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Outcomes and complications for portal vein or superior mesenteric vein injury: No improvement in the era of damage control resuscitation ☆,☆☆,★

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ABSTRACT

Introduction: Portal vein (PV) and superior mesenteric vein (SMV) injuries are lethal. We hypothesised outcomes have improved with modern trauma care.
Methods: We reviewed patients presenting to our Level 1 trauma centre over ten-years with PV/SMV injuries, analysing physiology, operative management, associated injuries, and outcomes.
Results: Twenty-four patients had 7 PV and 15 SMV injuries, 2 had both; all had operative exploration. Sixty-seven percent had penetrating trauma. While many had normal vitals, profound acidosis was common. All patients had ≥2 additional abdominal injuries, liver most common (50%). Additional abdominal vascular injuries were more common in non-survivors than survivors: IVC 46% vs 22%, common hepatic artery 20% vs 0%, SMA 26% vs 11%. The mean injury severity score (ISS) was 32.4, and the mean new injury severity score (NISS) was 44.5. Mortality was 63%. Eleven patients died from exsanguination, two from SMV thrombosis, and two from sequelae of other injuries. All survivors had venorrhaphy, as did 8 non-survivors. Non-survivors were also shunted; had ligation; or bypass, shunting, and ligation. Three exsanguinated prior to repair. Two survivors had SMV related complications. One with proximal SMV injury developed severe venous congestion and multiple enterocutaneous fistulae. Another developed an arterioportal fistula, managed with embolisation and percutaneous portal vein stenting.
Conclusion: Despite advances (REBOA, damage control surgery and resuscitation, liberal use of ED thoracotomy), PV and SMV injuries remain lethal. Injuries to other structures are ubiquitous. Early exsanguination is the major cause of death. All survivors had successful venorrhaphy; those who required more complex repairs died. Compromised mesenteric venous flow causes morbidity and mortality.

Background

The portal vein (PV) and superior mesenteric vein (SMV) are thin-walled, high-flow vessels, nestled deep in the root of the mesentery in close proximity to the inferior vena cava (IVC), pancreas, liver, and gastrointestinal tract. They are rarely injured, with PV injuries reported to occur in only 0.008% of patients presenting following injury [1]; this prevents all but the most experienced trauma surgeons from having significant experience with their management. Compromised blood flow following ligation or repair of either structure can cause venous congestion and ischaemia of the midgut. For all these reasons, PV and SMV injuries have been associated with mortality in between 40 and 72% of patients [1–8].

Given their rarity, there have been no prospective studies of optimal management of PV and SMV injuries. A variety of case series have been published describing them, either as a subset of abdominal vascular injuries, or as series specifically of PV or SMV injuries. The most recent of these series was published in 2009 [6]. Although novel operative techniques for definitive haemostasis have not been described, the intervening decade has seen significant progress in the development and adoption of resuscitative practices, including damage control resuscitation, blood product administration, and temporary in-flow control with resuscitative

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endovascular balloon occlusion of the aorta (REBOA). This study was designed to investigate whether survival has improved in the era of damage control resuscitation, and to determine the relationship between surgical technique and outcomes.

Methods

All patients presenting to a Level I adult trauma centre between January 1, 2008 and December 31, 2017 with mesenteric, splenic, or other major intra-abdominal venous injury were identified from our trauma registry. Their operative reports were reviewed; patients with injuries to the portal vein or superior mesenteric vein were included in the study population. Data was collected from the trauma registry regarding admission vital signs and laboratory values, fluid and blood product resuscitation, and outcome. Operative reports were reviewed for anatomic injury details, associated injuries, and operative technique. Additional clinical documentation was reviewed to identify complications and their management.

Statistical analysis was performed using Stata/IC 15 (StataCorp; College Station, TX), comparing physiologic markers and resuscitation volumes for survivors and non-survivors. Due to the small size of the study population, no assumptions were made regarding normal sample distribution, and no attempt was made to use logistic regression. Categorical variables were compared using Fisher's exact test, and continuous variables were compared using the Mann-Whitney U test.

