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Outcomes of penetrating carotid artery injuries: A South African multicentre study

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Abstract

Background: This multicenter study examines the contemporary management of penetrating carotid artery injury (PCAI) to identify trends in management, outcomes, and to determine prognostic factors for stroke and death.

Methods: Data from three large urban trauma centers in South Africa were retrospectively reviewed for patients who presented with PCAI from 2012 to 2020.

Results: Of 149 identified patients, 137 actively managed patients were included. Twenty-four patients (17.9%) presented in coma and 12 (9.0%) with localizing signs (LS). CT angiography was performed on admission for 120 (87.6%) patients. Thirty patients (21.9%) underwent nonoperative management, 87 (63.5%) open surgery, and 20 (14.6%) endovascular stenting. Eighteen patients (13.1%) died, and 15 (12.6%) surviving patients had strokes. Ligation was significantly related to death and reperfusion to survival. A mechanism of gunshot wound, occlusive injuries, a threatened airway, a systolic blood pressure <90 mmHg, hard signs of vascular injury, a low GCS, coma, a CT brain demonstrating infarct, a high injury severity score and shock index, a low pH or HCO3, and an elevated lactate were significant independent prognostic factors for death. Ligation was unsurvivable in all patients with severe neurological deficits, whereas reperfusion procedures resulted in survival in 63% (12/19) patients with coma and 78% (7/9) with LS although with high stroke rates (coma: 25.0%, LS: 85.7%).

Conclusions: Outcomes in PCAI, including patients with severe neurological deficit and stroke, are better when reperfused. Reperfusion holds the best promise of survival and ligation should be reserved for technically inaccessible bleeding injuries.

KEYWORDS
carotid, coma, infarct, injury, penetrating

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1 | INTRODUCTION

The management of penetrating carotid artery injuries (PCAI) remains controversial. These injuries are relatively uncommon, so prohibiting any single center from acquiring enough experience to develop definitive management guidelines.[1] Although death can result from both exsanguination and airway compromise, these patients additionally have a high incidence of stroke-related morbidity and mortality reported in the range between 11% and 34%.[1–4] Shock, coma, and involvement of the internal carotid artery (ICA) are known independent risk factors for poor outcome.[2–5] The recent adoption of computerized tomographic angiography (CTA) into algorithms for penetrating neck injury (PNI), has increased the incidence of 'minor' injuries detected.[6,7] Furthermore, advances in endovascular surgery have created a plethora of additional management options for PCAI.[8,9] The prevailing consensus for the management of most patients with PCAI is to urgently restore vascular continuity, as ligation is associated with a high risk of stroke. Restoration of perfusion in patients with established, severe neurological deficits can however exacerbate cerebral edema and convert an ischemic infarct into a hemorrhagic one.[2,3,10–22] Both processes can be fatal.

Controversy hence exists around the management of patients with established neurological deficits, although some authors advocate restoration of perfusion irrespective of neurological deficits.[2–4,22]

Although the management of PCAI has been vigorously debated in the past, there is a relative paucity of recent literature specifically addressing the management of PCAI in a civilian setting. This paucity exists despite the major advances made in both imaging and endovascular techniques in the management of PNI over the last 2 decades. Furthermore, no previous South African multicenter study on PCAI exists and the management tends not to be uniform across different centers.[3,8,21] This multicenter study examines the contemporary management of PCAI in three large trauma centers in South Africa, to identify trends in imaging, management, and outcomes and to determine prognostic factors for stroke and death. This will allow for a stronger, current, and unified management recommendation applicable in our setting.

2 | MATERIALS AND METHODS

This retrospective multicenter analysis of patients with PCAI examined data from three large public trauma centers in three provinces of South Africa: Charlotte Maxeke Johannesburg Academic Hospital (CMJAH), Groote Schuur Hospital (GSH) in Cape Town, and the Pietermaritzburg Metropolitan Trauma Service (PMTS). Patients presenting with PCAI to any of the study sites between January 01, 2012 and December 31, 2020 were identified from the hospital registries. At CMJAH, patients were identified from a paper-based trauma registry (Medibank), at GSH from an electronic trauma registry (REDCap) and at PMTS from the Hybrid Electronic Medical Registry. Data were merged onto an Excel spreadsheet. Additional data were retrieved from in-patient files when needed.

Only patients with PCAI involving the common carotid artery (CCA) or ICA were included. Demographic and clinical data pertaining to the injury mechanism, the involved neck zone, the injured segment of the carotid artery, whether the injury was occlusive or non-occlusive, physiological parameters at admission (airway and hemodynamic status, shock index (SI), signs of vascular injury, and neurological status), injury severity score (ISS), clinical investigations and findings, management, and outcome (length of hospital stay (LOS), stroke, and mortality) were analyzed.

3 | GENERAL MANAGEMENT

A "no-zone" approach to PNI based on clinical findings of injury and imaging with CTA is followed in the three centers.[23] Intra-operative shunts, measurements of intracranial pressure (ICP) or stump pressure measurement as a surrogate for ICP, are not routinely used, although a measure of ICP may play a role in patients with neurological deficits and coma. This requires further investigation. Systemic heparin is generally given at the time of vascular control and prophylactic Low Molecular Weight Heparin (LMWH) post operatively. Patients managed by nonoperative management (NOM) or by endovascular intervention are likewise commenced on prophylactic LMWH and generally discharged on antiplatelet therapy. Endovascular stents are deployed in angio-suits at the discretion of consulted vascular surgeons.

4 | DEFINITIONS

We defined an occlusive carotid injury as either an occlusion seen on CTA or as thrombosed or complete transection of the vessel on exploration. A pseudoaneurysm, intimal flap, dissection, or an arteriovenous fistula (AVF) seen on CTA or incomplete transection seen on exploration were defined as nonocclusive injuries. Emergency surgery was defined as surgery without imaging, undertaken for patients with hemodynamic instability and/or ongoing bleeding, and urgent surgery as surgery after imaging. Coma was defined as a Glasgow Coma Scale (GCS) of $\leq$8. Gross localizing signs (LS) were defined as clinical findings of lateralizing neurological fall out in keeping with contralateral cerebral ischemia from a PCAI, in

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patients with GCS >8. Neurological devastating injuries were defined as severe neurological injuries based either on radiological findings on CTB of extensive cerebral infarction and raised ICP or extensive traumatic changes and/or a clinical persistent vegetative state with GCS of 3.

5 | DATA ANALYSIS AND ETHICS

Quantitative methods were used, and all statistical tests were performed using STATA SE17/0. Relationships between categorical variables were examined using Pearson's chi-square, or the Fisher's exact test when frequencies were less than 5%. Relationships between numerical variables were analyzed using the Mann–Whitney *U* test. Univariate logistic regression confirmed the prognostic value of associated factors. The significance level was set at $P < 0.05$. The study was approved by institutional review boards at each hospital and ethics approval was granted by the three institutions' ethics committees (University of the Witwatersrand: M211033, University of Cape Town: 754/2022, University of KwaZulu-Natal: BCA 221/13). Additionally, the study was registered with the National Health Research Database for the three provinces (Gauteng, the Western Cape and KwaZulu-Natal).

