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Intralipid use in Trauma

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Abstract

Hemorrhage is responsible for most deaths that occur during the first few hours after trauma. Current evidence indicates that initial liberal fluid resuscitation strategies may be associated with higher mortality in injured patients. In the pre hospital arena, intravenous fluids have been associated with worse patient outcomes due to increased coagulopathy and time to definitive care. Once in the trauma bay, damage control resuscitation principles apply to the severely injured patient. Large volume crystalloid infusion increases mortality. The best patient outcomes have been found with transfusion of blood products in a ratio that closely mimics whole blood.

Intralipid increased renal blood flow, carotid vascular resistance and mesenteric vascular resistance. In the presence of Intralipid, L-NMA-induced pressor response and systemic, carotid and renal vasoconstriction were more pronounced than in control dogs. Except for the coronary and carotid circulations, Intralipid modulates the NO pathway in cardiac and regional blood flow.Intralipid infusion in trauma patients is first suggested in the medical literature.

Keywords

Intralipid; Trauma

Introduction

The use of Intralipid therapy has been gaining traction as a treatment option for an ever expanding range of toxicities. Veterinary literature has reviewed intravenous lipid emulsion therapy (ILE) [1, 2] and published case reports or studies are available noting efficacy in toxicities including macro cyclic lactones [3, 4], baclofen [5], beta-blockers, calcium channel blockers [6], NSAID [7, 8], bromethalin [9], lidocaine [10], permethrin toxicity [11,12], tricyclic antidepressants [13].

Intravenous lipid emulsion (ILE) in human literature has been reported as a therapy for local anesthetic [14, 15], calcium channel blocker [16, 17], psychotropic medication [18] , glyphosatesurfactant herbicide toxicities [19] and even cocaine over dosage [20].

Original work performed by Weinberg noted a response in rats with bupivacaine induced asystole with lipid emulsion [21]. How exactly ILE works is not certain but two theories are considered. The “lipid sink” theory is most commonly considered the primary mode of action. In this theory, the formation of a lipid compartment within the intravascular space can serve as a “sink” into which the lipophilic drug will be drawn into. The drug is then excreted/ metabolized. Determination of a drug’s lipophilicity may be noted by its log P value. A value >1 indicates lipophilic compound which may move into the temporary lipid phase and be less distributed throughout the body. The formulation of ILE utilized may play a role and supports the “lipid-sink” theory based on one study [22].

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This theory has been supported in two case reports that followed plasma ropivacaine [15] and serum verapamil concentrations [17]

An alternate theory is that the lipid provides an energy source for the cardiac myocytes by increasing the availability of FFA. The increase of FFA may also aid in increasing the activation of voltage-gated calcium channels in the myocardium, increasing cytosolic calcium channels This mechanism may be most important in cases of calcium-channel blockade [23, 24].

Intralipid use in Rapid Blood Loss

The following were studied in a perimortem mouse model of rapid blood loss: (a) efficacy of a prototypical micellar colloid, Intralipid 20%, (IL20), compared to albumin (b) comparison of intra-arterial and intravenous resuscitation, (c) efficacy of IL20 at a volume 2 × the volume of blood removed, and (d) efficacy of oxygenated IL20 after clinical death (CD). CD, the absence of breathing and zero blood pressure (BP), was produced by removing 55% of the blood volume within 3 minutes. After CD, the chest was opened to observe ventricular contraction. IL20, Ringer's lactate (RL), or albumin was infused perimortem.

Without resuscitation CD occurred in 2.85 ± 0.40 minutes. Ventricular contraction persisted 20.50 ± 1.11 minutes after CD. RL infused immediately after CD restored breathing if given intra-arterially but not intravenously. IL20 was superior to the prototypical colloid, albumin in maintaining the BP. Increasing the volume of IL20 further increased BP. Delayed RL infusion after CD failed to restore breathing. Delayed resuscitation after CD with oxygenated IL20 restored breathing and BP.

Micellar colloid is superior to the prototypical colloid albumin and can possibly be of use when signs of life are no longer present. In extremis, intra-arterial infusion is superior to intravenous infusion [25].

Fat emulsions, Intralipid 30% and Intralipid 10% were compared in terms of the resulting plasma levels of different lipid components and clinical tolerance in critically-ill patients with multi-injuries. Sixteen critically-ill patients with severe systemic inflammatory response were randomly assigned to two groups, each one comprised of eight patients. Each group was administered the same quantity of fat/Kg/day either Intralipid 30% or Intralipid 10%. The infusion lasted 12 h daily for 6 days.