This study was approved by the Institutional Review Board of the University of Maryland School of Medicine.

Results

Over the study period, 63,789 patients presented to the trauma centre. Of these, 83 were identified from the trauma registry with abdominal visceral venous injuries, of whom seven had injuries to the portal vein, 15 had injuries to the superior mesenteric vein, and two had injuries to both vessels. All injuries were lacerations; operative reports did not describe length or orientation in detail, but there were no complete transections or traumatic thromboses. Patients' median age was 33.5, and 83% were male. Overall mortality was 15/24 (62.5%). See Table 1 for demographics and injury characteristics.

Table 1
Demographic and clinical characteristics of patients with portal vein or superior mesenteric vein injuries ($N=24$). ISS: injury severity score, NISS: new injury severity score, REBOA: resuscitative endovascular balloon occlusion of the aorta.

Characteristic	Value
Age (median)	33.5
Male sex (%)	83
Penetrating mechanism (%)	66.7
Injury severity score (median)	38
Presenting heart rate >100 (n)	15
Presenting systolic blood pressure <90 (n)	9
ISS (median)	36.5
NISS (median)	42
Initial haemoglobin, g/dL (mean)	11.4
Initial lactate, mg/dL (mean)	7.7
Initial base excess (mean) ($n=22$)	−11.2
Cardiopulmonary arrest on presentation (n)	4
Cardiopulmonary arrest in resuscitation unit (n)	3
Resuscitative thoracotomy (n)	5
REBOA (n)	1
Damage control laparotomy (%)	88.2
Hospital days, survivors only (median)	33.2
Ventilator days, survivors only (median)	6.0
Mortality (n)	15

Associated injuries

All patients had associated intra-abdominal injuries. With the exception of the renal vein and the pancreas, all visceral or vascular injuries were more commonly diagnosed in non-survivors than in survivors (Table 2). Seven of the fifteen non-survivors exsanguinated in operating theatre, and these patients may have had undiagnosed associated injuries. The frequency of injury was not statistically significantly different between survivors and non-survivors for any intra-abdominal structure.

The median injury severity score (ISS) amongst survivors was 25 (SD 14.7, range 9–50), not significantly different from amongst non-survivors (median 38, SD 11.4, range 16–50). However, the new injury severity scores (NISS) amongst survivors (median 34, SD 10.5, range 22–57) were less than amongst non-survivors (median 48, SD 12.3, range 27–66) (Table 3).

Physiology and resuscitation

There was a trend toward higher initial heart rate for non-survivors (mean 131, SD 22, range 91–174) compared to survivors (mean 105, SD 34, range 56–150), although this did not reach statistical significance ($p=0.095$). There was a trend toward lower initial systolic blood pressure for non-survivors (mean 102 mmHg, SD 42, range 25–179) compared to survivors (mean 122 mmHg, SD 19, range 86–145), which also did not reach statistical significance ($p=0.183$). Temperature also did not significantly vary between non-survivors (mean 35.9 °C, SD 1.1, range 34–36.8) and survivors (mean 36.3 °C, SD 1.3, range 34.1–38.5) ($p=0.751$).

Admission haemoglobin was lower amongst non-survivors (mean 10.5, SD 2.7, range 4.4–14.2) than survivors (mean 12.9,

Table 2
Associated intra-abdominal injuries.

Injured vessel or organ	Survivors (%)	Non-survivors (%)
Inferior vena cava	22	47
Right renal vein	22	7
Hepatic artery	0	27
Superior mesenteric artery	11	27
Common bile duct	0	13
Liver	44	53
Right kidney	0	13
Pancreas	44	33
Spleen	11	13
Duodenum	22	33
Jejunum/ileum	22	33
Colon	33	40

Table 3
Median injury severity scores, mean physiologic values, and mean 24-hour resuscitation totals amongst survivors and non-survivors. Patients who presented in cardiac arrest (all non-survivors, $N=4$) were excluded from the calculations of heart rate, systolic blood pressure, and temperature. ISS: injury severity score, NISS: new injury severity score, PRBC: packed red blood cells, FFP: fresh frozen plasma.

