Intradialytic hypotension: a literature review

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Abstract

Background Intradialytic hypotension (IDH) is an acknowledged problem within haemodialysis care settings. Whilst the causes are recognised to be multifactorial, likewise, no one solution exists to combat or prevent the problem.

Aim The aim of this paper is to present the findings of a literature review into IDH, from medical and nursing perspectives. The intent was to identify pathological risks behind IDH that impact on the dialysis process, in reviewing current opinion into the best strategies — based on research studies where possible — in its prevention and treatment.

Method A systematic review of literature was performed via EBSCOhost, utilising databases relevant to nursing, medicine, the social sciences, health and research.

Results The information within medical literature regarding IDH was grouped into four categories. These reviewed pathophysiology of IDH in explaining potential long-term consequences; current interventions with specific machine technologies and preventative recommendations based on pharmacological administrations and dialysate electrolyte alterations. Nursing literature reviewed physiology and suggested interventional strategies.

Conclusions Recent papers allude to increasing evidence that repeated episodes of symptomatic as well as asymptomatic IDH contribute to adverse outcomes for the dialysis population. The lack of clarity in offering a succinct definition for IDH possibly hampers the effectiveness of preventative strategies. An individualised approach in ascribing acceptable blood pressure ranges for each patient — with recommended intervention levels — may go a long way to improving outcomes for haemodialysis patients.

Background

In the provision of haemodialysis for people with end-stage renal disease the most frequent and troubling side effect is hypotension. Its symptoms include nausea, vomiting, diaphoresis and cramps, to more dangerous conditions including angina, arrhythmias, unconsciousness, seizures, and cardiac arrest (Burton *et al.*, 2009; Chesterton *et al.*, 2010; Sherman, 1988).

Intradialytic hypotension (IDH) has been estimated to occur between 15 and 55% of all treatments (Bradley *et al.*, 1988; Calvo *et al.*, 2002; Damasiewicz & Polkinghorne, 2011; Daugirdas, 2001; Davenport, 2011; Dheenan & Henrich, 2001; Nakamura *et al.*, 1991; Palmer, 2009; Sherman, 1988) and with an ageing population exhibiting greater comorbidities such as diabetes and cardiovascular disease (Guzon & Dellsperger, 2006), the problem requires considerable rethinking and innovation.

Theoretical framework

The framework surrounding this literature review was the Knowledge to Action (KTA) framework. The KTA framework commences from a point of problem-based knowledge inquiry which is condensed through knowledge synthesis. Knowledge synthesis is a gathering of information pertaining to the identified problem, and the process used in this instance is the literature review itself. A further third-generation process will result in a knowledge product or tool, and Graham suggests that "the purpose of these tools is to present knowledge in a clear, concise and user friendly format ... thereby facilitating the uptake and application of knowledge" (Graham *et al.*, 2006, p. 19). Hopefully this framework will ensure enhanced information "becomes distilled and refined ... more useful to the stakeholders" (Sudsawad, 2007).

Methods

The search strategy had the intent of sourcing local and international nephrology literature. Consideration was given to the fact that nurses are the immediate carers within the haemodialysis unit, so nurse knowledge of blood pressure was used as additional search criteria.

The systemic review was performed via EBSCOhost, utilising databases relevant to nursing, medicine, the social sciences, health

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and research. Databases included: Academic search complete, CINAHL complete, Health source: Nursing/Academic edition, Medline complete, Medline with full text, ERIC, E-journals, Philosophers index and Psychology and behavioural sciences collection. The only exclusion criteria were for languages other than English.

The terms "h(a)emodialysis" and "hypotension" were variably mixed with "nursing", "blood pressure", "intradialytic hypotension", "diastolic", "systolic", "mean arterial pressure", and "MAP" to elicit nearly 1200 articles. These were scanned for relevance and on a subsequent occasion the terms "nurse", "knowledge" and "perceptions" were entered, which yielded 850 articles. The reference list of each article was examined to identify additional relevant sources. A final collection of 75 relevant articles were analysed.