6 | RESULTS

There were 149 patients (CMJAH: 63, GSH:30, and PMTS:56) with 105 CCA injuries and 44 ICA injuries. Eleven patients were palliated from the time of admission due to neurological devastating injuries and have been excluded from the complete analysis except that of the prognostic factors as indicated. In addition, one patient who died from associated injuries before receiving intervention for PCAI was excluded. The remaining 137 patients were actively managed and included. Of these, 28 patients (20.9%) had occlusive injuries, 24 (17.9%) presented in coma, and 12 (9.0%) had LS. The median time from injury to admission was 7.40 h (IQR: 2.98–16.78), with only six patients presenting within 1 hour from time of injury.

One hundred and twenty patients (87.6%) underwent CTA at admission. As illustrated in Figure 1, the use of CTA increased over the study period. Additionally, 34 patients (24.8%) had CT imaging of the brain (CTB). CTB revealed cerebral infarcts in five patients and severe cerebral edema without infarct in one patient. The characteristics of the study patient population are shown in Table 1 with patients additionally being grouped into those managed with intervention (endovascular/open surgery) and those managed with NOM. Multivariate logistic regression showed that only injuries to the CCA and hard vascular signs were significant independent predictors of need for intervention ($P < 0.001$). The odds of requiring intervention were 4.74 (95% CI 2.24–3.29) times higher with CCA versus ICA and 2.91 (95% CI 1.37–2.27) times higher with hard versus soft signs.

Associated injuries were present in 112 patients (81.8%) and are listed in Table 2. A detailed description of the management of these various injuries lies outside the scope of this paper although most aero-digestive and venous injuries were managed at the time of neck exploration for PCAI.

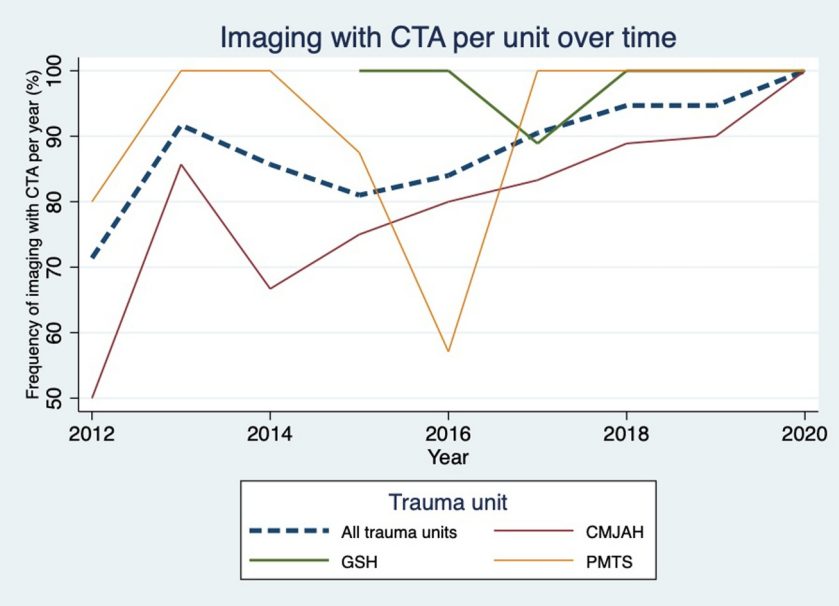


FIGURE 1 Imaging with CTA per unit over time. CMJAH, Charlotte Maxeke Johannesburg Academic Hospital; GSH, Groote Schuur Hospital; PMTS, Pietermaritzburg Metropolitan Trauma Service.

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TABLE 1 Study patient population and characteristics ($n = 137$).

Category	Variable	All ($n = 137$)[a]	Intervention ($n = 107$)[a]	NOM ($n = 30$)[a]
Demographics	Age, years	28 (24–35)	27 (23–34)	32 (26–36)
	Men	139 (94.2)	103 (96.3)	26 (86.7)
Mechanism of injury	GSW	42 (30.7)	29 (27.1)	13 (43.3)
	SW	95 (69.3)	78 (72.9)	17 (56.7)
Zones of the neck	I	17 (12.4)	13 (12.1)	4 (13.3)
	II	66 (48.2)	60 (56.1)	6 (20.0)
	III	28 (20.4)	19 (17.8)	9 (30.0)
	Posterior	3 (2.2)	2 (1.9)	1 (10.0)
	Multiple	21 (15.3)	13 (12.1)	8 (26.7)
	Infraclavicular	2 (1.5)	0	2 (6.7)
Anatomic segment of carotid artery injured	CCA	97 (70.8)	83 (77.6)	14 (46.7)[b]
	ICA	40 (29.2)	24 (22.4)	16 (53.3)
Patency of injured vessel	Occlusive	28 (20.9)	20 (19.2)	8 (26.7)
	Nonocclusive	106 (79.1)	84 (80.8)	22 (73.3)
	Not categorized	3	3	0
Time delay from injury to presentation	0–6 h	49 (47.6)	40 (52.6)	9 (33.3)
	>6–12 h	19 (18.5)	13 (17.1)	6 (22.2)
	>12 h	35 (34.0)	23 (30.3)	12 (44.4)
	Not categorized	34	31	3
Airway	Intervention for threatened airway	49 (35.8)	41 (38.3)	8 (26.7)
	Endotracheal tube	47 (95.9)	39 (95.1)	8 (100)
	Surgical airway	2 (4.1)	2 (4.9)	0
Breathing	RR, bpm	18 (15–20)	18 (16–20)	18 (15–20)
	Oxygen saturation, %	98 (96–100)	98 (96–100)	96 (94–100)
Circulation	HR	90 (80–108)	90 (78–108)	89 (82–111)
	SBP<90 mmHg	20 (14.6)	18 (16.8)	2 (6.7)
	Signs of vascular injury			
	Hard	80 (63.0)	68 (68.0)	12 (44.4)[b]
	Soft	47 (37.0)	32 (32.0)	15 (55.6)
	Not categorized	10	7	3
	FCBT	24 (17.5)	21 (19.6)	3 (10.0)
Disability	GCS	15 (10–15)	15 (9–15)	15 (14–15)
	Coma (GCS ≤8)	24 (17.9)	22 (21.0)	2 (6.9)
	Gross localizing signs but no coma	12 (9.0)	10 (9.5)	2 (6.9)
	No gross localizing signs or coma	98 (73.1)	73 (69.5)	25 (86.2)
	Not categorized	3	2 (1.9)	1
Exposure	Temperature, degree C	36.4 (35.9–36.6)	36.4 (35.8–36.7)	36.3 (36.0–36.5)
Imaging on admission	CTA neck	120 (87.6)	90 (84.1)	30 (100)
	CTB	34 (24.8)	19 (17.8)	15 (50.0)
	CTB positive for infarct or edema	6 (17.6)	6 (31.5)	0
	Catheter directed angiogram	22 (16.1)	22 (20.6)	0
	Duplex Doppler	3 (2.2)	1 (0.9)	2 (6.7)

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TABLE 1 (Continued)

