Oxygen as an Adjunct to Treat Intradialytic Hypotension during Hemodialysis

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Patient Profile

R.L. is a 54-year-old male with chronic kidney disease (CKD) Stage 5 secondary to type II diabetes mellitus. He had four-hour hemodialysis treatments three times a week. Intradialytic hypotension was an ongoing problem. Due to this, R.L. had a physician's standing order for a maximum ultrafiltration (UF) goal of 3,000 mL, since he was "unable to tolerate" greater volume removal. His estimated dry weight (EDW) was 91.5 kg.

R.L. had been monitored with the Crit-Line™ blood volume monitor each treatment over a three-week period. Over that time period, he consistently exhibited a flat slope, with an average blood volume reduction of 0.50% to 0.75% per hour. According to Agarwal (2010), patients with a flat slope, those with a blood volume reduction of less than 1.33% per hour, have a higher risk of mortality. Agarwal (2010) adds that blood volume slope is prognostic of mortality, whereas UF volume and UF rate indices (the UF rate in mL/Kg/hr) are not prognostic of mortality. Moreover, the relationship of the blood volume slope to mortality was independent of conventional (e.g., blood pressure) and unconventional cardiovascular risk factors, such as UF volume and UF rate indices. Based on these findings, R.L. would have a 72% increased risk of mortality due to a flat slope, which is indicative of hypervolemia.

R.L. had a starting hemoglobin (Hgb) of 10.8 g/dL and an arterial oxygen saturation (O_2 Sat) of 82.3% at the start of dialysis. The beginning oxygen saturation value was indicative of an ongoing problem. The blood volume monitor revealed low O_2 Sat levels during three weeks of prior treatments, falling as low as 77%. These numbers are clinically significant because fewer red blood cells are available for binding of oxygen molecules and subsequent delivery to the cells throughout the body.

In an optimal setting, the Hgb will combine with enough O_2 to deliver 20 mL O_2 per 100 mL blood. A calculation was performed on R.L.'s data to determine his hypoxic state (see Table 1).

With a Hgb of 10.8 gm/dL, the patient's oxygen carrying capacity was slightly over 12 mL O_2/100 mL blood. The reduced O_2 carrying capacity due to anemia is the definition of anemic hypoxia (Guyton & Hall, 2000).

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Diroll, D. (2014). Oxygen as an adjunct to treat intradialytic hypotension during hemodialysis. *Nephrology Nursing Journal, 41*(4), 420-423.

Key Words: **Blood pressure, blood volume, oxygen, intradialytic hypotension**

Intended Patient Outcomes

The intended patient outcomes included:

- Minimize/reduce intradialytic hypotension using adjunct O_2 therapy.
- Remove excess volume.
- Establish a new dry weight.
- Reduce mortality risk.

Problem

Intradialytic hypotension was a consistent problem for R.L. and contributed to inadequate fluid removal. Possible consequences of intradialytic hypotension and the lack of action to prevent it are addressed in the following statement, taken from the National Kidney Foundation (NKF) KDOQI Clinical Practice Guidelines for Cardiovascular Disease in Dialysis Patients (NKF, 2005):

> "*Intradialytic hypotension (IDH) is defined as a decrease in systolic blood pressure by ≥ 20 mmHg or a decrease in MAP by 10 mmHg associated with symptoms"* and *"IDH precludes the delivery of an adequate dose of dialysis."*
>
> "*Long-term effects of IDH include volume overload due to suboptimal ultrafiltration and use of fluid boluses for resuscitation; LVH, with its associated morbidity and mortality; and interdialytic hypertension*" (p. 576).

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Table 1
Hypoxic State Calculation

Hgb	10.8 g/dL
Hgb Binding Ability	1.34 mL O_2/g
Arterial O_2 Sat	82.3% or 0.823
Dissolved O_2 (est.)	0.003 mL O_2/dL
Partial Pressure of O_2 (pO_2) (est.)	100 mmHg
O_2 Capacity*	[Hgb Binding Ability x Hgb x (Sat/100)] + dissolved O_2 x pO_2 = mL O_2/100 mL blood [1.34 x 10.8 x (.823/100)] + 0.003 x 100 = 12.2 mL O_2/100 mL

*West, 1985.

In response to intradialytic hypotension, it is standard to administer saline and/or reduce the UF goal. The saline adds to the sodium and water load while decreasing the UF goal and precludes achieving euvolemia. In this instance, it is likely that R.L. is receiving inadequate dialysis, remaining fluid overloaded, and exhibiting dilutional anemia.