Literature review findings

There was a considerable amount of information within medical literature regarding IDH, discussing pathological concerns for long-term consequences; technological interventions; preventative pharmacological agents and alterations to dialysate electrolyte components. A smaller selection of nursing literature outlined preventative and interventional strategies.

Definitions of IDH

IDH is defined as either "a decrease in systolic blood pressure by >20 mmHg or a decrease in MAP by >10 mmHg associated with symptoms" (Kooman *et al.*, 2007; National Kidney Foundation, 2006) or "a fall of systolic BP below 100 mmHg and a fall in diastolic BP of at least 20 mmHg with symptoms" (Calvo *et al.*, 2002). It is additionally suggested that:

"Dialysis hypotension may occur in one of three clinical patterns: (i) acute (episodic) hypotension, defined as a sudden drop of systolic blood pressure below 90 mmHg or of at least 20 mmHg with accompanying clinical symptoms; (ii) recurrent, as detailed by prevailing in a minimum of 50% of dialysis sessions and (iii) chronic, persistent hypotension in which interdialytic systolic blood pressure is maintained at less than 90–100 mmHg" (Sulowicz & Radziszewski, 2006).

Some authors contrive their own definitions. In a study designed for the prevention of dialysis hypotension, the above definition is further expanded to explain "... an abrupt decrement ... of greater than 40 mmHg in systolic blood pressure or greater than 20 mmHg in diastolic blood pressure was considered a significant intradialytic hypotensive change" (Dheenan & Henrich, 2001). In another paper concerning impaired baroreflex sensitivity, "IDH was classified as systolic blood pressure (SBP) <100 mmHg, even in the absence of symptoms, or a fall in SBP >10% of the pre-dialysis reading in association with any symptom classically associated with hypotension" (Chesterton *et al.*, 2010). An additional project investigating hypertonic fluid administration for IDH stated the condition was "defined as a decrease in SBP to <100 mmHg during dialysis, with a decrease in SBP of at least 20 mmHg from the pre-dialysis value" (Shimizu, Kurosawa & Sanjo, 2008).

Impact of pathophysiological processes

Nearly all the literature concedes that the processes contributing to IDH are multitudinous and patient-dependent.

In considering IDH, Sherman suggests the importance of re-examining the determinants of mean arterial pressure (MAP), as derived from peripheral vascular resistance (PVR) and cardiac output, reminding that the reduction of plasma volume subsequent to ultrafiltration (UF) "will result in hypotension unless compensatory changes occur in PVR, heart rate or myocardial contractility" (Sherman, 1988).

The autonomic nervous system and its regulation of blood pressure via baroreceptor reflex control is significant in IDH. "Autonomic dysfunction is important in the pathogenesis of the complex hemodynamic abnormalities that exist in CKD patients" (Chesterton *et al.*, 2010). These authors suggest that haemodialysis may result in heart failure, which "is associated with sympathetic overactivity and impaired baroreflex sensitivity (IBRS) ... an accepted marker of autonomic dysfunction" (Chesterton *et al.*, 2010, p. 19). They compared groups of patients who they classified as either hypotensive resistant (HR) or hypotensive prone (HP) with normal or impaired baroreflex sensitivity and suggest "early recognition of those patients prone to relative symptomatic and asymptomatic hypotension remains important" (Chesterton *et al.*, 2010).

Other authors constructing studies around HP and HR patient classification, in regard to tolerance of blood volume changes, suggest that HP patients show a "severe and permanent reduction of systemic arteriovenous tone ... (which leads) to a poor cardiovascular compliance ... with a high frequency of hypotensive episodes" (Graziani *et al.*, 2010).

Daugirdas states that some vasoactive substances have vicious cycle effects on vascular tone: a sudden drop in arterial pressure elicits IDH, this induces localised tissue ischaemia resulting in the release of adenosine, a by-product of the ATP-AMP cycle. Adenosine inhibits the actions of norepinephrine, resulting in vasodilation which worsens the hypotensive episode. Additionally, "at any level of cardiac output, the level of arteriolar tone will determine the level of central blood pressure" (Daugirdas, 2001).

