Original Article

Management of petrous temporal bone fractures: a 5-year experience from an Australian major trauma centre

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Background: Petrous temporal bone (PTB) fractures result from high energy head trauma and are associated with specific neurological and otological complications, as well as substantial intracranial injuries. Timely and appropriate management is required to ensure optimal outcomes. This study aims to describe the incidence and complications of PTB fractures in an Australian adult population and produce an evidence-based clinical practice guideline for their management.

Methods: Five-year retrospective case series of all patients with a PTB fracture from a single level-one tertiary major trauma centre in Melbourne, Australia. Data was collected on demographic and injury characteristics as well as specific PTB fracture-related complications including blunt cerebrovascular injury (BCVI), traumatic facial nerve palsy (FNP), cerebrospinal fluid (CSF) leak and hearing outcomes. Both the traditional anatomical and otic capsule violating (OCV) classification systems were assessed on their ability to predict these complications.

Results: There were a total of 377 patients with 419 PTB fractures (42 bilateral). The main complications were FNP (9.3%), CSF leak (12.6%), all cause hearing loss (76.5%), and BCVI (9%). Most patients were male (78.2%). The most common mechanism was falls (52.5%), of which the majority were low falls from standing height, which has not previously been reported. All FNP were managed conservatively, with 81.3% achieving a good outcome. There was no significant association with steroid therapy and facial nerve outcome (P=0.37). Twenty-two fractures (5.3%) were OCV and this was significantly associated with increased risk of facial nerve injury, profound sensorineural hearing loss (SNHL) and vertigo. There was no association with the traditional anatomical classification and complications.

Conclusions: PTB fractures should still be suspected in 'lower' mechanism injuries such as a fall from standing height, particularly in the elderly. They are associated with severe traumatic brain injury (TBI) and initial management strategies should focus on stabilisation of other injuries. Ideally only the otic capsule classification system be used in future clinical practice as it better predicts clinical outcomes. We introduce an evidence-based clinical practice guideline for the management of PTB fractures at our centre, which could be used at any similar-level trauma centres across the world.

Keywords: Petrous temporal bone (PTB); trauma, fracture; facial nerve injury; otic capsule

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Introduction

Fractures of the petrous temporal bone (PTB) usually results from high energy head trauma and hence are often associated with substantial intracranial injuries that remain the priority in the initial treatment period (1). As more patients are surviving from severe traumatic brain injury (TBI), it would be expected more patients will be living with the sequalae from their PTB fracture.

Complications after PTB fracture reflect the complex anatomy of this bone and the important structures encased within it, and can include traumatic facial nerve palsy (FNP), cerebrospinal fluid (CSF) leak and varying degrees of hearing loss. There has been ongoing debate regarding the classification of these fractures, however they are increasingly being classified according to their involvement of the otic capsule (bony labyrinth) as this has been shown to be more prognostic (2).

This study aims to describe the incidence of PTB fractures in an Australian adult population and their complications presenting to a level-one major trauma centre in Melbourne, Australia. In addition, we develop an evidence-based clinical practice guideline for their management. We present the following article in accordance with the STROBE reporting checklist (available at https://www.theajo.com/article/view/10.21037/ajo-22-7/rc).

Methods

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). Individual consent for this retrospective analysis was waived. After local ethical review board approval (QA2012012) we conducted a retrospective case series of all patients with a PTB fracture from a single level-one Tertiary Major Trauma Service in Melbourne, Victoria. Patients were identified using the Royal Melbourne Hospital Trauma Registry (RMH-TR) over the 5-year period between October 2015–October 2020. Patients were excluded if they were under 16 years old, had incomplete medical records or temporal bone fractures not involving the petrous portion.