Category	Variable	All (n = 137)[a]	Intervention (n = 107)[a]	NOM (n = 30)[a]
Trauma scores	ISS	20 (16–25)	18 (16–25)	20 (16–25)
	SI	0.73 (0.63–0.89)	0.74 (0.64–0.91)	0.70 (0.63–0.80)
Biochemistry	pH	7.36 (7.30–7.41)	7.36 (7.3–7.4)	7.35 (7.32–7.42)
	pCO2	36.4 (32.7–44.1)	36.0 (32.6–44.5)	38.0 (33.5–42.9)
	HCO3 nmol/L	22.1 (18.8–24.3)	22.1 (18.8–24.3)	22.3 (18.3–24.4)
	Lactate nmol/L	2.5 (1.2–4.9)	2.3 (1.2–5.0)	2.7 (1.1–4.5)
	Hgb g/dL	10.9 (9.4–13)	10.8 (9.4–13.0)	12.1 (9.9–13.7)

Abbreviations: CCA, common carotid artery; CTA, computed tomography angiography; CTB, computed tomography of the brain; FCBT, Foley catheter balloon tamponade; GCS, Glasgow Coma Scale; GSW, gunshot wound; HCO3, bicarbonate; Hgb, hemoglobin; HR, heart rate; ICA, internal carotid artery; ISS, Injury Severity Score; pCO2, partial pressure of carbon dioxide; RR, respiratory rate; SBP, systolic blood pressure; SI, Shock Index; SW, stab wound.

[a]Values are given as median and interquartile range (IQR) for continuous data (unless specified otherwise) and frequencies (n) and percentages for categorical data.

[b]Multivariate logistic regression showed that only injuries to the CCA and hard vascular signs were significant independent predictors of need for intervention ($P < 0.001$). The odds of requiring intervention were 4.74 (95% CI 2.24–3.29) times higher with CCA versus ICA and 2.91 (95% CI 1.37–2.27) times higher with hard versus soft signs.

TABLE 2 Cervical and extra-cervical associated injuries (n = 112).

Cervical: 96 patients (70.1%)			**Extra-cervical: 55 patients (40.1%)**				
Vascular: 74 patients	**Aero-digestive: 21 patients**	**Others: 36 patients**	**Head: 6 patients**	**Max-fax: 26 patients**	**Thoracic: 29 patients**	**Abdominal: 2 patients**	**Limbs: 5 patients**
Venous: 65 patients	Trachea: 1	Spinal column: 27	Skull fractures: 4	Facial fractures: 22	Haemo-pneumo thoraces: 21	Portal vein and hepatic artery: 1	Upper limb fractures: 2
IJV: 62	Esophagus: 7	Spinal cord: 7	Intracranial hemorrhage or contusion: 3	Salivary glands: 2	Spine fracture: 3	Liver: 1	Brachial artery: 1
EJV: 2	Pharynx: 13	Brachial plexus: 3		Facial nerve: 3	Spinal cord: 1	Large bowel: 1	Axillary nerve: 1
Facial vein: 1		Vagus nerve: 4		Eye injuries: 3	Clavicle fracture: 1	Small bowel: 1	Median nerve: 1
Arterial: 16 patients		RLN: 2			Brachiocephalic vein: 3	Spine fracture: 1	Radial nerve: 1
ECA: 10		Hypoglossal nerve: 1			Brachiocephalic artery: 3	Spinal cord: 1	
VA: 6		Thoracic duct: 1			SCA: 3		
Facial artery: 1							
Maxillary artery: 1							
Lingual artery: 1							

Abbreviations: ECA, external carotid artery; EJV, external jugular vein; IJV, internal jugular vein; RLN, recurrent laryngeal nerve; SCA, subclavian artery; SCV, subclavian vein; VA, vertebral artery.

*Some patients had more than one associated injury and 39 patients had both cervical and extra-cervical injuries.

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6.1 | Nonoperative management

Thirty patients (21.9%) were treated medically (ICA:16, CCA:14). Eight patients had occlusive (ICA:7), and 22 had nonocclusive injuries (AVF:1, intimal flap:18, dissection:1, small pseudoaneurysms: 2). Of these medically treated patients only two presented in coma (6.7%) and two had LS (6.7%) but normal CTB.

6.2 | Open surgery

Eighty-seven patients (63.5%) underwent open surgery (CCA: 66, ICA: 21), with only 17 of these patients (19.5%) taken for emergency exploration. Nineteen injuries were occlusive (21.8%), 65 injuries (74.7%) were nonocclusive (pseudoaneurysm: 37, extravasation of contrast: 3, AVF with pseudoaneurysm: 3, AVF: 6, intimal flap: 6, partial transection: 10) and three injuries could not be categorized. Seventeen of the open surgery patients (19.5%) presented in coma and eight patients (9.2%) with LS. The index procedures were primary repair ($n = 51$), interposition grafts including vein grafts (VG; $n = 5$), synthetic grafts ($n = 15$), combined VG and synthetic graft ($n = 1$), venous patch repair ($n = 3$), temporary intravascular shunt (TIVS; $n = 2$), and ligations ($n = 10$).

6.3 | Endovascular management

Twenty hemodynamically normal patients (14.6%) were managed with endovascular stents (ICA: 3, CCA: 17). One additional stent was later deployed in a patient with a pseudoaneurysm, 12 days after open repair. All patients had prior imaging with CTA. Nineteen of these 20 patients (95.0%) had nonocclusive injuries (pseudoaneurysm: 14, intimal flap: 2, dissection: 1, pseudoaneurysm and AVF: 2), and one patient had an occlusive injury (thrombus with pseudoaneurysm). Five patients (25.0%) presented in coma, but with normal CTB and two with LS (10.0%).

6.3.1 | Outcomes

During the study period, the median (IQR) LOS was 6 (4–10) days and 18 (13.1%) of the actively managed patients died. Of those, two patients (11.1%) died secondary to associated injuries, with one patient having been managed with primary repair, dying from multiple organ failure after concomitant penetrating hepatic vascular injury and another having been managed endovascularly, dying due to quadriplegia-related pneumonia. One neurologically intact patient (5.6%) died from nosocomial pneumonia 18 days after ligation of a thrombosed ICA. Finally, 15 patients (83.3%) died from neurological devastating injuries after intervention for PCAI. One of these died after endovascular stenting and 14 after open surgery (reperfusion: 10, ligation: 4). Of these 15 patients, 12 (80%) presented with severe neurological deficits (coma: 9, LS:3).

Incorporating the 12 excluded patients who died without receiving active management the overall mortality of PCAI was 20.1%.

Of the 119 patients (86.9%) who survived, 15 (12.6%) had a stroke. Of these 15 patients, 10 (66.7%) presented with severe neurological deficits (LS: 7, coma: 3) and CTB on admission confirmed infarcts in four out of nine scanned patients. An additional 15 surviving patients (12.6%) had complications, which included seven patients with nosocomial sepsis, one with a retained hemothorax and seven with other vascular complications not resulting in strokes (type I endoleak:1, progression of pseudoaneurysms: 2, rebleed: 4).

Eight of 40 patients (20.0%) with injury to the ICA died, and four of 32 survivors (12.5%) had a stroke. Of the 97 patients with injury to the CCA, 10 (10.3%) died, and of 87 survivors, 11 (12.6%) had a stroke.