There is an important reminder that haemodialysis as a treatment modality itself causes considerable physiological burden to a patient's vascular system. In discussing specific physiological injuries hypotensive episodes may cause, it is proposed that " recurrent injury is critically dependent on intradialytic relative hypotension (not just conventionally defined hypotensive episodes) and the rate of UF" (McIntyre, 2010). Haemodialysis as a cardiovascular stressor has adverse

effects on key organs, including the heart, brain and gut. Within the brain, MRI have shown pathological findings including the presence of leukoariaiosis, "a nonspecific change in the brain white matter caused by loss of axons and myelin because of ischemic injury" (McIntyre, 2010, p. 449) which is thought to increase vascular ageing. Additionally, "This form of subcortical injury occurs precisely ... where episodic intradialytic reduced perfusion would be expected to have its maximal effect" (McIntyre, 2010, p. 449).

The suggestion of correlations between haemodialysis and reduced myocardial blood flow, characterised by left ventricular regional wall motion abnormalities (RWMAs) has been confirmed via echocardiography performed during the haemodialysis procedure, termed cardiac stunning (Burton *et al.*, 2009; Chesterton *et al.*, 2010; Dorairajan, Chockalingam & Misra, 2010). In recalling that the cardiac muscle receives its coronary blood supply during diastole (Hall & Guyton, 2011, p. 246), and that the majority of haemodialysis patients have a widened pulse pressure characterised by a lower diastolic blood pressure value, it may not seem surprising that "episodic intradialytic hypotension may be involved in the pathogenesis of evolving myocardial injury" (Owen *et al.*, 2009).

Elevated levels of endotoxins in dialysis patients (compared to patients with earlier stages of chronic kidney disease) are thought to be correlative to recurrent IDH episodes (Agarwal, 2012). McIntyre explains that "Bacterial endotoxin is a major lipopolysaccharide and the major glycolipid component of the outer membrane of gram-negative bacteria, which comprise 70% of the total bacteria in the healthy human gut" (McIntyre *et al.*, 2011) and its role in IDH is problematic.

"There is evidence that life-threatening conditions such as non-occlusive mesenteric ischemia are associated with frequent IDH" (Daugirdas, 2001) due to repeated ischaemic injury to vulnerable vascular beds during haemodialysis, with reduced splanchnic blood volume. Mesenteric ischemia, (with endotoxins introduced into the circulation) may finally result in "peripheral vasodilation and reduction in cardiac contractile performance" (McIntyre *et al.*, pp. 133–4). Critically, "this degree of dialysis induced 'gut stunning' occurs in haemodialysis patients and this phenomenon may be an important integrating component in the pathophysiology of inflammation, malnutrition and adverse CV outcomes" (McIntyre, 2010).

Review of technological interventions

Blood volume monitoring (BVM): The principle behind this real-time intervention is that relative blood volume, at any given time, reflects a comparative value to the patient's volume state at commencement of dialysis (which is 100%). The BVM monitors alterations in haematocrit and specific components inform when the UFR exceeds, is equal to or is lesser than the refill rate, and the progress of fluid removal is displayed graphically. It should be remembered through the course of the treatment that "it is the slope of the RBV curve rather than the absolute value that can provide information about the patient's haemodynamic stability" (Damasiewicz & Polkinghorne, 2011) inasmuch as the more volume-overloaded the patient, the more refill will be evident on the curve of the graph — especially if refill returns to or exceeds the initial 100% volume. Conversely, the steeper the curve, the greater the fluid removal rate compared to refill. Additionally, "there is no predetermined level of blood volume reduction required to precipitate hypotension ... (however, this) demonstrates low intra-individual variability ... therefore individual predictive models may be developed" (Lewicki, Kerr & Polkinghorne, 2013). The relative blood volumes and minimum relative blood volume (mRBV — the lowest blood volume value a patient reaches in one session) may be recorded at intervals over successive treatments to elicit individual patient patterns, although concession is made that "there is limited evidence that BVM can predict IDH in individual patients" (Damasiewicz & Polkinghorne, 2011) and published trials report varied outcomes (Damasiewicz & Polkinghorne, 2011; Davenport, 2011; Santoro *et al.*, 2002).