Parameter	Survivors	Non-survivors	p value
ISS	25	38	0.095
NISS	34	48	0.012
Heart rate (bpm)	105	131	0.095
Systolic blood pressure (mm Hg)	122	102	0.183
Temperature (C)	36.3	35.9	0.751
Haemoglobin (g/dL)	12.9	10.2	0.023
Lactate (mg/dL)	5.3	9.2	0.020
Base excess (mEq/L)	−8.3	−13.2	0.216
PRBC (units)	12.6	34.1	0.083
FFP (units)	11	27.7	0.244
Platelets (units)	1.4	3.3	0.249
Crystalloid (liters)	7.9	4.8	0.889
Total blood products (liters)	6.9	18.1	0.101

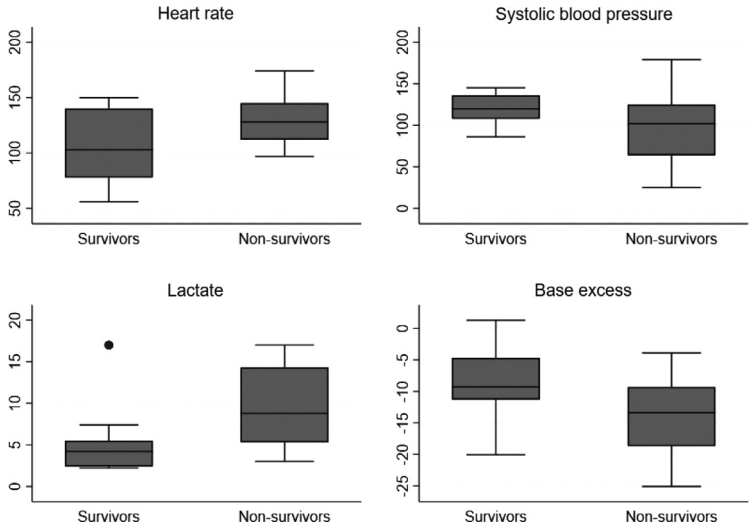

Fig. 1. Box plots of initial vital signs and laboratory values by mortality for 9 survivors and 15 non-survivors ($N = 24$). Patients presenting in cardiopulmonary arrest were excluded from graphs of vital signs ($N = 4$).

SD 1.5, range 9.8–15.3) ($p = 0.023$). Similarly, lactate was lower amongst survivors (mean 5.4, SD 4.7, range 2.2–17) than non-survivors (mean 9.1, SD 4.6, range 3–17) ($p = 0.020$). Non-survivors had a trend toward a lower base excess (mean −13.2, SD 6.6, range −25.1 to −3.9, compared to survivors' (mean −8.3, SD 6.8, range −20.1–1.3, $p = 0.22$) and higher INR (non-survivors mean 1.71, SD 1.98, range 1.0–4.4 compared to survivors mean 1.24, SD 0.27, range 1.0–1.9, $p = 0.315$) (Fig. 1).

Blood products were transfused in higher numbers to non-survivors than to survivors, although these differences were not statistically significant for any category: units of packed red blood cells (PRBC), units of fresh frozen plasma (FFP), units of platelets, or total volume of transfused blood (see Table 3). During the first 24 h following admission, a total of 624 units of PRBC, 514 units of FFP, and 63 units of platelets were transfused to the study population (ratio 1: 0.82: 0.10). There was no temporal trend in the ratio of transfused products over the study period. The mean volume of blood products transfused in the first 24 h was 13.9 L, in addition to 5.8 L of crystalloid and 167 mL of colloid.