Management methods related to mortality and stroke is illustrated in Table 3, with emergency surgery and ligation being significantly associated with death. Urgent surgery and reperfusion were statistically associated with survival. No statistically significant association between management and stroke in survivors was found (Table 3).

The mortality and stroke rate of the six patients presenting within 1 hour from time of injury was 16.6% (1/6) and 20.0% (1/5), respectively. Forty-nine patients were presented within six hours, 54 only after six hours and for 34 patients the time of injury was not recorded. For those presenting within 6 hours from time of injury, the mortality rate and stroke rate was 12.2% (6/49) and 7.0% (3/43) as opposed to 13.0% (7/54) and 19.9% (9/47) for those presenting more than 6 hours after injury, although these differences were not statistically significant ($P = 0.575$ and $P = 0.123$, respectively) these may be due to small numbers.

An overview of the above results is illustrated in Figure 2.

6.4 | Prognostic factors for poor outcome

Prognostic factors for death and stroke were investigated by including all mortalities except patients dying from associated injuries. This is illustrated in Table 4. A mechanism of GSW, occlusive injuries, a threatened airway, a SBP <90 mmHg, hard signs of vascular injury, a low GCS, coma, a CTB demonstrating infarct,

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TABLE 3 Patient management according to outcome (n = 137).

Parameter	All	Died (n = 18)	Alive (n = 119)	p value	Alive stroke (n = 15)	Alive no stroke (n = 104)	p value
Management							
NOM, n (%)	30 (21.9)	0 (0%)	30 (25.2)	0.019	2 (13.3)	28 (26.9)	0.536
Endovascular, n (%)	20 (14.6)	2 (11.1)	18 (15.1)		2 (13.3)	16 (15.4)	
Open surgery, n (%)	87 (63.5)	16 (88.9)	71 (59.7)		11 (73.3)	60 (57.7)	
Emergency, n (%)[a]	17 (19.5)	9 (56.3)	8 (11.3)	<0.001	2 (18.2)	6 (10.0)	0.601
Urgent, n (%)[a]	70 (80.5)	7 (43.8)	63 (88.7)		9 (81.8)	54 (90.0)	
Open reperfusion, n (%)[b]	77 (88.5)	11 (68.8)	66 (93.0)	0.016	11 (100)	55 (91.7)	1.000
Open ligation, n (%)	10 (11.5)	5 (31.3)	5 (7.0)		0	5 (8.3)	

Abbreviation: NOM, nonoperative management.

[a]Emergency surgery was defined as surgery without imaging and urgent surgery as surgery after imaging.

[b]Included all patients managed with primary repair, interposition graft, venous patch and temporary intravascular shunt.

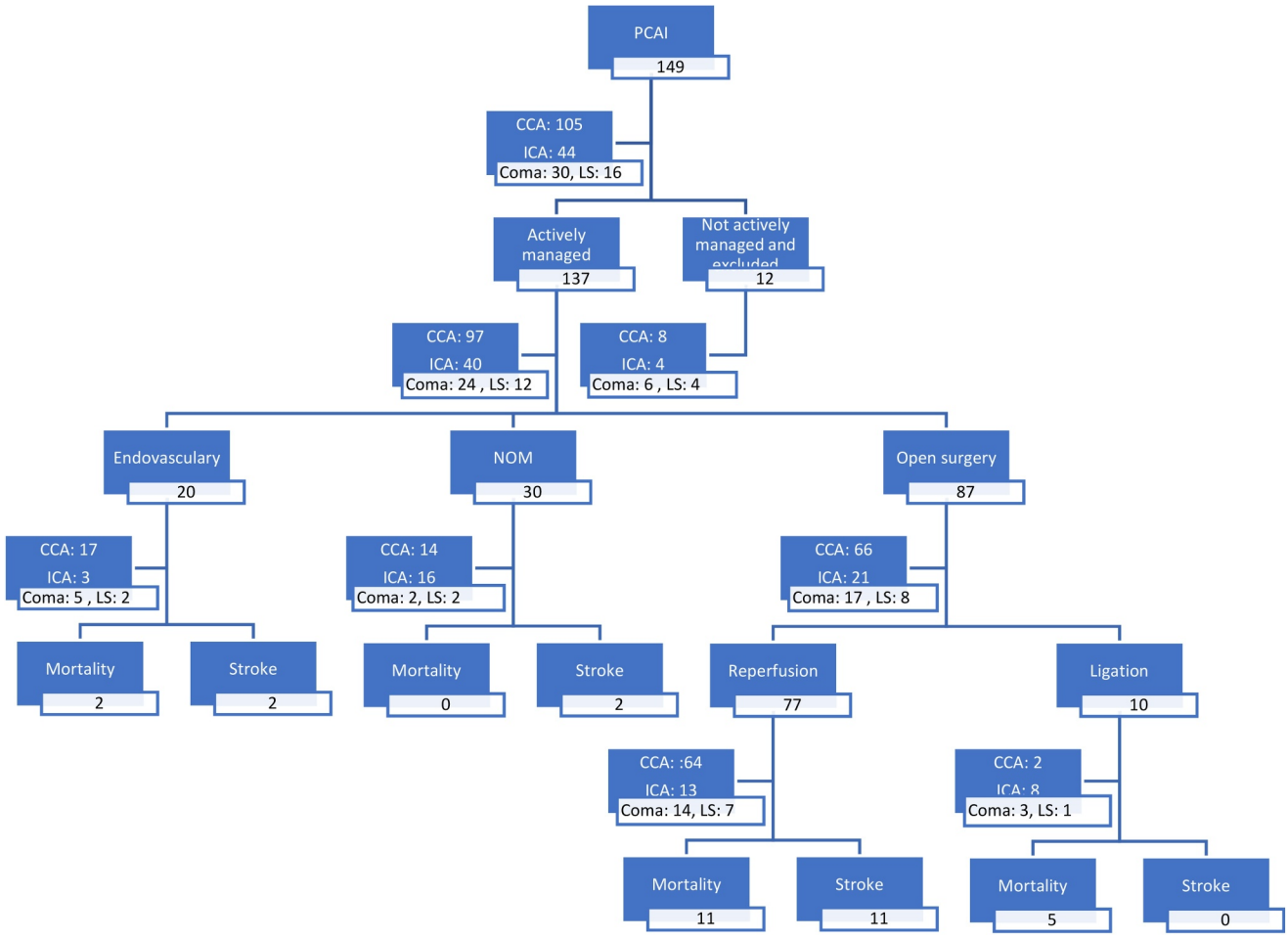


FIGURE 2 Overview of results. CCA, common carotid artery; ICA, internal carotid artery; LS, gross localizing signs; NOM, nonoperative management; PCAI, penetrating carotid artery injury.

a high ISS and SI, a low pH or HCO3, and an elevated lactate were significant independent prognostic factors for death. When again excluding the initially excluded patients from the analysis, a CTB demonstrating infarct, a SBP <90 mmHg and a high SI failed to show statistical significance as prognostic factors.

Statistically significant prognostic factors for stroke were: a threatened airway, SBP <90 mmHg, hard signs

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TABLE 4 Prognostic factors for outcome ($n = 144$)[a].

