

Case Report

First-Time Severe Intradialytic Hypotension and Increased Heart Rate Induced by Massive Gastrointestinal Hemorrhage: A Case Report

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Submitted: 11 January 2016

Accepted: 25 January 2016

Published: 27 January 2016

ISSN: 2379-0652

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Keywords

- Gastrointestinal ulcer
- Heart rate
- Intradialytic hypotension
- Massive gastrointestinal hemorrhage
- Risk factor

Abstract

A 69-year-old man with chronic hemodialysis (HD), who had end-stage renal disease secondary to diabetic nephropathy and had been on HD for 4 years, presented with first-time severe transient hypotension (60/32mmHg, base 140/65mmHg), elevated heart rate (90 beats/min, base 75 beats/min) and nausea during HD, recovered after premature termination of HD and bolus of 0.9% saline (400ml in total), except increased heart rate (90 beats/min), turned out to be massive gastrointestinal bleeding (large unshaped dark stools, occult blood positive), with diagnosis of ulcer. This case illustrates the importance to emphasize the first-time severe intradialytic hypotension and elevated heart rate, especially when no reasonable pathology exists. In addition, risk factors may give some clues to diagnose the gastrointestinal ulcer in HD patients, such as diabetes, coronary artery disease and use of NSAIDs.

INTRODUCTION

Intradialytic hypotension (IDH), as a serious problem, occurs in 15%-60% of the treatments [1], causing symptoms such as nausea, dizziness, cramps, fatigue and weakness, which significantly diminishes patient's quality of life and tolerability to dialysis. It possibly also necessitates premature termination of the session [1], limits fluid removal, which leads to volume overload and interferes with the delivery of an adequate dialysis dose. The primary etiologic factor of it seems to be the reduction of circulating blood volume due to massive ultrafiltration (UF) and sodium removal and subsequent imbalance between UF rate and plasma refilling rate. As IDH occurs so common during hemodialysis, transient hypotension is tend to be reported as a result of reasons above.

We present a case of 69-year-old man, who developed transient severe IDH but recovered instantly after treatment with increased heart rate left for the first time since initiating dialysis therapy 4 years ago, revealed to be gastric bleeding. To our knowledge, such case was rear reported, and no detailed data given, we herein discuss the clinical problems associated with

this case and emphasize that physicians should be cautious when treating such patients.

CASE REPORT

On November 12, 2015, in the afternoon, a 69-year-old man on chronic hemodialysis (HD) with predialysis blood pressure 140/65mmHg and heart rate 75 beats/min presented with nausea, hypotension 60 min after starting HD (Table 1). He had end-stage renal disease (ESRD) secondary to diabetic nephropathy and had been on HD for 4 years with an ultrapure dialysis solution schedule of 3 sessions per week. He had neither past history of hemorrhagic tendency nor peptic ulcer. A routine 2000 unit low molecular weight heparin (LMWH, enoxaparin) was used during HD. He had had diabetes mellitus for 15 years and hypertension for 10 years, cerebral infarction for 4 years with left limb paralysis, the implantation of cardiac pacemaker for 4 years for sinus bradycardia. Her only medications were aspirin enteric-coated tablet, levocarnitine and recombinant human erythropoietin (rhEPO). His usual predialysis blood pressure was 120-150/55-80 mmHg, heart rates 88-90 beats/min. On October 27, 2015, he had undergone a conventional blood analysis, which is done once monthly in chronic HD

Table 1: Blood pressure and heart rate measurement during hemodialysis sessions.