Intradialytic temperature controls: During dialysis treatment the reduction in skin and muscle blood flow leads to preservation of central plasma volume, which can result in rising core body temperature increasing the propensity to vasodilation and IDH (Davenport, 2011). Whilst patients are thought to have individual thermal thresholds, and despite the fact that some patients may suffer from shivering and cramping, most are tolerant to slight decreases in dialysate temperature, which counteracts the likelihood of central temperature-related vasodilation (Damasiewicz & Polkinghorne, 2011; Davenport, 2011; Dheenan & Henrich, 2001). Additional machine technology monitoring arterial and venous blood flow via the extracorporeal circuit may be used to feedback to machine thermostats, allowing for regulation of dialysate temperature, although a standard dialysate temperature is usually set. It is suggested that a dialysate temperature of 34–35°C instead of 37°C can improve cardiovascular stability and greatly reduce the incidence of IDH (Damasiewicz & Polkinghorne, 2011).

Ultrafiltration modulation: It is widely recognised that modifying the rate and frequency of UF may minimise the likelihood of IDH, although recommendations vary (Davenport, 2011; Dheenan & Henrich, 2001; Palmer, 2009). With improvement in machine UF technologies it is possible to perform patterned fluid removal over the course of the treatment, with isolated UF pulses, step down rates or UF volumes that progressively decline. Earlier RBV monitoring showed patients had smaller RBV changes at the beginning of dialysis, when more fluid overloaded, and Davenport suggests that several studies indicate IDH frequency is reduced if more fluid is removed at the beginning of the session, gradually decreasing in rate. He suggests that the highest frequencies of IDH occur when utilising UF pulses or steps (Davenport, 2009).

Interestingly, in a study comparing the efficacy of different treatment protocols in preventing IDH, it was concluded that "isolated ultrafiltration followed by isovolemic dialysis was notably less effective in reducing IDH" (Dheenan & Henrich, 2001).

Sodium profiling/modelling: Succinctly, the aim of sodium profiling "is to prevent osmotic disequilibrium ... support plasma refilling ... reduce the incidence of IDH" (Stiller *et al.*, 2001). As a treatment tool it was introduced in recognition of the sudden reduction of urea and small solutes that occurs immediately after dialysis initiation, which results in a fall in plasma osmolality. It was postulated that maintaining (or increasing) dialysate sodium would increase serum sodium concentration, allowing optimal UF and prevention of IDH. A progressive decrease in dialysate sodium through the course of the treatment would limit a positive sodium balance (Davenport, 2009); however, studies and results are variable, and the reputed adverse consequences related to sodium profiling include patients having a higher post-dialysis serum sodium load, resulting in increased thirst and subsequent increase in interdialytic weight gain. Stiller *et al.* suggest that future "application of existing mathematical algorithms for prediction of post-dialysis plasma sodium ...will make sodium profiling more reliable in avoiding sodium accumulation" (Stiller *et al.,* 2001) and another study suggests that the calculation of time average concentrations of serum sodium (TAC Na) may offer a way to prevent sodium load in successfully utilising sodium profiling (Song *et al.,* 2002).

Pharmacological interventions

Several suggestions are made within the literature regarding the potential for administration of pharmacologic agents to prevent, ameliorate or treat IDH.

Caffeine: It may be considered general knowledge that ingestion of caffeine (chiefly through coffee and tea consumption) raises blood pressure, at least transiently. This is thought to be through stimulation of sympathetic nervous activity and renin-antiotensin system activation (Perazella, 2001). One trial has been carried out to ascertain likely pathways of IDH development in relation to adenosine, in conjunction with the administration of caffeine. Twenty IDH prone patients were enlisted in this prospective placebo-controlled trial in a three-part study. The researchers differentiated sudden hypotension (decrease in MAP to <50 mmHg) from gradual hypotension (decrease in MAP to <70 mmHg but >55 mmHg). The findings showed that caffeine administration (250 mg by capsule) significantly reduced the frequency of sudden IDH episodes, by nearly 50%, compared to the placebo group; however, there was no significant reduction in gradual hypotensive episodes. Whilst also comparatively measuring adenosine metabolites, plasma norepinephrine and plasma renin, the authors suggest "it appears likely that the action of caffeine to prevent sudden hypotension is mediated by the adenosine-receptor antagonism ... (in that) adenosine contributes to the development of sudden hypotension but not to the development of gradual hypotension" (Shinzato *et al.,* 1994). No recommendations for caffeine administration are given.

