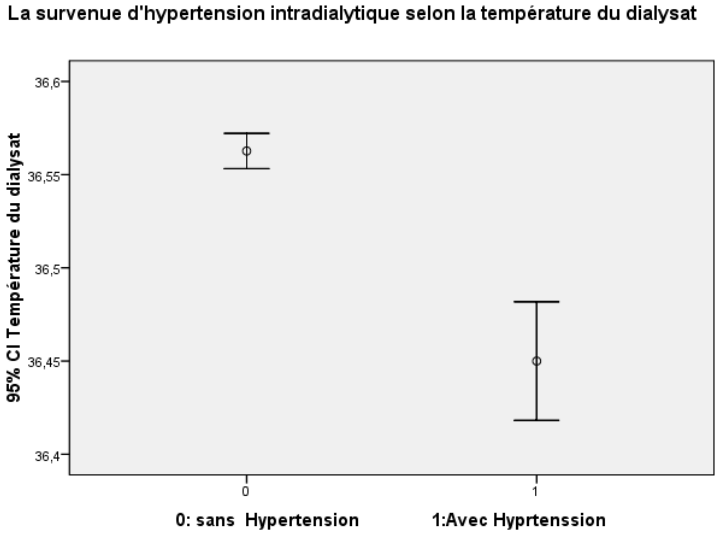
Table I: prevalence of intra-dialytic hypertension as risk factors

	prevalence of intra-dialytic hypertension	p
Diabétic patient	11.60%	**0.000**
No diabete	7%	
Male	8.90%	**0.000**
Femal	6.20%	
Age < 60 years	5%	**0.000**
Age >60 years	12.20%	
With cardiopathy	11.90%	**0.000**
Without cardiopathy	7.20%	
Dialysis vintage < 2 yrs	8%	0.360
Dialysis vintage > 2yrs	7%	

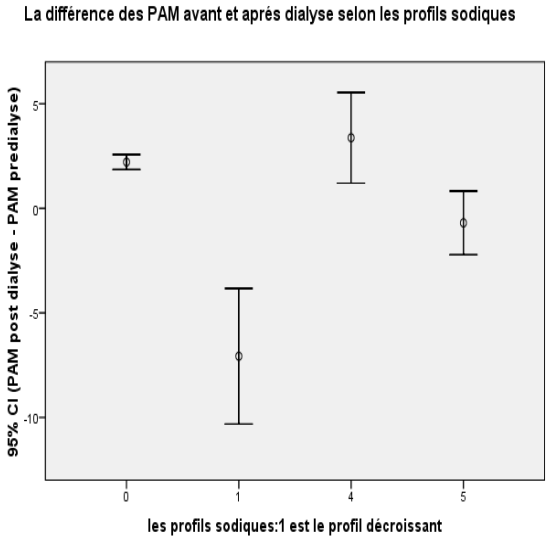
Table 2: Comparison of the average occurrence or not of high intra-dialytic hypertension

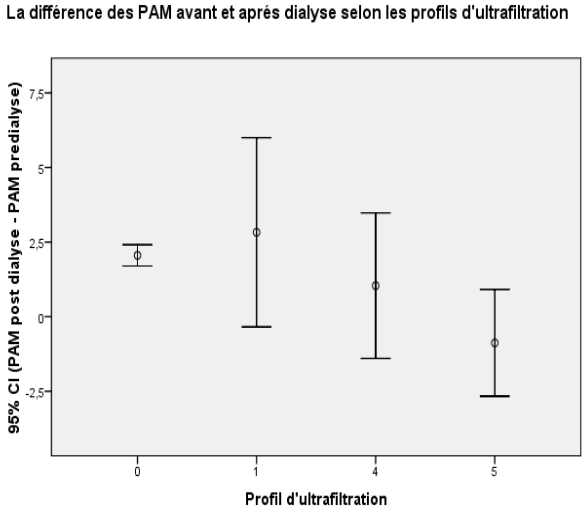
	With intradialytic hypertension	Without intradialytic hypertension	p
Weight gain/Dry weight	0,032±0,02	0,034±0,022	0.003
Température of dialysate °C	36,40± 0,58	36,56± 0,44	0
Conducitivity mS/cm	13,98± 0,12	13,96± 0,20	0.08
Blood flow (ml/mn)	289± 36	288±35	0.09





The comparison of the average of the average difference before and after dialysis blood pressures (ΔPAM) was significant as sodium profiles (p = 0.000) and not significant as ultrafiltration profiles (p = 0.18).