Operative interventions

All four patients who presented in cardiopulmonary arrest received resuscitative thoracotomies in the Trauma Resuscitation Unit (TRU), as did a fifth patient who arrested in the TRU. Two additional patients arrested in the TRU; one of them received resuscitative balloon occlusion of the aorta (REBOA), while the other achieved ROSC and was transferred to the operating theatre. Of these seven patients who arrested prior to operation, none survived to discharge, and only two were transferred out of the operating theatre alive.

With regards to the management of their portal and superior mesenteric vein injuries, 17 patients received direct suture venorrhaphy, including all 9 survivors. Three patients exsanguinated in the operating theatre before any attempt was made at definitive management of their mesenteric venous injury. One patient had her portal vein ligated but expired during attempted hepatic explantation for a devastating gunshot wound to the hepatic vein. For another patient, an attempt was made at bypass from the SMV to the portal vein; when this failed, an attempt was made to shunt the vessels, and when this failed the portal vein was ligated just prior to the patient's exsanguination in the operating theatre. Two patients received shunts, one of them as the first attempt at control of the vessels, and the other after being unable to achieve haemostasis with direct suture venorrhaphy. For the two patients in whom the first attempt at haemostasis was not successful, both died from exsanguination on the operating theatre table.

Complications

Four patients developed complications related to their mesenteric venous repair. Two patients developed venous thrombosis, one developed multiple enterocutaneous fistulae, and the fourth patient developed an arterioportal fistula from the superior mesenteric artery (SMA) to the portal vein.

Patient 1

This patient sustained a gunshot wound to his upper abdomen, and underwent a damage control laparotomy with non-anatomic hepatic resection and enterectomy for multiple small bowel perforations. He was taken back to the operating theatre two days later, and was found to have a near-transection of the neck of the pancreas, with branches shorn off the SMA and SMV; the vessels were repaired and the pancreatic injury was managed via pancreaticoduodenectomy. His abdomen was re-explored the following day and was found to have narrowing and nonocclusive thrombosis of his SMV, with severe venous congestion of the bowel. The narrowed section of vein was resected, balloon embolectomy was performed, and a bypass graft was created with reversed saphenous vein graft. On the following day he underwent enterectomy and transverse colectomy for venous ischaemia, and had a second balloon thrombectomy with anastomotic revision of the SMV bypass graft. On his next exploration, he was discovered to have sustained complete mesenteric thrombosis, which did not improve despite additional distal thrombectomies. He underwent a completion enterectomy, which was in turn complicated by breakdown of multiple staple lines, and support was withdrawn on post-operative day 14 from his initial presentation and operation.

Patient 2

This patient presented with a gunshot wound to his right back. He was taken to the operating theatre for a damage control laparotomy, gastrorrhaphy, SMV venorrhaphy, and packing of a pancreatic neck injury. The following day, he underwent ERCP which demonstrated an injury to the pancreatic duct, following which he was taken back to the operating theatre for pancreatic neck

resection with stapling of the proximal margin and a Roux-en-Y pancreaticojejunostomy to the tail of the pancreas, with wide drainage of the pancreatic head and placement of a distal feeding jejunostomy tube. He became haemodynamically unstable three days later; on exploration he was found to have retroperitoneal saponification and thrombosis of the SMV. The interventional radiology service performed transhepatic stenting of the SMV, and his haemodynamic instability improved. On post-operative day 14 from his original operation, he was emergently taken back to the operating theatre for haemorrhage via his drains, and his SMV stent was discovered to have eroded through the vessel. This was exposed by resecting the transverse colon, and the SMV was ligated on both sides of the stent; the pancreatic anastomosis was noted to be completely dehisced during exposure of the SMV. The patient developed venous ischaemia of the small bowel and expired the following day.