Midodrine: Midodrine was initially used to control orthostatic hypotension in patients with autonomic neuropathy, by improving venous return through a concomitant decrease of venous pooling and increase in vascular tone. Perazella describes how several studies — including his own — have shown its effectiveness in treating IDH, when administered prior dialysis, for patients with a known history of severe IDH. Studies indicate increase in mean blood pressures throughout and after the dialysis session, with fewer episodes of IDH and decreased need for fluid resuscitation and nursing interventions if IDH occurred (Lim & Yang, 1998; Perazella, 2001). However, caution is made that "because of the long half-life, midodrine should not be dosed more than once per day ... to avoid supine hypertension" (Perazella, 2001).

Fludrocortisone: This mineralocorticoid has also been successful in treating orthostatic hypotension (in persons with normal renal function) by enhancing sodium reabsorption in the distal nephron to "increase body salt content and expand the intravascular space" (Perazella, 2001). For this reason its use in the treatment of IDH in anuric haemodialysis patients has uncertain results; although "it has been postulated that fludrocortisone may raise blood pressure in these patients through an extrarenal hemodynamic effect (perhaps enhanced alpha-1 receptor sensitivity), there appears to be limited evidence of its success in the literature "(Perazella, 2001).

Vasopressin: As an endogenous compound, vasopressin is normally released (under baroreflex control) to conserve blood volume through arteriolar constriction in response to low blood pressure (Hall & Guyton, 2011; van der Zee *et al.,* 2007). "In some forms of hypotension without appropriate vasoconstriction or with frank vasodilation we recently found that the plasma concentration of arginine vasopressin (vasopressin) was inappropriately low" (van der Zee *et al.,* 2007), so these authors performed a randomised, double-blinded placebo-controlled trial in 22 hypertensive patients. The intent was to ascertain whether administration of the exogenous hormone would facilitate fluid removal and they concluded that "inadequate vasopressin secretion during hemodialysis-induced fluid removal is a likely contributor to the IDH that limits fluid removal ..." (p. 318) and "Clinical outcome trials are needed to determine whether prevention of IDH with vasopressin may improve chronic control of ECV in patients with ESRD" (van der Zee *et al.,* 2007, p. 322). Intranasal lysine vasopressin has also been used with limited intradialytic success, although there were concerns for possible coronary artery constriction and localised irritations due to the intranasal administration route (Perazella, 2001).

Hypertonic solution administration: Volume expanders such as Mannitol, Dextran and concentrated albumin may assist in raising blood pressure in episodes of IDH; however, these expensive, hyperoncotic fluids are not without problem (van der Sande *et al.,* 2000). A comparative study reviewing the efficacy of hypertonic saline (3%), albumin (20%) and HES (hydroxyethylstarch — 10%) showed "that HES is an effective solution in maintaining the SBP course, similar to albumin but superior to hypertonic saline" (van der Sande *et al.,* 2000) and the authors expressed concern over reports of increased risk to hypovolemic patients with albumin administration (p. 200).