During the infusion of the fat emulsion, a lower median plasma concentration of triglycerides, phospholipids and free cholesterol was observed in patients who received Intralipid 30% compared with those who received Intralipid 10%. The above observations were sustained 4 h after the termination of the infusion.

Free fatty acids had a higher mean plasma concentration in the group of patients who received Intralipid 30%. There were no differences between the two groups as far as the median plasma concentration of cholesterol and lipoproteins (LDL, HDL, VLDL) are concerned. On the contrary, there was an increase in LpX in the Intralipid 10% group. From the above findings, we draw the conclusion that Intralipid 30% revealed better profiles of different lipid components than Intralipid 10% in critically-ill patients. The new emulsion of higher concentration in triglyceride was proved clinically safe and its use is suggested for critically-ill patients who require total parenteral nutrition [26].

Intralipid Modulates the NO Pathway in Cardiac and Regional Blood Flow

The commercial propofol preparation in an Intralipid solution causes marked vasodilatation. Both propofol and its solvent seem to stimulate the nitric oxide (NO) pathway. The role of Intralipid in cardiac and regional hemodynamic changes induced by propofol and their respective interactions with the NO pathway was assessed.

Dogs were instrumented to record arterial pressure, heart rate, cardiac output, dP/dt (the first derivative of left ventricular pressure) and vertebral, carotid, coronary, mesenteric, hepatic, portal and renal blood flows. Experimental groups were as follows. Group 1 (control; n = 11): N-methyl-L-arginine (L-NMA) 20 mg kg-1 i.v.; Group 2 (n = 8): propofol (10 mg ml-1) 4 mg kg-1 i.v. bolus followed by 0.6 mg kg-1 min-1; Group 3 (n = 6): Intralipid 0.25 ml kg-1 bolus followed by 0.06 ml kg-1 min-1. After 60 min, L-NMA was injected in Groups 2 and 3.

Propofol induced increases in heart rate, coronary and carotid blood flows, and decreases in systemic vascular resistance and dP/dt. Intralipid increased renal blood flow, carotid vascular resistance and mesenteric vascular resistance. In the presence of Intralipid, L-NMA-induced pressor response and systemic, carotid and renal vasoconstriction were more pronounced than in control dogs.

Except for the coronary and carotid circulations, Intralipid modulates the NO pathway in cardiac and regional blood flow [27].

Propofol is a commonly used anesthetic. Despite its favorable safety profile, propofol causes hypotension which can result in end-organ hypo perfusion. Intralipid is a lipid emulsion that has been shown to reverse the vasodilatory effects of propofol in isolated vessels; however, whether these effects are recapitulated in vivo is not known. The objectives of this study were to determine if Intralipid reverses the hypotensive and anesthetic effects of propofol in rats. Under isoflurane anesthesia, male Sprague Dawley rats were instrumented with indwelling catheters for mean arterial pressure (MAP) assessments as well as subdural electrodes for cortical activity assessments by electroencephalography (EEG). Propofol (10 mg/kg IV) caused hypotension (55±2% drop in MAP, $P<0.001$) and Intralipid (4mL/kg IV) caused greater reversal (80±9%) of blood pressures compared to saline (19±1%; $P<0.001$).

Blockade of the autonomic nervous system with chlorisondamine (2.5 mg/kg IV) caused marked hypotension (56±3% lowering of MAP, $P<0.001$) which could be reversed with a constant infusion of phenylephrine (300 μg/kg/hr); under these conditions, propofol nevertheless caused hypotension (12±4% lowering of MAP) which was completely reversed by Intralipid. Propofol induced cortical burst suppression was not affected by Intralipid (2±3%), saline (-4%) or 20% BSA (-2±1%; $P=0.27$). These results demonstrate that Intralipid reverses propofol-mediated hypotension with minimal effects on its anesthetic profile. Intralipid could be particularly useful as a rescue against propofol in patients prone to hemodynamic instability such as the elderly [28].

Sepsis is one of the most serious complications that can occur during total parenteral nutrition (TPN) procedures. In this experimental study, we investigated the effects of TPN, with or without lipid emulsion, on vascular endothelial damage.

In total, 50 rabbits were used, divided into 5 groups of 10 each. TPN with lipids (group 1), TPN without lipids (group 2), and 0.09% saline (group 3) were given for 10 days via a central venous catheter. Group 4 received no treatment other than placement of a central venous catheter for 10 days. Group 5 was a control group. At the end of day 10, rabbits were sacrificed and tissue samples of liver, kidney, and inferior vena cava were prepared and examined by immunohistochemical methods for vascular cellular adhesion molecule (VCAM)-1 expression.