Parameter	All	Died ($n = 25$)	Alive ($n = 119$)	p value	Alive stroke ($n = 15$)	Alive no stroke ($n = 104$)	p value
Demographics							
Age, (years), median (IQR)	28.0 (24.0–35.0)	29.5 (24.0–36.5)	28.0 (24.0–34.0)	0.603	29.0 (26.0–35.0)	27.5 (23.0–34.0)	0.324
Men, *n* (%)	135 (93.8)	24 (96.0)	111 (93.3)	>0.999	14 (93.3)	97 (93.3)	1.000
Mechanism of injury							
GSW, *n* (%)	44 (30.6)	13 (52.0)	31 (26.1)	0.010	2 (13.3)	29 (27.9)	0.348
SW, *n* (%)	100 (69.4)	12 (48.0)	88 (74.0)		13 (86.7)	75 (72.1)	
Anatomic segment of artery							
CCA, *n* (%)	103 (71.5)	16 (64.0)	87 (73.1)	0.359	11 (73.3)	76 (73.1)	1.000
ICA, *n* (%)	41 (28.5)	9 (36.0)	32 (26.9)		4 (26.7)	28 (26.9)	
Patency of vessel							
Occlusive, *n* (%)	31 (22.0)	11 (45.8)	20 (17.1)	0.002	3 (20.0)	17 (16.3)	0.720
Non-occlusive, *n* (%)	110 (78.0)	13 (54.2)	97 (82.9)		12 (80.0)	85 (81.7)	
Time delay from injury to presentation							
0–6 h, *n* (%)	53 (49.5)	10 (58.8)	43 (47.8)	0.252	3 (25.0)	40 (51.3)	0.026
>6–12 h, *n* (%)	18 (16.8)	4 (23.5)	14 (15.6)		5 (41.7)	9 (11.5)	
>12 h, *n* (%)	36 (33.6)	3 (17.7)	33 (36.7)		4 (33.3)	29 (37.2)	
Presenting features							
Airway intervention, *n* (%)	55 (38.2)	20 (80.0)	35 (29.4)	<0.001	8 (53.3)	27 (26.0)	0.030
HR, median (IQR)	91 (80–110)	98 (78–118)	90 (80–108)	0.470	91 (85–117)	90 (80–107)	0.398
SBP <90 mmHg, *n* (%)	25 (17.4)	9 (36.0)	16 (13.4)	0.007	5 (33.3)	11 (10.6)	0.016
Signs of injury							
Hard signs, *n* (%)	88 (61.1)	25 (100.0)	63 (57.3)	<0.001	11 (84.6)	52 (53.6)	0.039
Soft signs, *n* (%)	47 (32.6)	0	47 (42.7)		2 (15.4)	45 (46.4)	
Admission GCS, median (IQR)	15 (10–15)	7 (6–11)	15 (12–15)	<0.001	13 (9–15)	15 (14–15)	0.038
Neurological status on admission							
Coma, *n* (%)	29 (20.1)	15 (60.0)	14 (12.1)	<0.001	3 (21.4)	11 (78.6)	<0.001
LS, *n* (%)	16 (11.1)	7 (28.0)	9 (7.8)		7 (77.8)	2 (22.2)	
No coma or LS, *n* (%)	96 (66.7)	3 (12.0)	93 (80.2)		5 (5.4)	88 (94.6)	
Imaging on admission							
CTA neck, *n* (%)	127 (88.2)	16 (64.0)	111 (93.3)	<0.001	13 (86.7)	98 (94.2)	0.265
CTB positive for infarct or edema, *n* (%)	15 (10.4)	10 (40.0)	5 (4.2)	<0.001	4 (26.7)	1 (1.0)	0.017
Trauma scores							
Injury severity score, median (IQR)	20 (16–25)	29 (25–51)	17 (16–25)	<0.001	24 (16–25)	17 (16–25)	0.135
Shock index, median (IQR)	0.74 (0.64–0.92)	0.82 (0.68–1.29)	0.73 (0.64–0.88)	0.042	0.92 (0.74–1.16)	0.73 (0.63–0.83)	0.004
Biochemistry,							
pH, median (IQR)	7.36 (7.30–7.41)	7.30 (7.14–7.39)	7.36 (7.32–7.41)	0.009	7.32 (7.23–7.38)	7.37 (7.32–7.41)	0.077

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TABLE 4 (Continued)

Parameter	All	Died ($n = 25$)	Alive ($n = 119$)	p value	Alive stroke ($n = 15$)	Alive no stroke ($n = 104$)	p value
HCO3 (nmoL/L), median (IQR)	22.2 (18.4–24.6)	20.1 (14.6–23.5)	22.7 (19.3–24.7)	0.031	21.1 (17.6–24.7)	22.7 (20.2–24.4)	0.483
Lactate (nmoL/L), median (IQR)	2.5 (1.2–5.3)	4.9 (2.3–7.1)	2.3 (1.2–4.5)	0.003	5.7 (1.4–6.5)	2.2 (1.1–4.1)	0.129
Hgb (g/dL), median (IQR)	10.9 (9.4–13.0)	9.7 (8.8–12.9)	11 (9.6–13.0)	0.178	10.4 (8.9–10.9)	11.6 (9.8–13.3)	0.049

Abbreviations: CCA, common carotid artery; CTA, computed tomography angiography; CTB, computed tomography of the brain; GCS, Glasgow Coma Scale; GSW, gunshot wound; HCO3, bicarbonate; Hgb, hemoglobin; HR, heart rate; ICA, internal carotid artery; ISS, Injury Severity Score; SBP, systolic blood pressure; SI, Shock Index; SW, stab wound.

[a]Excluded 5 patients who died from associated injuries. Univariate logistic regression confirmed the prognostic value of associated factors except for admission GCS, pH, and Hgb as prognostic factors for stroke.

of vascular injury, LS, CTB demonstrating infarct, a high SI, as well as a delay from injury to presentation between 6 and 12 h.

The increasing use of imaging with CTA on admission over the study period had no effect on outcome of mortality or stroke (data not shown).

6.5 | Outcome for patients with severe neurological deficits

This study included only 37 patients with coma, LS and/or a CTB positive for infarct and/or severe cerebral edema. Only four of these patients were managed with ligation, which resulted in a mortality rate of 100% (coma: 3/3, LS: 1/1). Compared to ligation, reperfusion procedures, either endovascular or open, resulted in a significant lower overall mortality rate of only 31% (9/29) ($P = 0.017$) with 63.2% (12/19) of patients in coma and 77.8% (7/9) of patients with LS surviving, albeit with a high overall stroke rate of 50% (10/20) (coma: 25.0% (3/12), LS: 85.7% (6/7)). Even for patients with established infarcts and/or severe edema documented on CTB on admission, a reperfusion procedure was associated with survival in 83.3% (5/6), although only one of these patients, who had isolated gross cerebral edema on CTB, had full neurological recovery at time of discharge. For patients with severe neurological deficits presenting more than 6 hours from the time of injury, 58.3% (7/12) survived after reperfusion although with a stroke rate of 71.4% (5/7) among survivors as opposed to a mortality rate of 100% with ligation (1/1). For patients presenting within 6 hours from the time of injury, reperfusion resulted in survival in 62.5% (5/8), with 40.0% (2/5) of survivors having strokes, as opposed to ligation, where all patients died (3/3).