Another project reviewed the mechanisms behind volume expansion due to administration of hypertonic solutions to treat IDH, which was undertaken to additionally examine "whether arginine vasopressin (AVP) is involved in this mechanism of blood pressure control" (Shimizu, Kurosawa & Sanjo, 2008). The non-randomised trial considered the effects of administering hypertonic saline and glucose as well as AVP infusion for IDH episodes. The results indicated that hypertonic saline (10% saline in 20 ml infused over 5 minutes) increased plasma osmolality, increased (endogenous) AVP plasma levels and mean arterial pressure; with glucose administration (50% glucose in 20 ml given over 2 minutes) yielding similar results. Low and high doses of AVP by infusion were also administered and these increased MAP in both doses. Suggesting that "an abrupt increase in plasma osmolality possibly may cause vasoconstriction of peripheral vessels", the authors go on to state that, "infusions in hypertonic saline or glucose increases blood pressure, plasma osmolality and, in particular plasma AVP levels in IDH ... furthermore, exogenous administration of AVP to similar plasma levels also increases BP in a dose dependent fashion ... ; however, the contribution to plasma volume by the solutions (volume) was too small to increase the BP by itself ... therefore the increase in osmolality is believed to result in increase in BP through both AVP secretion and possible effect on plasma volume" (Shimizu, Kurosawa & Sanjo, 2008, p. 300). In reviewing the trial, Henrich comments, "The observed increase in blood pressure is rapid and could either be due to a direct effect of the change in the plasma osmolality on resistance vessels, the rise in AVP levels or both" (Henrich, 2008).

Dialysate composition

A selection of papers explored the possibility of altering dialysate electrolyte composition to reduce IDH incidence.

Potassium: The excretion of accumulated potassium during dialysis is determined by the amount in dialysate concentrate and the levels, through the treatment period can vary rapidly and significantly. A study examining the effects of actively altering potassium concentration through a dialysis session was carried out with patients randomised into six different dialysis sequences over 12 sessions. Each dialysis session was divided into three equal parts (tertiles) with a differing potassium concentration in each part. The incidence of IDH was recorded (defined as a systolic BP of <90 mmHg) and alterations in haemodynamic parameters were monitored. The results indicated, "significant differences in systolic and mean blood pressure and peripheral resistance ... within the tertiles ... (measures which were all) lower for the tertiles using the dialysate with the lower potassium concentration" (Gabutti, Luca *et al.,* 2011). The authors concluded that, "A rapid decrease in the concentration of serum potassium during the initial stage of dialysis — obtained by reducing the concentration of potassium in the dialysate — translated into a decrease of systolic and mean blood pressure ... (and that) The risk of intra-dialysis hypotension inversely correlates to the potassium concentration in the dialysate" (Gabutti, Luca *et al.,* 2011).

Calcium and bicarbonate: In general, a higher dialysate calcium concentration will lead to higher ionised serum calcium, which is important for vascular and myocardial contractility, although consideration needs to be given to the potential for calcium accumulation and possible, subsequent smooth muscle deposition (Wick & Vijil Jr, 2008).

An early study (1989) examining calcium induced changes in intradialytic blood pressure indicated a positive relationship between systolic blood pressure and ionised calcium levels in eight dialysis patients who underwent three dialysis sessions of differing dialysate calcium amounts. The patients were stable throughout and the authors concluded that the changes in serum calcium affected (mildly increased) systemic blood pressure "through changes in left ventricular output rather than in peripheral vascular tone" (Fellner *et al.,* 1989).

A more recent study considered the changes in systemic haemodynamics due to alterations in calcium and bicarbonate. In this randomised trial (single-blind, crossover design) dialysate bicarbonate and calcium amounts were sequentially changed, over a total of 756 sessions. Bicarbonate ranges were between 26 and 35 mmol/L, and calcium between 1.25 and 1.5 mmol/L. The authors had "previously demonstrated that mild metabolic alkalosis resulting from standard bicarbonate haemodialysis induces hypotension" (Gabutti *et al.,* 2009); however, after acknowledging the inotropic effect of calcium, in their conclusions in this trial they stated, "both high bicarbonate and calcium concentrations in the dialysate improve the haemodynamic pattern during dialysis. Bicarbonate reduces arterial stiffness and ameliorates the heart tolerance for volume overload in the interdialytic phase, whereas calcium directly increases stroke volume ..." (Gabutti *et al.,* 2009) ultimately suggesting alteration in bicarbonate only with the failure of other methods for IDH prevention. Additionally, "in patients who are prone to IDH, avoidance of low dialysate calcium may be of benefit ..." (Palmer, 2009).