	Baseline	60min	120min	180min	End
Previous treatment (no IDH, n = 30)					
SBP(mmHg)	127.32±14.24	125.25±14.09	124.75±14.37	119±11.19	111.25±9.30
DBP(mmHg)	63.51±8.49	62±7.50	64±5.76	62.25±8.35	59.25±6.13
Heart rate(bpm)	75.67±2.02	76.23±3.14	75.38±2.23	75.89±2.16	74.87±2.94
IWG(kg)	1.62±0.52				
UF rate(mL/h)	494.36±121.57				
This session (IDH, n = 1)					
SBP(mmHg)	140	60(Pret)	127(after- Pret)	129(after- Pret)	125(after- Pret)
DBP(mmHg)	65	32(Pret)	50(after- Pret)	55(after- Pret)	57(after- Pret)
Heart rate(bpm)	75	90(Pret)	90(after- Pret)	88(after- Pret)	90(after- Pret)
IWG(kg)	1.2				
UF rate(mL/h)	375				
Abbreviations: IDH: Intradialytic Hypotension; Pret: Premature Termination; After- Pret: After Premature Termination; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; IWG: Interdialytic Weight Gain; UF Rate: Ultrafiltration Rate					

patients. Laboratory values showed: Hb 100 g/L, Hct 32.0%, WBC 7.5×10^9 /L, PLTs 124×10^9 /L. An initial physical examination revealed that he suffered from pale face, sweating. However, pathological signs of the nervous system were unremarkable. His vital signs were: blood pressure 60/32 mmHg, respiratory rate 20 breaths/min, and heart rate 90 beats/min. His blood glucose concentration was 5.9 mmol/L by glucometer. The ultrafiltration rate was immediately reduced to zero and a bolus of 0.9% saline (250ml) was rapidly administered. After the above-mentioned treatment, his blood pressure returned to 115/47 mmHg and nausea disappeared. However, he then presented with another episode of hypotension (80/48 mmHg) after HD ultrafiltration was resumed. HD was stopped and his BP returned to normal (BP 115-129/47-60mmHg, heart rates 88-90 beats/min) again after a bolus of 0.9% saline (150 ml).

His electrocardiogram showed sinus rhythm, no ventricular pace(VP), with serum TNI, CK-MB all normal, but serum Hb, HCT and PLTs were 88 g/L, 29.0% and 71×10^9 /L, respectively which were lower than 2 weeks earlier. This patient refused to be hospitalized and left hospital despite told bleeding suspected and close monitoring needed by our staff.

On the early morning of November 13, 2015, this patient complained of large unshaped dark stools (total volume 1500g), was hospitalized. On admission, his stool was examined urgently, and occult blood was positive. On admission, physical examination was unremarkable, except for pallor and upper abdominal pain. His blood pressure was 113/63mmHg, and pulse was 90 beats/min in regular sinus rhythm. Laboratory findings on hospitalization were as follows: RBC 2.0×10^{12} /L, Hb 56 g/L, Hct 9.0%, PLTs 104×10^9 /L and WBC 10.5×10^9 /L. Serum Na was 145.2 mmol/L, K 5.86 mmol/L, Cl 113.0 mmol/L, Bun 47.32mmol/L, Cr 607 mmol/L, Alb 30.3g/L and TC 2.24 mmol/L, TG 1.28 mmol/L. Serum liver biochemistry, amylase, lipase and blood coagulation function were normal. Ultrasound of the upper abdomen revealed a normal gallbladder, liver and spleen. Upper gastrointestinal endoscopy showed an deep ulcer in the antrum of the stomach with a 3- to 4-mm diameter along with mucous

hyperemia and mucosal edema around the ulcer. Multiple antral biopsies were taken and there was no evidence of malignancy. A Campylobacter-like organism test for Helicobacter pylori yielded a negative result.

DISCUSSION

Intradialytic hypotension (IDH), one of critical intradialytic complications, is an independent risk factor for mortality in patients with end-stage renal disease [2]. Up to 30% of HD patients are estimated to require interruption of HD on account of development of HD-induced hypotension. Until now, how IDH is defined and reported varies in the literature. Variation results from different blood pressure (BP) thresholds at which hypotension is deemed to occur and inconsistency whether associated symptoms such as nausea, vomiting, dizziness, and cramp are a prerequisite for a diagnosis of IDH. David *et al.* suggested that a reduction in SBP by >20% from baseline to <100mmHg (or a reduction n SBP by >20% alone in those with a baseline SBP <100mmHg) used to define those at risk of intradialytic hypoperfusion injury, regardless of symptoms [3]. Our patient was diagnosed as IDH, supported by intradialytic BP 60/32 mmHg, which was a 57.1% reduction in SBP from baseline (BP 140/65mmHg), and nausea, which was hypotension associated symptom.