Patient 3

This woman presented after a motor vehicle collision with a heart rate of 150 and severe hypotension. She was emergently taken to the operating theatre and underwent a partial colectomy and multiple enterectomies for distal mesenteric avulsions, as well as lateral venorrhaphy of the proximal SMV where multiple jejunal branches had been avulsed. Proximal branches of the SMA had also been avulsed and were ligated. She was left in gastrointestinal discontinuity with an open abdomen. She was taken back to the operating theatre the following day for orthopaedic repairs and abdominal re-exploration. She had severely congested small bowel with areas of enteric and colonic necrosis, which were resected and she was again left in discontinuity. She was brought back to the operating theatre on post-operative day three from her initial operation, and three hand-sewn bowel anastomoses were performed, restoring gastrointestinal continuity. The following day she was re-explored for a third time; her anastomoses were intact, a polyglactin mesh was used as a fascial interposition, and her subcutaneous tissue and skin were closed over drains. On post-operative day 11 from her initial exploration, she was again taken to the operating theatre for enteric output from her subcutaneous drains; her bowel was adherent and could not be mobilised, so enteric leakage was controlled with drains. She eventually had a split thickness skin graft placed over her abdominal wound, and was discharged from the hospital on total parenteral nutrition on post-operative day 78 from her initial presentation.

She was re-admitted for elective takedown of her enterocutaneous fistulae five months later. This operation was notable for severe adhesions and intra-abdominal abscesses, and it was complicated by numerous enterotomies. A completion enterectomy was performed, as had been discussed with the patient as a possibility prior to her elective operation. This hospital course was again complicated by intra-abdominal infection, but she was discharged on post-operative day 27 and referred to a small bowel transplantation program at a nearby institution. She died 40 months later, almost four years following her initial injury.

Patient 4

This patient presented with gunshot wounds to the right upper quadrant and left back, with a systolic blood pressure of 80 and a heart rate of 60. His blood pressure normalised after 2 units of PRBCs, and he was taken to CT scan where he was found to have large volume haemoperitoneum and injuries to the IVC, pancreas, liver, and duodenum. He was taken to the operating theatre, where he underwent lateral venorrhaphy of his IVC, portal vein, and SMV and a cholecystectomy. He had a non-expanding haematoma around the head of the pancreas and in the mesentery of the duodenum. This area was packed, his abdomen was left open and he was taken to interventional radiology for a visceral arteriogram. He was noted to have a cutoff of the gastroduodenal artery and a pseudoaneurysm from his inferior pancreaticoduodenal artery with fistulization to the portal vein; the artery was embolised with coils. He was taken back to the operating theatre the following day for gastrojejunostomy feeding tube placement and abdominal closure.

Repeat CTA of his abdomen demonstrated a persistent arterioportal fistula on hospital day 3. He was taken back to IR, where the feeding artery could not be identified. A transhepatic portal vein stent graft was placed, and the pseudoaneurysm was embolised via a transcutaneous approach. Repeat CT angiogram of his abdomen on hospital day 12 demonstrated a persistent arterioportal fistula. The pseudoaneurysm was embolised with coils and thrombin via a transcutaneous, transhepatic approach. His portal vein was noted to have thrombosed, but he had adequate venous collateralization and had no symptomatic small bowel venous congestion. He was discharged from the hospital to a rehab facility on hospital day 26.

He was subsequently readmitted with upper gastrointestinal bleeding 8, 10, and 11 months following his initial injury. During each of these admissions, he underwent endoscopic removal of coils that had eroded into his duodenum. He was last seen in follow-up 19 months from his initial injury, clinically stable apart from continued intravenous drug use and chronic lower extremity venous stasis secondary to venous thromboembolic disease.