In tissue sections of liver, kidney, and inferior vena cava, VCAM-1 activity was increased prominently in TPN with and without lipids compared with the control group. VCAM-1 activity in the TPN with lipids group was decreased versus the TPN without lipids group ($P>0.05$).

The TPN procedure results in vascular endothelial cell damage not only in the vein where the solution is introduced but also in other parts of the vascular system. Even if it is not statistically significant, lipids in the TPN formula may decrease this endothelial cell damage, as shown by immunohistochemistry [29].

Ischemia-reperfusion injury is a determinant in liver injury occurring during surgical procedures, ischemic states, and multiple organ failure. The pre-existing nutritional status of the liver, i.e., fasting, might contribute to the extent of tissue injury. This study investigated whether Intralipid, a solution containing soybean oil, egg phospholipids, and glycerol, could protect ex vivo perfused livers of fasting rats from anoxia-reoxygenation injury.

The portal vein was cannulated, and the liver was removed and perfused in a closed ex vivo system. Isolated livers were perfused with glucose 5.5 and 15 mM, and two different concentrations of Intralipid, i.e., 0.5:100 and 1:100 (v/v) Intralipid 10%:medium ($n = 5$ in each group). The experiment consisted of perfusion for 15 min, warm anoxia for 60 min, and reoxygenation during 60 min. Hepatic enzymes, potassium, glucose, lactate, bilirubin, dienes, trienes, and cytochrome-c were analyzed in perfusate samples. The proportion of glycogen in hepatocytes was determined in biopsies.

Intralipid attenuated transaminases, lactate dehydrogenase, potassium, diene, and triene release in the perfusate (dose-dependent) during the reoxygenation phase when compared with glucose-treated groups. The concentration of cytochrome-c in the medium was the highest in the 5.5-mM glucose group. The glycogen content was low in all livers at the start of the experiment.

Intralipid presents, under the present experimental conditions, a better protective effect than glucose in anoxiareoxygenation injury of the rat liver [30].

The Effect of Intralipid on Protein Consumption in Severe Burned Patients

Rong et al. (31) investigated the effect of Intralipid on protein consumption in severe burned patients. Sixty-seven non operative patients with severe burns were divided into Intralipid treatment group and non-Intralipid treatment group (control group), and the former was treated with 20% Intralipid (500 ml once a day) from post burn day 4 for 10 consecutive days. Venous blood samples

were collected from these patients for testing total protein, albumin, total cholesterol and triglyceride on postburn days 1, 7 and 14, respectively.

The levels of total protein, albumin, total cholesterol and triglyceride were within normal range on post burn day 1 in both groups, and only the albumin level was lowered in the groups on day 7 but at comparable magnitudes (32+/- 4.83 vs. 31+/-5.04 g/L, P<0.05). In contrast, the levels of total protein, albumin, total cholesterol and triglyceride were below the normal range on post burn day 14 in both groups, but Intralipid treatment group showed more albumin loss than the control group (28+/-6.46 vs 23+/-7.03 g/L, P<0.01).

Intralipid (20%) provides good energy source to ameliorate albumin loss in severe burned patients (31).

A concentrated fat emulsion (Intralipid 30%) with a phospholipid/triglyceride ratio of 0.04 was tested for clinical tolerance and metabolic effects in the short-term parenteral nutrition of septic and trauma critically ill patients and compared with Intralipid 20% (phospholipid/ triglyceride ratio of 0.06).

This was a prospective, randomized, multicenter study in the intensive care units in 10 university hospitals, including 90 adult patients in 2 groups: 55 septic and 35 trauma patients. Patients in each group were randomly divided into 2 subgroups according to the fat emulsions administered (1.4 g/kg per day) as part of the calories for at least 6 days of continuous total parenteral nutrition (TPN). One subgroup was treated with 30% long-chain triglycerides (phospholipid/ triglyceride ratio: 0.04) and the other with 20% long-chain triglycerides (phospholipid/ triglyceride ratio: 0.06).The parenteral nutrition formula was isocaloric and isonitrogenous with 0.25 g of nitrogen/ kg per day and 40% of the non protein calories as fat. Clinical tolerance was assessed during the study. At baseline and after 3 and 6 days of TPN, the following biochemical parameters were measured: prealbumin, retinol-binding protein, serum albumin, hematologic, hepatic and renal function variables, triglycerides, phospholipids, total and free cholesterol, nonesterified cholesterol, nonesterified fatty acids, and lipoproteins.