Sodic profiles significantly influence the prevalence of intra-dialytic hypertension (p = 0.001), which does change the ultrafiltration profiles (p = 0.36).

Prévalence of intradialytic hypertension depending on sodium profil				
	sodium profil			
	0	1	4	5
intradialytic hypertension	8%	5.50%	9.70%	6%

Prévalence of intradialytic hypertension depending on ultrafiltration profil				
	ultrafiltration profil			
	0	1	4	5
intradialytic hypertension	8%	10.50%	8.70%	6%

Discussion:

a) Definition:

Several definitions of intradialytic hypertension have been described [1]. Indeed there is no consensus on the level of blood pressure required to meet this definition. [1] Some definitions have been described for this hypertension also described as paradoxical:

• An increase in mean arterial pressure above 15mmHg during or immediately after the dialysis [2].
• HTA during the second or third hour of dialysis after an effective ultrafiltration [3].
• Increased blood pressure resistant ultrafiltration
• Systolic blood pressure greater than the post ultrafiltration pre ultrafiltration pressure [4].

b) Prevalence:

The prevalence is unknown and highly variable across studies from 5 to 15% according to the definition considered [1]. In our study it was 7.9 %.

c) Pathophysiology:

The pathophysiology is complex, poorly understood involving several theories:

1) The increase in vascular resistance :

KJ Chou was a significant elevation of blood pressure resistance in patients with Intra-dialytique hypertension [5]. This increased resistance could be explained by:

a) An overactive renin-angiotensin system:

Intra-dialytic hypertension appears to be due to hyperactivity of the renin angiotensin system in response to a rapid ultrafiltration [1]. Fellner observed that patients with tubulointerstitial nephritis or nephrectomized have a low risk of developing intra-dialytic hypertension and explained this by a decreased activity of the renin system in these patients [2]. Bazzato G et al found that the use of enzyme inhibitors (captopril) at the beginning of the dialysis session possible to reduce the incidence of intradialytic hypertension [6].

However, stimulation of the secretion of renin by the ultrafiltration is in constant. [1] C.Robert et al studied the response of plasma aldosterone and cortisol response to volume depletion in the chronic

and in recently nephrectomized subjects anephric subjects, and found that the increase in plasma aldosterone during the ultrafiltration was seen only in recently nephrectomized patients [7].

b) The sympathetic hyperactivity

Kooman et al showed that there was a sympathetic hyperactivity in chronic hemodialysis characterized by high levels of catecholamines [8]. Izzo and camps suggest that the decrease in preload and venous return are responsible for the hyperactivity through cardiopulmonary baro - reflex [25]. However it should be noted that the level of circulating catecholamines is not systematically associated with intra-dialytic hypertension [1]. KJ Chou et al found in a study that physiological changes in patients with intra-dialytic hypertension could not be explained by sympathetic stimulation which shows the need for further studies. [5]

c) Other vasoactive substances

Other vasoactive substances may be involved 1. EM. EL- Shafey et al have shown that the hypotension and hypertension (intradialytic) could be explained by changes of endothelin [8]. DOMINIC SC et al suggested that the imbalance between endothelin and NO could contribute to an increase in peripheral resistance and a paradoxical hypertension [9]

2) The volume expansion:

The hypervolemia is considered the first case of hypertension in dialysis patients [1]. Leypoldt et al have objectified the decrease in plasma volume was accompanied by a decrease in systolic and diastolic blood pressure [11]. Several authors have shown that intra-dialytic hypertension may be secondary to hypervolemia due to an underestimation of baseline weight. [4] In our study, the ratio of the weight taken on the basis weight was lower in case of intra-dialytic hypertension which is in favor of the under-estimation of dry weight.