Despite a strong trend demonstrated toward improved outcomes with reperfusion procedures compared to ligation and some of the results obtaining statistical significance, the total number of patients included were small and p values should be read with caution. Additionally, all four hemodynamically stable patients in this subgroup managed with NOM survived, with only one patient having a stroke. However, none of these four patients had infarcts on CTB done on admission. The above results are illustrated in Table 5.

7 | DISCUSSION

PCAI remains associated with a high morbidity and mortality rate.[1–4] Table 6 summarizes the South African and the recent international literature on PCAI.[3–5,8,9,17,19,21,22] Our overall mortality rate of 20.1%, with approximately 80% dying from neurologically devastating injuries and a stroke rate of 12.6% in survivors, is in keeping with this.[1]

Injuries to the ICA have a higher reported mortality and stroke rate than those to the CCA.[2–4] Although our data also showed a higher mortality rate with injuries to the ICA compared to the CCA (20.0% vs. 10.3%), this was not statistically significant once adjusted for deaths unrelated to PCAI. A significant time delay from injury to admission was observed in our study and our results probably demonstrates an element of natural selection with some of the most unstable patients presumably not surviving to hospital admission. This seems to be reflected in the observation that the mortality rate between patients presenting before and after 6 h from time of injury was similar although the late presenters had an almost three-fold increase in stroke rate.

Minor carotid arterial injuries in asymptomatic patients are often treated with observation with or without anticoagulation.[5,18,20,24] Our data suggests that NOM is suitable for hemodynamically stable patients without neurological deficits or with a normal CTB, and who have occlusive injuries, especially to the ICA, or non-occlusive minimal arterial injuries. For these patients, the stroke rate among survivors was only 6.7%.

Endovascular management of PCAI seems to be an attractive option for very distal and proximal injuries but the literature is limited.[5,8,9] A recently published review

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TABLE 5 Characteristics and outcomes of patients with severe neurological deficits ($n = 37$).

Parameter	All	Died ($n = 13$)	Alive ($n = 24$)	p value	Alive stroke ($n = 11$)	Alive no stroke ($n = 13$)	p value
Anatomic segment of artery							
CCA, *n* (%)	23 (62.2)	7 (53.8)	16 (66.7)	0.443	7 (63.6)	9 (69.2)	>0.99
ICA, *n* (%)	14 (37.8)	6 (46.2)	8 (33.3)		4 (36.4)	4 (30.8)	
Patency of vessel							
Occlusive, *n* (%)	9 (25.0)	6 (50.0)	3 (12.5)	0.036	2 (18.2)	1 (7.7)	0.576
Non-occlusive, *n* (%)	27 (75.0)	6 (50.0)	21 (87.5)		9 (81.8)	12 (92.3)	
Time delay from injury to presentation							
0–6 h, *n* (%)	12 (44.4)	6 (50.0)	6 (40.0)	0.885	2 (25.0)	4 (57.1)	0.549
>6–12 h, *n* (%)	9 (33.3)	4 (33.3)	5 (33.3)		3 (37.5)	2 (28.6)	
>12 h, *n* (%)	6 (22.2)	2 (16.7)	4 (26.7)		3 (37.5)	1 (14.3)	
Presenting features							
Admission GCS, median (IQR)	7 (6–10)	7 (7–8)	7.5 (5.5–11)	0.641	10 (7–15)	6 (4–8)	0.007
Neurological status on admission							
Coma, *n* (%)	24 (64.9)	10 (76.9)	14 (58.3)	0.655	3 (27.3)	11 (84.6)	0.011
LS, *n* (%)	12 (32.4)	3 (23.1)	9 (37.5)		7 (63.6)	2 (15.4)	
No coma or LS, *n* (%)	1 (2.7) (GCS 10 and infarct on CTB)	0	1 (4.2)		1 (9.1)	0	
Imaging on admission							
CTA neck, *n* (%)	28 (75.7)	7 (53.8)	21 (87.5)	0.042	10 (90.9)	11 (84.6)	>0.99
CTB, *n* (%)	17 (45.9)	3 (23.1)	14 (58.3)	0.082	8 (72.7)	6 (46.2)	0.240
CTB positive for infarct/edema, *n* (%)	6 (35.3)	1 (33.3)	5 (35.7)	>0.99	4 (50.0)	1 (16.7) (edema)	0.301
Management							
NOM, *n* (%)	4 (10.8)	0	4 (16.7)	0.110	1 (9.1)	3 (23.1)	0.124
Endovascular, *n* (%)	7 (18.9)	1 (7.7)	6 (25.0)		1 (9.1)	5 (38.5)	
Open surgery, *n* (%)	26 (70.3)	12 (92.3)	14 (58.3)		9 (81.8)	5 (38.5)	
Emergency, *n* (%)[a]	9 (34.6)	6 (50.0)	3 (21.4)	0.218	1 (11.1)	2 (40.0)	0.505
Urgent, *n* (%)[a]	17 (65.4)	6 (50.0)	11 (78.6)		8 (88.9)	3 (60.0)	
Open reperfusion, *n* (%)[b]	22 (84.6)	8 (66.7)	14 (100)	0.033	9 (100)	5 (100)	
Open ligation, *n* (%)	4 (15.4)	4 (33.3)	0		0	0	
Ligation versus any reperfusion[c]							
Any reperfusion, *n* (%)	29 (87.9)	9 (69.2)	20 (100)	0.017	10 (100)	10 (100)	
Ligation, *n* (%)	4 (12.1)	4 (30.8)	0		0	0	
Ligation versus reperfusion if coma[c]							
Any reperfusion, *n* (%)	19 (86.4)	7 (70.0)	12 (100)	0.078	3 (100)	9 (100)	
Ligation (%)	3 (13.6)	3 (30.0)	0		0	0	
Ligation versus reperfusion if LS[c]							
Any reperfusion, (%)	9 (90.0)	2 (66.7)	7 (100)	0.300	6 (100)	1 (100)	
Ligation, *n* (%)	1 (10.0)	1 (33.3)	0		0	0	

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TABLE 5 (Continued)

Parameter	All	Died ($n = 13$)	Alive ($n = 24$)	p value	Alive stroke ($n = 11$)	Alive no stroke ($n = 13$)	p value
Ligation versus reperfusion if CTB positive[c]							
Any reperfusion, *n* (%)	6 (100)	1 (100)	5 (100)		4 (100)	1 (100) (edema)	
Ligation, *n* (%)	0	0	0		0	0	
Ligation versus reperfusion if > 6 h delay[c]							
Any reperfusion (%)	12 (92.3)	5 (83.3)	7 (100)	0.462	5 (100)	2 (100)	
Ligation, *n* (%)	1 (7.7)	1 (16.7)	0		0	0	
Ligation versus reperfusion if 0–6 h delay[c]							
Any reperfusion (%)	8 (72.7)	3 (50.0)	5 (100)	0.182	2 (100)	3 (100)	
Ligation, *n* (%)	3 (27.3)	3 (50.0)	0		0	0	

Abbreviations: CCA, common carotid artery; CTA, computed tomography angiography; CTB, computed tomography of the brain; GCS, Glasgow Coma Scale; ICA, internal carotid artery; LS, gross localizing signs; NOM, nonoperative management; SBP, systolic blood pressure.