At baseline, no differences in age, gender, severity of the condition [Acute Physiology and Chronic Health Evaluation (APACHE II) score], or clinical chemistry were found between the subgroups. The levels of plasma proteins studied and the renal, hematologic, or hepatic function variables did not vary during the study period. Total cholesterol increased significantly, owing to esterified cholesterol, with 20% long-chain triglyceride in septic patients (baseline: 2.1 +/- 0.8 mmol/L, day 6: 2.8 +/- 0.6 mmol/L, p = .026). In septic patients receiving 20% long-chain triglycerides, plasma triglycerides had a similar behavior (baseline: 1.4 +/- 0.6 mmol/L, day 3: 2.2 +/- 0.8 mmol/L, p < .05). The very-low-density lipoprotein content of cholesterol, triglycerides, and phospholipids showed a tendency to decrease in septic patients treated with 30% long-chain triglycerides (NS). None of the emulsions induced the synthesis of lipoprotein X.

The results indicate that while both fat emulsions used in the TPN of critically ill patients are clinically safe, the 30% long-chain triglyceride fat emulsion with a phospholipid/triglyceride ratio of 0.04 causes fewer lipid metabolic disturbances (32).

A 4-year-old boy with an abdominal trauma had lipiduria following treatment with total intravenous nutrition for 4 days.

Renal function was normal throughout the course and the lipiduria ceased after withdrawal of the intravenous nutrition. The lipids were possibly excreted through the kidneys or entered the urine through a traumatic communication between the lymphatic vessels and the urinary system. Control of renal function and lipiduria after 1 year revealed normal conditions (33).

Current Fluid Resuscitation and the Vasopressor Support in Severe Trauma Patients

Harrois et al. (34) discuss the fluid resuscitation and the vasopressor support in severe trauma patients.

A critical point is to prevent a potential increase in bleeding by an overly aggressive resuscitative strategy. Indeed, large-volume fluid replacement may promote coagulopathy by diluting coagulation factors. Moreover, an excessive level of mean arterial pressure may induce bleeding by preventing clot formation.

Fluid resuscitation is the first-line therapy to restore intravascular volume and to prevent cardiac arrest.

Thus, fluid resuscitation before bleeding control must be limited to the bare minimum to maintain arterial pressure to minimize dilution of coagulation factors and complications of over fluid resuscitation. However, a strategy of low fluid resuscitation needs to be handled in a flexible way and to be balanced considering the severity of the hemorrhage and the transport time. A target systolic arterial pressure of 80-90 mmHg is recommended until the control of hemorrhage in trauma patients without brain injury. In addition to fluid resuscitation, early vasopressor

support may be required to restore arterial pressure and prevent excessive fluid resuscitation. It is crucial to find the best alchemy between fluid resuscitation and vasopressors, to consider hemodynamic monitoring and to establish trauma resuscitative protocols [34].

The ideal strategy for prehospital intravenous fluid resuscitation in trauma remains unclear. Fluid resuscitation may reverse shock but aggravate bleeding by raising blood pressure and haemodilution. We examined the effect of prehospital i.v. fluid on the physiologic status and need for blood transfusion in hypotensive trauma patients after their arrival in the emergency department (ED).

Retrospective analysis of trauma patients (n=941) with field hypotension presenting to a level 1 trauma centre.

Regression models were used to investigate associations between prehospital fluid volumes and shock index and blood transfusion respectively in the emergency department and mortality at 24h.

A 1L increase of prehospital i.v. fluid was associated with a 7% decrease of shock index in the emergency department ($p<0.001$). Volumes of 0.5-1L and 1-2L were associated with reduced likelihood of shock as compared to volumes of 0-0.5L: OR 0.61 ($p=0.03$) and OR 0.54 ($p=0.02$), respectively. Volumes of 1-2L were also associated with an increased likelihood of receiving blood transfusion in ED: OR 3.27 ($p<0.001$). Patients who had received volumes of >2L have a much greater likelihood of receiving blood transfusion in ED: OR 9.92 ($p<0.001$). Mortality at 24h was not associated with prehospital i.v. fluids.

In hypotensive trauma patients, prehospital i.v. fluids were associated with a reduction of likelihood of shock upon arrival in ED. However, volumes of >1L were associated with a markedly increased likelihood of receiving blood transfusion in ED. Therefore, decision making regarding prehospital i.v. fluid resuscitation is critical and may need to be tailored to the individual situation. Further research is needed to clarify whether a causal relationship exists between prehospital i.v. fluid volume and blood transfusion. Also, prospective trials on prehospital i.v. fluid resuscitation strategies in specific patient subgroups (e.g. traumatic brain injury and concomitant haemorrhage) are warranted [35].