Hypothesis

Blood pressure is defined as cardiac output times peripheral vascular resistance. For every increase or decrease in either cardiac output or peripheral vascular resistance, there will be a corresponding increase or decrease in blood pressure (Smith & Kampine, 1990). The occurrence of vasodilation and subsequent reduction in peripheral vascular resistance due to hypoxia has been known for over 100 years (Diesen, Hess, & Stamler, 2008).

Oxygen is necessary for cardiac output and vascular tone, especially since the cardiac muscle is an organ that extracts a high volume of oxygen. Low O_2 supply to the heart muscle causes a decline in function and ability to respond to O_2 requirements throughout the body (Smith & Kampine, 1990).

By supporting cardiac function through the use of adjunct O_2, cardiac output should increase. Supporting cardiac output will help maintain adequate blood pressure and mitigate intradialytic hypotension. If intradialytic hypotension is avoided, a more effective hemodialysis treatment can be accomplished. Euvolemia and cardiac decongestion will also be obtained, further easing the workload of the heart.

Intervention and Outcome

The goal in this treatment was to normalize arterial O_2 Sat through the use of O_2 at 2 L per nasal cannula using a portable oxygen concentrator.

After evaluating the O_2 Sat and treating the hypoxia to target an O_2 Sat of greater than 90%, the patient had plasma refill. This was evidenced by the blood volume increasing from -2.0% to -0.5% within 10 minutes of supplemental O_2, as seen at the 0:40 time mark (see Table 2 and Figure 1). In response to plasma refill, the UF goal was increased from 3,000 mL to 4,900 mL – the goal for patient to achieve his stated EDW of 91.5 kg. With the UF goal change, the slope for R.L. was converted from a flat slope, in previous treatments, to a steep slope, in the current treatment.

R.L.'s blood pressure made progressive improvements toward normotension over the entire run. First, the blood pressure was observed to improve in response to supplemental O_2. Second, the blood pressure was observed to make continued improvements after the UF rate was increased, even as O_2 Sat remained in the target range.

The hematocrit (Hct) increased from 32.3% to 35.1% as a result of the blood volume reduction. The patient's ending blood volume change was -7.3%, which translates to an average blood volume change of -2.1% per hour. This meets Agarwal's (2010) definition of a "steep slope" and a lower mortality risk.

The staff continued to probe for R.L.'s estimated dry weight (EDW) in subsequent hemodialysis treatments over two weeks without episodes of intradialytic hypotension. A reduction of 4.3 kg was reached over that time period. R.L. achieved a new EDW of 87.2 kg. The intended patient outcomes were accomplished.

Discussion

With the adoption of blood volume monitoring, by which O_2 Sat and Hct are measured continually and percent blood volume change is calculated continually, it is possible to ascertain the relationship between blood pressure, blood volume, and O_2 Sat. Further, it is possible to analyze these data and make appropriate interventions to improve outcomes.

For the patient on hemodialysis, O_2 Sat should be an indicator of impending intradialytic hypotension. In their study of red blood cell (RBC) saturation levels, Diesen et al. (2008) found that vasodilation is inversely proportional to O_2 Sat. Thorn, Kyte, Slaff, and Shore (2011) found that a decrease in O_2 Sat of about 7% triggers a vasodilatory response. In other words, O_2 Sat affects the level of vasodilation and subsequently intradialytic hypotension.

Table 2
Blood Volume Monitor Guided Interventions

Elapsed Time	Blood Pressure	Pulse	Blood Volume Change	Oxygen Saturation	Intervention
0:00	112/63	61	0.0%	82.3%	HD commenced
0:30	86/46	62	-2.0%	85%	O_2 at 2L per nasal cannula
0:40	97/51	62	-0.5% (refill)	94%	UF Goal increased from 3.0L to 4.9L
1:00	108/51	65	-1.5%	94%	
1:15	118/56	66	-2.0%	94%	
1:30	140/72	69	-2.0%	95%	
2:00	136/78	70	-2.5%	94%	
2:30	125/64	71	-4.5%	90%	
3:00	137/74	71	-5.0%	93%	
3:30	125/68	72	-7.3%	92%	
4:00			-7.3%	93%	HD ended