[a]Emergency surgery was defined as surgery without imaging and urgent surgery as surgery after imaging.

[b]Included all patients managed with primary repair, interposition graft, venous patch, and temporary intravascular shunt.

[c]Included both patients managed with open reperfusion and endovascular stenting.

of patients with PCAI, from the American National Trauma Data Bank from 2002 to 2016, included 3391 patients, of which only 154 (4.5%) were managed endovascularly compared to 1192 (35.2%) with open surgery.[5] There was, however, no significant difference in the outcome between these groups. Our data suggests that endovascular management has a role in managing hemodynamically stable patients with nonocclusive injuries such as pseudoaneurysms and AVF.

The majority of PCAI can be repaired primarily and sometimes with an interposition graft.[1,17,24] Only 10 patients were managed with ligation, but the mortality rate was 50%.

The management of patients with severe neurological deficits remains controversial. The concern persists that restoring perfusion may convert an ischemic infarction to a hemorrhagic one or precipitate severe cerebral edema.[2,3,10–22] Although recognizing coma as an independent risk factor for poor outcomes, several authors suggest restoring arterial continuity even in patients with coma as in the acute clinical setting it can be difficult to reliably distinguish between the various causes of a depressed mental status.[2–4,15–18,20,22] In a 1989 study from Baragwanath Hospital, Johannesburg, Demetriades' group demonstrated that the most common autopsy finding in patients post PCAI repair was cerebral edema.[19] They advised against repair in patients with established ischemic infarcts as well as cerebral edema. Navsaria et al. in 2002 reported on their experience at GSH in Cape Town and suggested investigation with CTB of all stable patients with delayed presentation and central neurological deficits or prolonged coma (4–6 h) and to ligate patients with established infarcts or edema.[21] Conversely, Du Toit et al. in 2003, reporting on their experience with PCAI from Tygerberg Hospital in 2003, reported no improvement in preoperative neurological deficits after ligation compared to an improvement of 80% after repair.[3] Although cerebral infarcts were confirmed in 25 patients at autopsy and five of these were hemorrhagic in nature and found in patients post repair, they argued that the occurrence of such infarcts cannot be predicted and advised against ligation. In a more recent review by White et al., on their experience with PCAI in the wars in Iraq and Afghanistan (2002–2015), recommendations were made for the vascular restoration of these injuries whenever possible.[4] However, their data did not include preoperative neurological status of the patients. A similar recent report by Reva et al., mainly on experiences from combat operations in Chechnya (1999–2002) and including 46 patients, reported a 100% poor outcome when ligation was undertaken, defined as stroke or death, compared to 30% poor outcome when repair was undertaken.[22] Of the five patients in this report with severe neurological deficits preoperatively who died after repair, hemorrhagic stroke was found as the cause of death in one patient and brain edema in the remaining four. Despite these findings, the authors recommended arterial repair even in the presence of prolonged preoperative neurologic damage.

Similarly, to the latter studies, we did not identify any situations where ligation of PCAI is associated with a better outcome. Our study showed a significant relationship between ligation and death and between reperfusion and survival in general. Still, it did not include enough patients with severe neurological deficits to provide strong statistical evidence for the management for this subgroup of patients, although a trend toward improved outcome certainly was observed with reperfusion. For this subgroup, all patients managed with ligation died. In contrast, a majority of the 29 patients managed with reperfusion survived (69%),

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TABLE 6 Summary of the South African literature on PCAI and additionally of the international literature in the 21st century.

Author, year and country	PCAI (ICA and CCA)	Coma	LS but no coma	NOM	Open ligation	Open reperfusion	Endovascular	Mortality	Alive with stroke	Mortality: open ligation	Mortality: open reperfusion	Alive with stroke: open ligation	Alive with stroke: open reperfusion
Robbs et al., 1985, South Africa[17]	52 (+19 ECA, 10 VA, 4 BA)	7	15	0	7	45	0	9.6% (5/52)	17.0% (8/47	28.6% (2/7)	6.7% (3/45	20% (1/5)	16.7% (7/42)
Demetriades et al., 1989, South Africa[19]	114 (+10 ECA)	5	6	66 (dead on arrival)	2	46	0	68.4% (78/114)	5.6% (2/36)	0% (0/2)	26.1% (12/46)	0% (0/2)	6.3% (2/32)
Navsaria et al., 2002, South Africa[21]	29 (+3 ECA)	1	4	0	3	26	0	6.9% (2/29)	14.8% (4/27)	33.3% (1/3)	3.8% (1/26)	50% (1/2)	12% (3/25)
Du Toit et al., 2003, South Africa[3]	130 (+21 BA)	26[a]	13[a]	0	20[a]	131[a]	0	18.4% (24/130)	16.0% (17/106)	45%[a] (9/20)	17.6[a] (23/131)	18.2%[a] (2/11)	14.8[a] (16/108)
Du Toit et al., 2009, South Africa[8]	19	1	2	0	0	0	19	5.3% (1/19)	11.1% (2/18)	NA	NA	NA	NA
Herrera et al., 2011, Columbia & USA[9]	21 (+15 ECA)	?	3	0	0	0	21	0%	14.3%[b] (3/21)	NA	NA	NA	NA
Reva et al., 2011, Russia, military report[22]	46	? (18 had GCS </ = 12)	6	0	9	37	0	28.3% (13/46)	21.2% (7/33)	44.4% (4/9)	24.3% (9/37)	100% (5/5)	7.1% (2/28)
White et al., 2020, USA, military report[4]	56 (+11 ECA)	?	?	7	9	38	2	10.7% (6/56)	34.0% (17/50)	22.2% (2/9)	10.5% (4/38)	?[c] 88.9% (8/9)	?[c] 34.2% (13/38)
Blitzer et al., 2020, USA, National Trauma Data Bank[5]	3911	1810	?	1976 (+520 who died in ED)	1192 had open surgery but procedures were not specified)		154 (+69 combined open and endovascular)	20.4%[d] (578/2830)	8.5%[d] (241/2830) for entire cohort)	17.9%[d] (55/308) for all open procedures		8.4%[d] (19/225) for all open procedures	
Current study, 2024, South Africa	137	24	12	30	10	77	20	13.1% (18/137)	12.6% (15/119)	50.0% (5/10)	14.3% (11/77)	0% (0/5)	16.7% (11/66)

Abbreviations: BA, brachiocephalic artery; CCA, common carotid artery; ED, emergency department; ICA, internal carotid artery; LS, gross localizing signs; MCA, middle cerebral artery; NOM, nonoperative management; VA, vertebral artery.

[a]For PCAI and brachiocephalic artery injuries combined as given data does not specify PCAI alone.

[b]The study includes 3 patients admitted with stroke 2nd to embolisms to the MCA from PCAI but reports no procedural related complication in patients with injuries to the ICA and CCA.

[c]Not specified for alive patients only.

[d]Calculations for outcome are based on data from propensity score matching tables and not from the total numbers given under management.