Feinman et al. (36) explore the recent literature regarding the optimal type and amount of intravenous fluids for the trauma patient from the time of injury through their ICU stay. It discusses damage control principles as well as targeted resuscitation utilizing new technology.

In the prehospital arena, intravenous fluids have been associated with worse patient outcomes due to increased coagulopathy and time to definitive care. Once in the trauma bay, damage control resuscitation principles apply to the severely injured patient. Large volume crystalloid infusion increases mortality. The best patient outcomes have been found with transfusion of blood products in a ratio that closely mimics whole blood. Thrombelastography is a useful adjunct in resuscitation and can help guide the judicious use of blood products. New technology can help providers ascertain when a patient is appropriately resuscitated by determining adequate global and regional perfusion.

During the resuscitation of the acutely injured patient, crystalloids should be limited in favor of blood components.

Damage control principles apply until definitive hemostasis is obtained, at which point the focus should change to targeted resuscitation using traditional global endpoints of resuscitation in conjunction with determinants of regional perfusion [36].

Hemorrhage is responsible for most deaths that occur during the first few hours after trauma. Animal models of trauma have shown that restricting fluid administration can reduce the risk of death; however, studies in patients are difficult to conduct due to logistical and ethical problems. To maximize the value of the existing evidence, we performed a meta-analysis to compare liberal versus restricted fluid resuscitation strategies in trauma patients.

Medline and Embase were systemically searched from inception to February 2013.

We selected randomized controlled trials and observational studies that compared different fluid administration strategies in trauma patients. There were no restrictions for language, population, or publication year.

Four randomized controlled trials and seven observational studies were identified from 1,106 references. One of the randomized controlled trials suffered from a high protocol violation rate and was excluded from the final analysis.

The quantitative synthesis indicated that liberal fluid resuscitation strategies might be associated with higher mortality than restricted fluid strategies, both in randomized controlled trials (risk ratio, 1.25; 95% CI, 1.01-1.55; three trials; I(2), 0) and observational studies (odds ratio, 1.14; 95% CI, 1.01-1.28; seven studies; I(2), 21.4%). When only adjusted odds ratios were pooled for

observational studies, odds for mortality with liberal fluid resuscitation strategies increased (odds ratio, 1.19; 95% CI, 1.02-1.38; six studies; I (2), 26.3%).

Current evidence indicates that initial liberal fluid resuscitation strategies may be associated with higher mortality in injured patients. However, available studies are subject to a high risk of selection bias and clinical heterogeneity. This result should be interpreted with great caution [37].

Massive transfusion protocol seems to improve outcome in massively bleeding trauma patients, but not pelvic fracture patients. The aim of this study was to evaluate the effect of massive transfusion protocol on the mortality and fluid resuscitation of shocked pelvic fracture patients.

This is a trauma register study from a single hospital. From the trauma registry patients with pelvic fracture, injury severity score >15, admission base excess below -5, age >15 years, blunt trauma, and primary admission from the scene were identified. Patients were divided into two groups: Group 1-pre-massive transfusion protocol (2006-2009) and Group 2-post-massive transfusion protocol (2010-2013). Basic characteristics and intensive care unit length of stay, mortality, and fluid resuscitation data were retrieved from the registry. Standardized mortality ratio was assessed using revised injury severity classification, version II methodology.

Altogether, 102 patients were identified. Group 1 (n = 56) and Group 2 (n = 46) were comparable in their basic characteristics. The observed mortality was 35.7% and 26.1% in Groups 1 and 2, respectively. The standardized mortality ratio failed to reveal any difference between observed and expected mortality in either group. In the emergency room, the use of crystalloids decreased from 5.3 ± 3.4 to 3.3 ± 1.8 L (p = 0.002) with increased use of fresh frozen plasma (2.9 ± 4.4 vs 5.1 ± 5.3, p = 0.007).

No improvement in the adjusted survival of shocked pelvic fracture patients is apparent after implementation of massive transfusion protocol. Implementation of massive transfusion protocol is associated with a higher use of fresh frozen plasma and improved ratio of fresh frozen plasma : red blood cell toward the targeted 1:1 and decreased use of crystalloids [38].

Conclusion

Intralipid increased renal blood flow, carotid vascular resistance and mesenteric vascular resistance. In the presence of Intralipid, L-NMA-induced pressor response and systemic, carotid and renal vasoconstriction were more pronounced than in control dogs.

Except for the coronary and carotid circulations, Intralipid modulates the NO pathway in cardiac and regional blood flow.

Intralipid infusion in trauma patients is first suggested in the medical literature.

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