Discussion

This series of 24 patients had a mortality of 62.5%, placing it within the 40–72% range of mortality rates reported in other series [1–8]. As with these other series, all patients had associated intra-abdominal injuries. This is a near anatomic certainty in penetrating trauma, where any trajectory that includes the PV or SMV must pass through other anatomic structures which surround these vessels. Given the magnitude of force necessary to cause shearing injuries to proximal mesenteric veins, concomitant abdominal injuries are likely mandatory blunt PV and SMV injuries as well. NISS correlated with survival better than ISS, as would be expected for a population of mostly penetrating trauma patients whose injuries were clustered in the upper abdomen. Not surprisingly, greater degrees of shock–as demonstrated by tachycardia, hypotension, acidosis, and anaemia–were all associated with greater mortality, although perhaps due to the small size of the patient population, only haemoglobin and lactate had statistically significant correlations with mortality.

Only two series have reported on outcomes following resuscitative thoracotomy in PV or SMV injuries, with only one out of 13 patients surviving [3,6]. All five patients undergoing resuscitative thoracotomy in the current series died, all of them in the operating theatre, bringing the overall reported mortality to 94%. REBOA has become much more widely used since Fraga et al. published the most recent series of PV or SMV injuries in 2009, and became a relatively common resuscitative manoeuvre in our institution during the study period [9]. Major abdominal venous injury is a form of noncompressible torso trauma, making this a potential indication for zone 1 (descending thoracic aorta) REBOA use. Romagnoli et al. reported only slightly longer time to aortic occlusion with REBOA compared to resuscitative thoracotomy and aortic cross-clamping [10], and REBOA reduces the potential for iatrogenic thoracic injuries. While in theory REBOA provides greater benefit for arterial injuries than venous injuries, global inflow control likely leads to major reduction in infradiagmatic venous bleeding as well [11]. Our series may include the first published use of zone 1 REBOA for global haemorrhage control with a PV or SMV injury. This patient did not survive, although given the dismal overall outcomes when patients with these injuries present in severe

shock, this should not be interpreted as a sign that REBOA does not have a role in the resuscitation of these injuries.

The other major development in trauma resuscitation since the Fraga et al.'s series has been the widespread acceptance of damage control resuscitation (DCR). In addition to damage control laparotomy, DCR incorporates early balanced blood product administration, aggressive treatment of hypothermia, and use of point-of-care testing to guide the use of procoagulants [12]. Although the publication of PROPPR trial did not solidify the evidence base for balanced resuscitation until 2015 [13], the concept of damage control resuscitation was already well established in our institution at the beginning of the study period in 2008, as evidenced by the nearly 1:1 ratio of PRBC to FFP administration in our population. Platelets were transfused in lower ratios, due to both patients who developed cardiac arrest before significant blood products were transfused, and also to the several patients who only required 6 or fewer units of PRBC and never had platelets given as part of their massive transfusion event.

The 24-hour volume of crystalloid administration surpassed blood product administration for survivors but not for non-survivors. Presumably most of this crystalloid was given after haemostasis was achieved for survivors, to keep pace with fluid loss to the extravascular compartment, so this does not show a betrayal of the DCR concept of minimizing crystalloids. Point-of-care thromboelastography (TEG) entered widespread use in our Trauma Resuscitation Unit and OTs around the mid-point of the study period; given the relatively small number of patients in this study, no meaningful conclusions can be drawn about the utility of TEG for PV or SMV injuries.

The most relevant question for any surgeon who encounters a proximal mesenteric venous injury is how haemostasis should best be achieved. Several prior authors, going back to Stone et al. in 1982, have described ligation as a plausible alternative to venorrhaphy [14]. In our series, venorrhaphy was attempted in 17 of the 24 patients, including all 9 survivors. While the details of exposure and repair technique were generally not described in operative notes, the standard technique in our institution is that described by Henry et al. [15] and reported in the Advanced Trauma Operative Management course [16,17], using Judd–Allis clamps to reapproximate the disrupted vein edges and then serially releasing these clamps as the laceration is sutured. This technique allows for rapid haemostasis, and does not require proximal or distal control, making it particularly useful for the confluence of the PV, SMV, and splenic vein where three high-flow vessels are all relatively inaccessible behind the head of the pancreas. Although untested, it is the authors' belief that this technique provides more rapid haemostasis than ligation, which requires circumferential control of both sides of a laceration. Ligation was not attempted as a primary haemostatic technique for any patient in this series, making comparison impossible from this data. Of particular note, all patients who required more than one attempt at haemostasis from the PV or SMV died, highlighting the unforgiving nature of these injuries.