The main pathogenesis of IDH has been reported to be acute decrease in extracellular space and blood volume during ultrafiltration and inadequate compensatory mechanisms such as left ventricular dysfunction, inappropriate plasma refilling [1], and impaired vasoconstriction, induced by dysregulation of sympathetic nervous activity [4].The relationship between the percentage of blood volume depletion and blood pressure response is depicted in Table 2 [5]. As total blood volume decreases, blood pressure drops, the heart rate increases along with more forceful myocardial contractility in an attempt to restore blood pressure to a normal physiologic state. Obvious variations of blood pressure and heart rate had not been observed in our patient during previous HD sessions, except the latest one. During this HD session, IDH occurred after 60min,

Table 2: Volume Depletion and Blood Pressure Response.

Volume depletion	Blood pressure response
5% to 10% loss	Minimal changes in blood pressure
12% to 20% loss	Symptomatic hypotension
20% to 30% loss	Early reversible shock
30% to 40% loss	Shock, potentially irreversible

while heart rate increased with hypotension symptom (nausea), so volume depletion was suspected. After termination of HD and bolus of 400ml, our patient did not complain of nausea, dizziness, abdominal pain, melena and hematemesis, with no orthostatic hypotension observed. It was worth calling that heart rate of our patient still fluctuated between 88 and 90 beats/min, which raised the possibility for bleeding, while no massive ultrafiltration, diarrhea, vomiting or profuse sweating during HD. Unfortunately, our patient refused to receive further observation, and did not refer to us until next day with complaint of dark stools, when gastric ulcer diagnosed. Thus, we certainly had missed a clinically opportunity to preventing our patient from risking his life of massive bleeding. We therefore conclude that episode of IDH and variation of heart rate in such patient should be emphasized.

Patients on long-term HD have gastrointestinal bleeding events (GIB) at a considerably high rate of 6 events per 100 person-years [6], and a higher rate of bleeding after the development of ulcers [1]. Hemodialysis is a predisposing factor for gastroduodenal mucosal lesions [7]. Diabetes, coronary artery disease [8], abnormalities in blood coagulation [9], cirrhosis and use of NSAIDs [10] were risk factors for ulcer bleeding in these patients [11]. Our patient did not have any gastrointestinal symptoms, except gastric ulceration. Endoscopy confirmed the presence of a significant mucosal injury. There was no personal or family history of ulcer disease. There were, however, some clues with respect to the gastrointestinal hemorrhage in this patient, such as HD, diabetes, coronary artery disease and use of NSAIDs and heparin. It is important for our stuff to be familiar with risk factors of gastrointestinal ulcer and bleeding which may provide a clue of diagnosis in the event of IDH.

In summary, our patient presented with first-time transient IDH during 4-years HD, recovered after treatment, except increased heart rate, turned out to be massive gastrointestinal bleeding, with diagnosis of ulcer. This observation adds to our clinical experience in coping with IDH. It also raises questions

about the importance of heart rate variation to the volume assessment during HD treatment and risk factors to the diagnosis the gastrointestinal ulcer in HD patients.

ACKNOWLEDGMENTS

We thank the investigators, staff, and patient who participated in this case. This manuscript is dedicated to my wife Qin JY.

Supported by grant no.2016ZQ028 from Provincial Bureau of traditional Chinese Medicine, Zhejiang

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Cite this article

Yang LR, Yan QJ, Hong HR, Jin Y, Guoming Y, et al. (2016) First-Time Severe Intradialytic Hypotension and Increased Heart Rate Induced by Massive Gastrointestinal Hemorrhage: A Case Report. *J Clin Nephrol Res* 3(1): 1031.