Data was extracted from electronic medical records and RMH-TR for demographics, mechanism of injury, length of hospital stay, mortality, radiologic characteristics, Injury Severity Score (ISS), and associated injuries. Additional data was collected on PTB fracture-related complications including blunt cerebrovascular injury (BCVI), traumatic FNP and CSF leak was also collected. Subsequent hearing outcomes were assessed at first follow-up (average 12.6 weeks) using audiology data where available. Fractures were classified using the traditional 'longitudinal *vs.* transverse *vs.* mixed' system as well as the newer 'otic capsule violating (OCV) *vs.* otic capsule sparing (OCS)' classification. Facial nerve (CN7) outcomes were graded using the House-Brackmann equivalent (HB) scale with grade 1–3 classified as 'good' (signifying non-disfiguring weakness and complete eye closure), and grade 4–6 classified 'poor' (*Figure 1*). Patients with FNP had their images reviewed by a lateral skull base otologist (CI) to determine if there was a visible fracture line that appeared to transgress the pathway of CN7, and if so which section(s) were involved.

Statistical analysis

Data was recorded using REDcap database. Statistical analysis was performed using IBM SPSS v20 for both descriptive and comparative testing. Ordinal variables were compared using chi-squared or Fishers exact analysis. Multivariable logistic regression was performed to look for predictors of OCV fractures. Significance level for any given P value was set at 0.05.

Results

Patient demographics

Over the study period 22,479 patients were referred to the RMH trauma service. Of these, 377 patients had a total of 419 PTB fractures (42 bilateral injuries). Most patients were male (78.2%) with a median age of 42 years (IQR, 27–64 years). The most common mechanism of injury was a fall (52.5%) with 32.1% resulting from a fall from standing height (<1 m) (median age 63 years; IQR, 42–79) and 20.4% from a high fall (>1 m) (median age 56 years; IQR, 31–74). The next most common mechanism was road traffic crashes including pedestrian *vs.* car (12.2%), motor vehicle crashes (MVC) 10.3%, motorbike crashes (MBC) 8.5%, and cyclist injuries 4%. The remainder were blunt head injuries of various nature, including 9.3% assaults (*Table 1*).

Of the patients admitted with a PTB fracture, 79.8% were classified as major trauma (ISS >12). One third (32.1%) suffered a severe TBI. On initial assessment, 45.5% required intubation, precluding proper assessment of their CN7 function, with 48% admitted to the Intensive Care

Grade	Description	Characteristics	Outcome
I	Normal	Normal facial function	'Good'
II	Mild dysfunction	Slight weakness on close inspection; normal tone and symmetry at rest	
III	Moderate dysfunction	Obvious weakness +/– asymmetry, but not disfiguring; synkinesis, contracture or hemifacial spasm; complete eye closure with effort	
IV	Moderately severe dysfunction	Obvious weakness or disfiguring asymmetry; severe synkinesis, mass movement, spasm; incomplete eye closure	'Poor'
V	Severe dysfunction	Barely perceptible motion; asymmetry at rest	
VI	Total paralysis	No movement	

Figure 1 House-Brackman scale. The House-Brackmann grading system classification used to assess for 'good' *vs.* 'poor' facial nerve outcomes.

Table 1 Patient demographics descriptive data

Characteristic	Patients (N=377), n (%)
Age (years), median [IQR]	42 [27–64]
Sex	
Male	295 (78.2)
Female	82 (21.8)
Mechanism of injury	
All falls	198 (52.5)
Fall <1 m	121 (32.1)
Fall >1 m	77 (20.4)
Ped *vs.* car	46 (12.2)
MVC	39 (10.3)
Assault	35 (9.3)
MBC	32 (8.5)
Cyclist	15 (4.0)
Other	7 (1.9)
Sport	4 (1.1)
Penetrating	1 (0.3)
Hospital length of stay (days), median [IQR]	7 [3–16]
Intubated within first 24 h	171 (45.4)
Admitted ICU	181 (48.0)
Ventilation, median [IQR]	3 [1–8]
Major trauma (ISS >12)	301 (79.8)
Severe TBI (GCS <9)	121 (32.1)
Imaging	
CT carotid angiogram	245 (58.5)
CT-TB (reformats)	80 (19.1)
CT-TB (dedicated)	17 (4.1)
Death	64 (17.0)
Disposition	
Home	148 (47.3)
Rehabilitation centre	147 (47.0)
Other	18 (5.7)

IQR, interquartile range; MVC, motor vehicle crashes; MBC, motor bike crashes; ICU, Intensive Care Unit; ISS, Injury Severity Score; GCS, Glasgow Coma Score; TBI, traumatic brain injury; CT-TB, CT temporal bone.