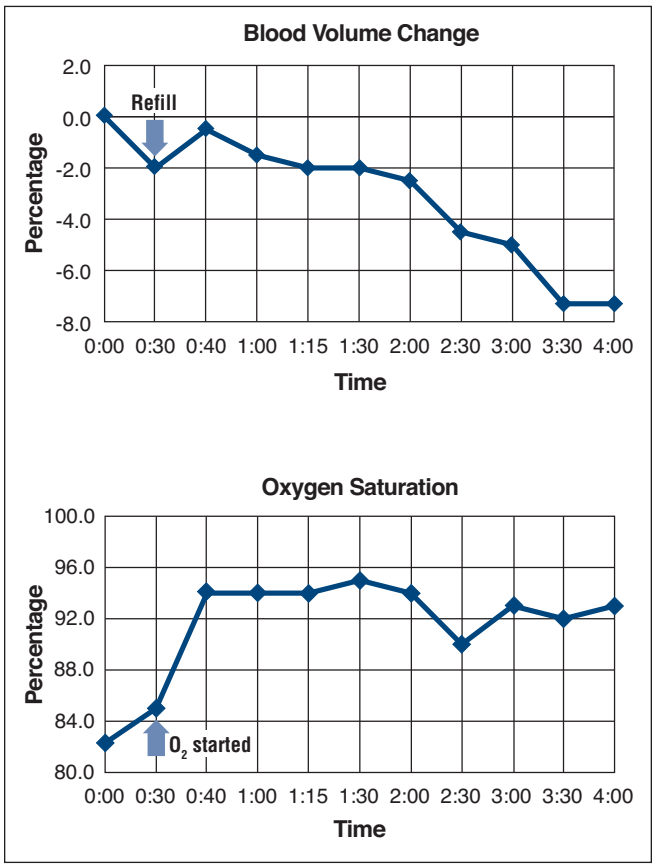
It is unfortunate, in this type of scenario, that a patient with low O_2 Sat is treated for the *symptom* of intradialytic hypotension, rather than the *cause* of intradialytic hypotension.

In a typical scenario at the chronic hemodialysis clinic, declines in blood pressure (or intradialytic hypotension) are usually met with a saline "challenge" or bolus. The rationale for this action is that the blood pressure declines as a result of low blood volume. This line of thinking would make sense in a static system, a system that could not dilate, constrict, diminish, or refill. But the vascular tree is anything but static. Muscles line the system throughout the arterial side of the tree all the way down to the arterioles. Plasma proteins and other metabolites affect water movement to and from the vascular tree. These, in turn, have an impact on fluid volume in the intravascular space, interstitial space, and the intracellular space. In effect, water dynamically moves among the three compartments (Ahmad, 1999).

The point of hemodialysis is to remove fluids. It is unfortunate for a patient who is fluid-overloaded to receive more fluids in response to intradialytic hypotension. In a vicious cycle, intradialytic hypotension is accompanied by a decreased ultra filtration rate and a larger blood volume. Fluid overload in patients on hemodialysis has been shown to have an inflammatory affect and leads to erythropoietin stimulating agent (ESA) resistance. ESA resistance exacerbates anemia. Anemia leads to hypoxia.

Pulmonary problems are common, and hypoxemia occurs in nearly 90% of patients during hemodialysis (Gheuens, Daelemans, & De Broe, 2000). Hypoxemia may cause significant morbidity in patients with previously compromised cardiopulmonary status and may contribute to intradialytic hypotension, nausea, and muscle cramps in others (Gheuens et al., 2000). Gheuens and col-

Figure 1
Relationship Between Oxygen Saturation and Vascular Compartment Refill



leagues (2000) note that the utilization of supplemental oxygen (FiO_2 of 28%) is one measure to be taken to minimize the risk of hypoxemia in patients on dialysis.

A second measure encouraged by Gheuens and colleagues (2000) is the optimization of Hct. Because blood volume and Hct are inversely related, Hct can be increased by achieving optimal blood volume reduction. Importantly, Anand, Sinha, and Agarwal (2012) have shown improved O_2 Sat with increasing Hct.

Conclusion

Hypoxia is a common problem in patients on hemodialysis and is known to cause vasodilation. Vasodilation causes intradialytic hypotension. Intradialytic hypotension precludes the optimal removal of fluid and achieving euvolemia. By treating known hypoxia with supplemental O_2, cardiac output is supported, which assists in the maintenance of adequate blood pressure and mitigation of intradialytic hypotension.

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