Magnesium: The authors of a study investigating the effects of magnesium on intradialytic blood pressure state that

hypomagnesemia is an acknowledged contributor to myocardial ischaemia, which may increase cardiac morbidity and mortality (Kyriazis *et al.,* 2004). Within their trial, they non-invasively measured cardiac index and BP parameters before, during (at hourly intervals) and after dialysis in patients who underwent four treatments of differing magnesium and calcium concentrations. They also recorded episodes of IDH, defining symptomatic hypotension as " the requirement of volume expansion therapy due to either a decrease in SBP of more than 40 mmHg below pre-dialysis SBP, a decrease in SBP below 95 mmHg, or a decrease in BP accompanied by symptoms requiring intervention" and "Asymptomatic hypotension was defined as a SBP of <90 mmHg without symptoms" (Kyriazis *et al.,* p. 1223). As their study included the alteration of calcium as well as magnesium, they concluded that "a dialysate solution containing 0.25 mmol/L Mg and 1.25 mmol/L Ca (is) a significant risk factor for developing IDH and as such it's use should be avoided in IDH prone individuals ... and increasing dialysate Mg to 0.75 mmol/L could prevent IDH frequently seen with the use of Ca 1.25 mmol/L ... (but that) the potential role of dialysate Mg on the cardiovascular system during dialysis requires further investigation"(Kyriazis *et al.,* 2004).

Sodium: In noting that serum and dialysate sodium are not strictly comparable, and that there is great variability in individual patient serum sodiums, Lewicki *et al.*, in reviewing DOPPS data, state "the relationship of dialysate sodium to mortality was serum sodium dependent, with dialysate sodium >140 mmol/L associated with decreased mortality when serum sodium was <137 mEq/l ... and trends seen to increased mortality in those with serum sodium ≥140 mEq/l" concluding that, "overall, lowering dialysate sodium consistently improves IDWG ... and may improve BP although poor dialytic tolerance is noted with higher rates of cramping and IDH" (Lewicki, Kerr & Polkinghorne, 2013).

Nursing literature

Of the nephrology nursing content reviewed, only 10 articles had direct relevance to nurse management of IDH.

These articles discussed the causes of hypotension (Ellis, 2011; Holechek, 2003) and also considered interventions relating to machine technologies, in particular blood volume monitoring and ultrafiltration profiling (Dasselaar, 2007; Yung, 2008). Yung suggested tailoring UF profiling in an individualistic manner, giving advice on how this could be achieved with a specific model dialysis machine (Yung, 2008).

One article discussed an inverse relationship between blood volume and blood pressure in case study format (Dirroll & Hlebovy, 2003), although this ultimately involved the care of a haemodialysis patient within an intensive care unit.

Two journal articles considered correct blood pressure monitoring regarding body location of appliance (Schrauf, 2012; Storck, 1998), these discussions centring around accurate sphygmomanometer cuff placement and technique.

Several papers briefly reviewed treatment practices that would be familiar and basic to dialysis nurses: reclining the patient into Trendelenberg position; administering intravenous solutions to maintain blood volume; administration of oxygen and pausing or ceasing ultrafiltration (Bradshaw, Ockerby & Bennett, 2011; Ellis, 2011; Evans, 2012; Hossli, 2005).

The results of a survey on the clinical management of IDH were presented, due to a recognised deficit stated by the author, as "no recent publication has studied how IDH is managed in dialysis clinics" (Hossli, 2005). The objectives for the study were concerned with incidence, prevalence and clinical management. One objective stated was to "analyse the need for further research into the incidence and management of IDH ..." although a detailed analysis was not presented, and unfortunately only unit managers were surveyed. Definitions of IDH were discussed with brief analyses of interventions. Overall, the author stated the responses "suggest the need for education on the pathophysiology of IDH" (Hossli, 2005, p. 290) and suggested "there is room for improvement of care" (2005, p. 291).

Two articles considered encouraging critical thinking skills in fluid management and concern for the potential of backfiltration during UF pause for IDH, respectively (Dale, 2012; Evans, 2012), and these articles stress the importance of individual patient assessment; however, blood pressure implications and IDH are mentioned only incidentally.