Table 2 Injury characteristics descriptive data

Characteristic	n (%)
Total fractures	419
Side	
Right	201 (48.0)
Left	218 (52.0)
Bilateral	42 (11.1)
Other BOS fracture	243 (64.5)
ICH	320 (84.9)
Classification 1	
Longitudinal	299 (71.4)
Transverse	96 (22.9)
Mixed	24 (5.7)
Classification 2	
OCV	22 (5.3)
OCS	397 (94.7)
Involving carotid canal	128 (30.5)
Involving facial nerve canal	32 (7.6)
OCD	29 (6.9)

BOS, base of skill; ICH, intracerebral haemorrhage; OCV, otic capsule violating; OCS, otic capsule sparing; OCD, ossicular chain discontinuity.

Unit (ICU). Median time ventilated was 3 days (IQR, 1–8 days). The median length of stay (LOS) in hospital was 7 days (IQR, 3–16 days). Sixty-four patients (17%) died during their admission, all due to unsurvivable head injury. Of those who survived (n=313), 47.3% were discharged home, with the remainder transferred to rehabilitation centre (47%) or other hospital facilities (5.7%) (*Table 1*).

PTB injury characteristics

There were 243 patients (64.5%) with additional base of skull (BOS) fracture/s and 84.9% had associated intracerebral haemorrhage (ICH). There were 71.4% longitudinal fractures, 22.9% transverse and 5.7% mixed or comminuted fractures. Out of the 419 fractures, 5.3% were OCV with the remaining 94.7% being OCS. The carotid canal was involved in 30.5% of fractures and 7.6% involved the facial nerve canal. Ossicular chain discontinuity (OCD) was seen in 6.9% of fractures (*Table 2*).

Complications

BCVI

There were 22 cases (9%) of BCVI out of the total of 245 injuries (58.5%) that underwent CT carotid angiography. Most were Denver grade 1 injuries (54.5%) (3). Ten (22.7%) of these patients were managed with antiplatelet agents and 2 (9%) with anticoagulants. When these injuries were present, there was a mortality rate of 45%, with 22.7% developing neurological sequalae (*Table 3*). Involvement of the carotid canal was significantly increased in OCV fractures (82% *vs.* 28%; OR 10.4; P≤0.001), but not with the rate of BCVI.

Facial nerve injury

There were 39 cases of traumatic FNP (9.3%). Nine were clearly immediate onset and 30 delayed onset. Almost half of the patients in the delayed category were intubated pre-hospital or in the emergency department precluding assessment of facial nerve function in the first 24 h. There were 2 patients (5.4%) with bilateral injuries. Nine (23%) injuries resulted in eye complications. OCV fractures were associated with an increased risk of FNP (27% *vs.* 8%; OR 3.7; P=0.01). Nerve conduction studies (ENOG/EMG) were performed in 7 cases (18%). No patients underwent surgery on their facial nerve in our cohort. Twenty-six patients with FNP (66.7%) were treated with steroid therapy. Patients received either oral prednisolone or intravenous dexamethasone for an average duration of 7 days. The remaining 13 injures (33.3%) had no treatment.

Of the injuries with documented follow up (n=32), 26 injuries (81.3%) achieved a good HB outcome (HB 1–3), while the remaining 6 injures (18.8%) had a poor outcome (HB 4–6). A total of 12 injuries (37.5%) achieved complete return of normal function (HB 1). The median follow-up time from injury was 11.6 weeks (IQR, 7.6–21.2 weeks). We found no significant difference in outcome between patients who were treated with steroids compared to those who did not (P=0.37). When considering which part of the nerve was crossed by the fracture line, the tympanic segment was the most affected (68.8%) followed by the geniculate area (34.4%) (*Table 3*).