3 **) The increase in cardiac output and Frank Starling law :**

Guan et al suggest that intra-dialytic hypertension is due to an increase in cardiac output especially in patients with cardiac dilation cavities [11]. This could be explained by changes in myocardial contractility depending on the size of the expansion (Frank Starling law) [11]. According to this law the increase initial stretching of myofibrils in diastole results in an increase of force of contraction during systole. And the stroke volume increases with preload and therefore blood volume to a certain level or there is a decrease in contractility despite the expansion (or curve) explained by the theory of sliding of actin filaments and myosin [12]

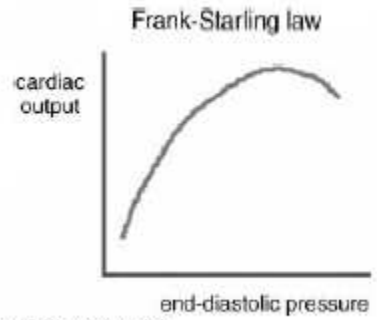


FIG. 1. Frank-Starling curve

The presence of heart disease was a risk factor for intra-dialytic hypertension in our study.

4) Other causes of intra-dialytic hypertension:

Other causes have been described to explain the hypertension, we quote:

- Purification of antihypertensive drugs during dialysis [2]
- An increase in viscosity secondary to hemoconcentration and use of erythropoiesis stimulating agents [14.15]
- Some electrolytic movement hypernatremia, hypokalemia stimulates the renin and hypercalcemia which increases myocardial contractility [16.17]

Prognosis:

The prognosis is not well understood. However Zager et al found that the cardiovascular risk is greater in patients with higher numbers tensionels post dialysis [18.19].

Treatment:

Intra-dialytic hypertension may be too high so that it can cause a reluctance on the part of practitioners that grows to lower ultrafiltration see stop the session treatment [4] , hence the importance of anticipate this complication and provide appropriate therapy . However, few studies have focused on the evaluation of management strategies of intra-dialytic hypertension; strategies that are based in most cases on the opinions of experts [20].

1 Optimization of ultrafiltration. :

As recommended KDOQI , The paradoxical increase in blood pressure secondary to ultrafiltration could be caused by the increase in cardiac output mediated by hypervolemia and can be treated by increased depletion , which should be done with care [21] . Chou et al suggest that the optimization of basis weight is proposed to be effective common strategy that allows a tensionel control [5]. This strategy was effective in 75% of our cases.

2 Inhibition of the renin-angiotensin system. :

G.Bazzato et al suggest that the use of inhibitors of angiotensin converting enzyme is useful in preventing this complication. [6]

3 The decrease in sympathetic hyperactivity. :

O.Zilich et al suggest that sympathetic overactivity may be reduced by increasing the frequency of dialysis sessions which allows better control of blood pressure and a decrease in arterial resistance [22]. The use of adrenergic blocking agents can be discussed even more that they could play a role in reducing cardiovascular morbidity and mortality [8].

4 Adaptation of dialysate. :

To our knowledge no study has evaluated the role of sodium profiles in the prevention of intra-dialytic hypertension. However on the hemodynamic tolerance, studies are contradictory; Straver et al have shown that they are associated with hemodynamic stability, while Song et al found no significant difference [23.24]. In our patients using a sodium descending profile associated with optimizing the ultrafiltration or inhibitors of angiotensin converting enzyme yielded satisfactory results. Our statistical analysis confirms that the sodium profiles significantly influence the risk of developing this complication.

CONCLUSION

The intra-dialytic hypertension is a rare complication, its pathophysiology is poorly understood and its treatment is not codified mainly based on optimizing the basis weight that should be done with

caution. Further studies are needed to assess the epidemiology, pathophysiology and management of this complication.