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although with high stroke rates (coma: 25%; LS: 86%). Even for patients with established infarcts and/or severe edema documented on CTB at time of admission reperfusion procedures resulted in an 83% survival although these patients were unlikely to have full neurological recovery. Those patients with extensive infarction and signs of raised ICP on CTB were selected for palliation.

Our suggested algorithm for PCAI is illustrated in Figure 3.

7.1 | Limitations of the study

The retrospective nature of this study, using data from three different trauma registries of different sizes, and an overall sample size too small to provide strong significant results for the subgroup analysis on management methods and outcome for patients with severe neurological deficits were obvious limitations to the study. Additionally, poor follow up by trauma patients in our setting makes it difficult to make conclusive remarks about the long-term outcomes. Finally, although applicable for the management of PCAI in high volume trauma settings like ours, we acknowledge that trauma in South Africa is unique and that our proposed management algorithm would have to be validated.

8 | CONCLUSION

Outcomes in PCAI, including patients with severe neurological deficit and stroke, are better when managed with vascular reperfusion, and ligation should be reserved for technically inaccessible bleeding injuries. Reperfusion holds the best promise of survival although stroke rates remain high in patients presenting with severe neurological deficits. NOM is a reasonable option for hemodynamically stable patients with no neurological deficits and non-occlusive 'minimal' arterial injuries or occlusive injuries to the ICA. Endovascular intervention for PCAI can be considered in hemodynamically stable patients with pseudoaneurysms or AVF to the distal ICA or proximal CCA where

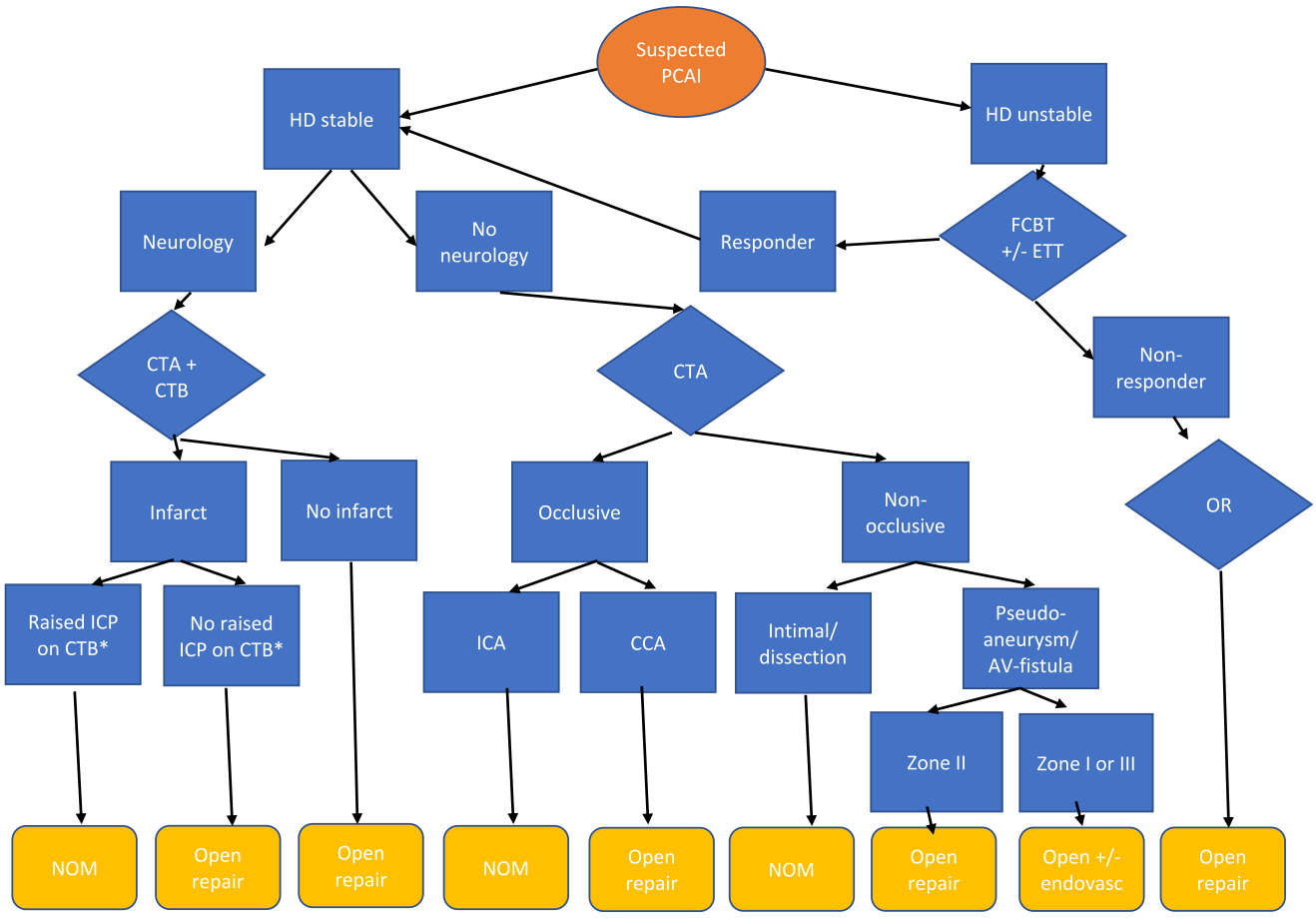


FIGURE 3 Suggested management algorithm for patients with PCAI. * Refers to radiological signs of raised ICP on CTB such as herniation, midline shift, ventricular/sulcal and cistern effacement. CCA, common carotid artery; CTA, computed tomography angiography; CTB, computed tomography of the brain; ETT, endotracheal tube; FCBT, Foley catheter balloon tamponade; HD, hemodynamically; ICA, internal carotid artery; ICP, intracranial pressure; OR, operating theater; PCAI, penetrating carotid artery injury.

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surgical access is challenging. Hemodynamically stable patients with extensive infarction and raised ICP on CTB are best managed with palliation.

AUTHOR CONTRIBUTIONS

Andre Steiner Madsen: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; visualization; writing – original draft; writing – review & editing. **Deirdre Kruger**: Conceptualization; data curation; formal analysis; methodology; supervision; writing – review & editing. **Damian Luiz Clarke**: Conceptualization; data curation; formal analysis; investigation; methodology; supervision; writing – original draft; writing – review & editing. **Pradeep Navsaria**: Conceptualization; data curation; formal analysis; methodology; supervision; writing – original draft; writing – review & editing. **Matthias Scriba**: Data curation; investigation; methodology; resources; software; writing – review & editing. **Wanda Bekker**: Data curation; investigation; methodology; resources; writing – review & editing. **Maeyane Steve Moeng**: Conceptualization; data curation; formal analysis; investigation; methodology; supervision; writing – original draft; writing – review & editing.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest. This research did not receive any grant from funding agencies in the public, commercial, or not-for-profit sectors.

ETHICS STATEMENT

Ethics approval was granted by the three institutions' ethics committees (University of the Witwatersrand: M211033, University of Cape Town: 754/2022, University of KwaZulu-Natal: BCA 221/13). Additionally, the study was registered with the National Health Research Database for the three provinces (Gauteng, the Western Cape and KwaZulu-Natal).

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