CSF leak

There were a total of 53 CSF leaks (12.6%) with the majority presenting with otorrhoea. The average duration of leak was 7 days with 2 patients reporting further

Table 3 PTB fracture specific complications

Characteristic	n (%)
CN7 injury	39 (9.3) (37 patients)
Bilateral FNP	2 (5.4)
Onset	
Immediate	9 (23.1)
Delayed	30 (76.9)
Intubated preventing assessment	20 (51.3)
CN7 section involved	
Meatal	3 (9.4)
Labyrinth	4 (12.5)
Geniculate	11 (34.4)
Tympanic	22 (68.8)
Mastoid	9 (28.1)
Nerve conduction tests (ENOG/EMG)	7 (17.9)
Management	
None	13 (33.3)
Steroids (any)	26 (66.7)
Mean duration steroid	7 days
Eye complications	9 (23.1)
Outcome (n=32)	
Good (HB 1–3)	26 (81.3)
Bad (HB 4–6)	6 (18.8)
Total CSF leak	53 (12.6)
Otorrhoea	44 (83.0)
Rhinorrhoea	15 (28.3)
Average duration of leak	7 days
Management	
Lumbar puncture	2 (3.8)
Lumbar drain	5 (9.4)
Average timing of drain	Day 7
Antibiotic cover for CSF leak	26 (49.1)
Meningitis	2 (3.8)
Hearing loss type (n=115)	
None	27 (23.5)
CHL	20 (17.4)
SNHL	50 (43.5)
Mixed HL	18 (15.7)
Extent hearing loss (n=115)	
None	27 (23.5)
Mild	33 (28.7)
Moderate	22 (19.1)
Mod-severe	6 (5.2)
Severe	9 (7.8)
Profound	18 (15.6)
Hearing management	
Referred for hearing aids	23 (7.3)
Referred for cochlear implant/other	3 (1.0)
Surgery	3 (1.0)
Carotid injuries	22 (9.0)
Denver grade	
Grade 1	12 (54.5)
Grade 2	4 (18.2)
Grade 3	3 (13.6)
Grade 4	3 (13.6)
Management	
Conservative	5 (22.7)
Antiplatelet	10 (45.5)
Anticoagulant	2 (9.0)
Endovascular	0
Outcome	
Neurological sequalae	5 (22.7)
Death	10 (45.5)

PTB, petrous temporal bone; CN7, facial nerve; FNP, facial nerve palsy; ENOG, electroneuronography; EMG, electromyography; HB, House-Brackmann; CSF, cerebrospinal fluid; CHL, conductive hearing loss; SNHL, sensorineural hearing loss.

intermittent rhinorrhoea after discharge. Five patients (9.4%) required a lumbar drain with resolution of the leak on average after 7 days. Antibiotics were administered for 26 patients (49%). Two patients developed meningitis (both of whom had received antibiotics).

Hearing loss

Of the 313 patients who survived their admission, audiology testing was conducted for one-third (n=115; 37%). Sensorineural hearing loss (SNHL) was seen in 50 injuries (43%), conductive hearing loss (CHL) in 17% and 16% with a mixed picture. Only 23% had no hearing loss. There was a statistically significant association between having an OCV fracture and SNHL pattern (88% *vs.* 40%; OR 10.1; P≤0.05) which was more likely to be profound in nature (88% *vs.* 10%; OR 66.9; P≤0.001) (*Table 4*). Having an ossicular chain injury was not significantly associated with a CHL. Of the 55 patients with at least moderate hearing loss, twenty-three (42%) were referred for hearing aids, three (5%) referred for cochlear implants and three (5%) for ossicular reconstructive surgery.

Other complications

A significant number of patients with OCV fractures suffered vertigo (12%) in the weeks after their injury (P<0.05) and although similar number suffering from tinnitus this was not significant between the fracture groups. There were infrequently reported other complications including loss of taste (3.5%), tympanic membrane (TM) perforation (1.9%) and ear canal stenosis (0.6%) (*Table 3*).

Discussion

This study of PTB fractures from an adult major trauma service is the first to describe these injuries in an Australian adult population and is one of the largest series to date. Previous studies have shown that PTB fractures require high energy transfer (up to 8,000 newtons of force from cadaveric studies) and have historically been associated with MVC's (1). However, in this series, we found falls to be the most common mechanism (52.5%) which appears to be an emerging trend in the literature (4,5). Interestingly, we found that 20.4% occurred after low-falls (from standing height) which has not previously been reported. The reason for this is unclear but could in part be attributed to the ageing population as the median age for these patients was 20 years greater than the median age for the entire population (42 *vs.* 63 years).