REFERENCES

[1]. Chen J, Gul A, Sarnak M (**2006**): Management of intradialytic hypertension: the ongoing challenge. Semin Dial 19:141–145

[2]. Amerling RCG, Dubrow A, Levin N, Osheroff R: Complications during hemodialysis. In: Nissenson A, Gentile D (eds). Clinical Dialysis. Stamford, CT: Appleton & Lange, **1995**:236–267

[3]. Fellner S: Intradialytic hypertension II. Semin Dial 6:371–373, **1993**

[4]. Cirit M, Akcicek F, Terzioglu E, Soydas C, Ok E, Ozbasli CF, Basci A, Mees EJ: "Paradoxical" rise in blood pressure during ultrafiltration in dialysis patients.Nephrol Dial Transplant 10:1417–1420, **1995**

[5]. Chou K-J et al :Physiological changes during hemodialysis in patients with intradialysis hypertension. Kidney Int. **2006** May;69(10):1710-1.

[6]. Bazzato G. Prevention of intra- and post-dialytic hypertensive crises by captopril. Contr Nephrol **1984**; 41: 292-98

[7]. C. ROBERT COOK : Hormonal responses to acute volume changes in anephric subjects Kidney International, Vol. 23 (**1983**), pp. 71—78

[8]. Koomans HA, Blankestijn PJ, Joles JA: Sympathetic hyperactivity in chronic renal failure: a wake-up call. J Am Soc Nephrol 15:524–537, **2004**

[9]. E. M. El-Shafey et al. Is there a role for endothelin-1 in the hemodynamic changes during hemodialysis? Clin Exp Nephrol (**2008**) 12:370–375

[10]. DOMINIC S.C et al. Hemodynamic changes during hemodialysis: Role of nitric oxide and endothelin Kidney International, Vol. 61 (**2002**), pp. 697–704

[11]. Leypoldt JK, Cheung AK, Delmez JA, Gassman JJ, Levin NW, Lewis JA, Lewis JL, Rocco MV: Relationship between volume status and blood pressure during chronic hemodialysis. Kidney Int61:266–275, **2002**

[12]. Gunal AI, Karaca I, Celiker H, Ilkay E, Duman S: Paradoxical rise in blood pressure during ultrafiltration is caused by increased cardiac output. J Nephrol 15:42–47, **2002**

[13]. Rémi NEVIERE. Contraction cardiaque et loi du cœur de Starlin *www.lille.inserm.fr/download.asp?download.../rneviere.pdf*

[14]. Neff MS, Kim KE, Persoff M, Onesti G, Swartz C: Hemodynamics of uremic anemia. Circulation 43:876–883, **1971**

[15]. Raine AE, Roger SD: Effects of erythropoietin on blood pressure. Am J Kidney Dis 18 (4 suppl 1):76–83, **1991**

[16]. Locatelli et al. Optimal composition of the dialysate, with emphasis on its influence on blood pressure Nephrol Dial Transplant (**2004**) 19: 785-796.

[17]. JC Maynard, C Cruz, M Kleerekoper. Blood pressure response to changes in serum ionized calcium during hemodialysis.annals of internal medicine. Volume 104: 358-61 **1986**

[18]. Zager PG, Nikolic J, Brown RH et al (**1998**) ''U'' curve association of blood pressure and mortality in hemodialysis patients. Medical Directors of Dialysis Clinic, Inc. Kidney Int 54:561–569.

[19]. Emanuela Rizzioli et al. Management of intradialytic hypertension: old problem, old drug?. Intern Emerg Med (**2009**) 4:271–272.

[20]. F. Locatelli. The growing problemn of intradialytic hypertension. Treatment approaches. Renal week **2008**. Philadelphie

[21]. K/DOQI clinical practice guidelines for hemodialysis adequacy. guideline 5. Am J Kidney Dis **2006**.

[22]. Zilch O et al. Sympathetic hyperactivity in haemodialysis patients is reduced by short daily haemodialysis.J Hypertens **2007**; 25: 1285-89.
[23]. Straver et al,.The effect of profiled hemodialysis on intradialytic hemodynamics when a proper sodium balances applied Blood Purif **2002**, 20 : 364-369.
[24]. Song et al. Effect of Sodium Balance and the Combination of Ultrafiltration Profile during Sodium Profiling Hemodialysis on the Maintenance of the Quality of Dialysis and Sodium and Fluid Balances. JASN **2005**, 16 : 237-246.
[25]. Izzo JE Jr, Campese VM: Hypertension and renal disease. In: Brenner BM (ed).Brenner and Rector's The Kidney. Philadelphia: WB