A previous study has indicated that MAP assessment during haemodialysis may provide an important marker for changes in intravascular status, which may be helpful in anticipating possible IDH episodes (Bradshaw, Ockerby & Bennett, 2011). As MAP ultimately represents perfusion pressure — as necessary for end organ tissue perfusion and as essential for adequate brain and vital organ oxygenation — such reduction in value could contribute to localised ischaemia as previously described. The pilot study undertaken found that monitoring changes in MAP could suggest when the intravascular compartment may be contracting. Instigation of a 10-minute UF pause in response to low MAP — in patients not symptomatic of IDH — could allow vascular refill and possibly prevent some episodes of IDH. Through the eight-week intervention period in this study, the incidence of IDH was decreased by 61%, though further studies with a larger sample are needed to replicate the findings (Bradshaw, Ockerby & Bennett, 2011).

In consideration of patient safety and the problem of IDH, one paper made argument for the UF rate to be utilised as a quality indicator within the haemodialysis setting (Lindberg & Ludvigsen, 2012). The authors suggest that this control is within the nurse's scope of practice and that it "may serve as a reward for perceived performance among haemodialysis nurses" (p. 1322). They comment, "Hypotensive episodes

related to ultrafiltration rate may be misinterpreted by renal nurses as an indication that the patient is hypovolemic and therefore cause clinical responses of premature termination of the dialysis session or abandonment of further ultrafiltration" (Lindberg & Ludvigsen, 2012, p. 1320). However, no direct link is made between the idealism of their suggestions to any recommendations for IDH prevention.

Discussion

It is interesting that no one definition for IDH has been universally accepted, and that some of the suggested parameters, especially "a fall in systolic pressure of >20 mmHg", could include the blood pressure ranges of the majority of patients in any one dialysis session. Of related relevance to this is the suggestion that a hypotensive episode need not be symptomatic to be of significant harm, which would infer recommendation of a succinct definition all the more important. With suggestion that asymptomatic episodes of relative hypotension may be just as damaging as the process of haemodialysis itself (McIntyre *et al.,* 2011), it is surprising that recommendations for intervention at specific blood pressure measures are not given.

Whilst it may be helpful to consider dialysis patients as hypotension-resistant or hypotension-prone, the variability of blood pressure ranges amongst haemodialysis patients must surely take into account individualised patient patterns, instead of a one-size-fits-all approach in defining and preventing the condition.

Although several treatment-specific recommendations have been made, it must be conceded that alterations to many of the parameters discussed here, for example, dialysate electrolytes, and administration of specific pharmacological agents are probably outside the scope of practice of most dialysis nurses. However, newer technologies may incorporate some of these in the future, and advanced familiarity of these processes may be of benefit. Additionally, it is suggested that mindfulness of physiological processes may allow confidence in performing further technological interventions and preventative recommendations as has been outlined.

Whilst most of the literature surrounding hypotension during haemodialysis necessarily offers suggestions regarding implementations to prevent and treat the condition, no one research paper proposes a definitive strategy towards the alleviation of IDH taking blood pressure into account.

Conclusion

Whilst informative and illuminating, the coverage of the problem of IDH from various medical perspectives rarely acknowledges that it is haemodialysis nurses assessing, monitoring, planning treatment strategies and performing interventions for these patients. There has been little dialogue regarding recommendations or standardisations of blood pressure management in the prevention of IDH. There has been much variation in suggestions to define the condition with little inquiry of nurses as to what knowledge bases or clinical influences shape their practice habits. And despite the many interventions and strategies available, IDH still occurs with alarming frequency.

An overarching approach in encouraging individualised patient assessment in consideration of "normal" blood pressure parameters (normal for each patient) is required, along with definitions of IDH that are informed by a patient's specific care needs, that will suit their individual treatment plan.

Additionally, guidelines centred around suggestion of early intervention — for instances of ascribed asymptomatic hypotension — may go a long way to decrease the incidence, and subsequently improve the outcomes, of the haemodialysis population.

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