This study describes four surgical complications of mesenteric venous repair, and is the first published series to do so. Two patients developed thrombosis at the site of their venorrhaphy; one of them died from mesenteric venous ischaemia, the other was successfully treated with a percutaneous stent graft but died when this eroded through the SMV wall in the setting of a pancreatic leak. There are two published reports of successful venous bypass of ligated SMVs complicated by mesenteric venous congestion, one using femoral vein and the other using 8 mm ringed polytetrafluoroethylene (PTFE) [18,19]. Our attempt to reconstruct Patient 1′s SMV with greater saphenous vein was not successful, due either to size mismatch, local inflammation, or simply poor luck in a severely injured patient. Anticoagulation or antiplatelet therapy may potentially have a role in preventing post-operative thrombosis in suitable patients. However, in a study of 27 patients undergoing oncologic pancreaticoduodenectomy with portal vein resection, thrombosis occurred in four of six patients who were treated with therapeutic anticoagulation using intravenous heparin [20].

Patient 3 developed multiple enterocutaneous fistulae following combined proximal and distal mesenteric venous injuries requiring bowel resections. Even if venorrhaphy does not lead to thrombosis, some stenosis is guaranteed, and with it a high chance of at least some venous congestion of the intestine. This is likely to be less well tolerated in a patient who also requires bowel resections and anastomoses. Patient 4 developed a SMA-SMV fistula, which was successfully treated with multiple endovascular and percutaneous procedures by the interventional radiology service. Arteriovenous fistula appears to be a rare complication of penetrating injury to the root of the mesentery. Due to acute inflammation, as with our patient, or significant adhesions in more delayed presentations, open surgical therapy brings significant risk of injury to nearby vital structures. Successful endovascular therapy with a combination of embolization and stent graft placement has been described in several case studies [21–23].

This study has several limitations. Foremost among them is its basic design as a relatively small case series, which makes meaningful comparisons difficult. Due to the rare nature of PV and SMV injuries, significant variations in associated injuries, and the physiologic distress of most patients with these injuries, prospective studies comparing management techniques are unlikely to ever be performed. The retrospective nature of the study makes it reliant on contemporaneously recorded operative notes, and important details of the anatomic injuries and the precise surgical techniques used to expose and repair the injuries were typically lacking in the clinical documentation. Added to this is the inherent difficulty of studying patients during periods of physiologic distress, which may lead to inaccuracy and loss of detail in documentation and subsequent inability to piece together the precise, minute-by-minute timeline of interventions and physiologic parameters which are used to make clinical decisions in real-time. Several potentially useful data which could shed light on factors associated with survival, including scene and transport times, were missing for so many patients that they could not be used for the study.

Despite its limitations, a few conclusions can be drawn from this study. First and foremost, despite advances in trauma resuscitation, PV and SMV injuries remain highly lethal, with the vast majority of deaths occurring from exsanguination. While no firm conclusions can be drawn about techniques for repair, it seems likely that a surgeon typically has only one chance to achieve haemostasis. Whether venorrhaphy, ligation, shunting, or bypass is used first, salvage techniques are highly unlikely to succeed if the first attempt fails. Finally, the risk of post-repair thrombosis is real, and is likely to be poorly tolerated.

Declaration of Competing Interest

RE: Howley IW, Stein DM, Scalea TM. Outcomes and complications for portal vein or superior mesenteric vein injury: no improvement in the era of damage control resuscitation.

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Supplementary materials

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