PTB fractures have traditionally been classified anatomically based on the orientation of the fracture line with respect to the long axis of the petrous bone (longitudinal, transverse or mixed). Longitudinal fractures are more common (50–80%) (6), with similar rate seen in this study of 71.4%. Although intuitive, this classification has previously been scrutinised for its relevance as it has shown no correlation with clinical outcomes and does not provide useful prognostic information (2,7). We found that transverse fractures were more likely to be OCV (P≤0.001), with longitudinal fractures more likely to be OCS (P≤0.001), but we found no significant association with the traditional anatomical classification and complications (*Table 4*). This study adds to the growing body of evidence that would suggest this classification system be abandoned in future research and clinical practice.

Classification of PTB fractures defined by involvement of the otic capsule, has previously shown to give more clinically relevant prognostic information. OCV fractures invariably result in some degree of SNHL and are associated with increased risk of other complications, including traumatic FNP, CSF leak and intracranial injury (2,8-10). While OCS fractures are more likely to result in CHL (2,7,8).

In our study we found 5.3% were OCV which is in keeping with the literature (2–7%) (6). We found OCV fractures were more likely to result in increased rates of CN7 injury (P≤0.001), SNHL (P≤0.05) which is more likely to be profound (P≤0.001) and vertigo (P≤0.05). We also found OVC fractures were more likely to be associated with other BOS fractures (frontal & occipital), severe TBI, and death. However, these are likely independent and attributable to the significant forces required to cause the injury. Our findings support the continued move to classify PTB fractures based on the involvement of the otic capsule.

Facial nerve injury

Traumatic FNP was seen in 9.3% of our cohort, with a range of around 7–15% reported in the literature (9-12). OCV fractures are associated with up to 5 times higher risk of FNP compared to OCS fractures (2). This was similarly seen in our population (27% *vs.* 8%; OR 3.2; 95% CI: 9.5–69.1; P≤0.001). It is typically classified into immediate onset FNP happening at the time of injury and being obvious on initial presentation *vs.* delayed onset with paralysis occurring over subsequent days. It can be difficult to make

Table 4 Multivariable logistic regression analysis of complications in OCV versus OCS fractures

Variable	Univariable (unadjusted)				Multivariable logistic regression analysis adjusted for age & sex			
	Total (n=419)	OCV (n=22)	OCS (n=397)	P value	OR	95% CI lower	95% CI higher	P value
Male	332 [79]	21 [96]	311 [78]	0.088	1.76	0.77	43.79	0.088
Age (years) (median)	40	39	40	0.202				
Severe TBI	159 [38]	16 [73]	143 [36]	0.002	4.40	1.67	11.61	0.003
Death	76 [18]	9 [41]	67 [17]	0.007	4.62	1.80	11.84	0.001
ICH	356 [85]	22 [100]	334 [84]	0.997				
BOS fracture (any)	285 [68]	20 [91]	265 [67]	0.032	4.41	1.01	19.27	0.049
Frontal	55 [13]	7 [32]	48 [12]	0.011	3.16	1.21	8.25	0.019
Sphenoid	172 [41]	10 [45]	162 [41]	0.667				
Occipital	145 [35]	14 [64]	131 [33]	0.005	3.50	1.42	8.60	0.006
Contralateral PTB	103 [25]	6 [27]	97 [24]	0.764				
Fracture class 1								
Longitudinal	299 [71]	5 [23]	294 [74]	<0.001	0.11	0.04	0.31	<0.001
Transverse	96 [23]	12 [55]	84 [21]	<0.001	4.20	1.74	10.13	0.001
Mixed	24 [6]	5 [23]	19 [5]	0.002	5.23	1.71	15.94	0.004
BCVI	22 [11]	2 [9]	20 [5]	0.452				
Traumatic FNP	39 [9]	6 [27]	33 [8]	0.006	3.77	1.35	10.51	0.011
CSF leak	53 [13]	5 [23]	48 [12]	0.153				
OCD	29 [7]	5 [23]	24 [6]	0.043	3.22	0.10	10.41	0.051
Involving CN7 canal	32 [8]	12 [55]	19 [5]	<0.001	21.91	8.25	58.22	<0.001
Involving carotid canal	128 [31]	18 [82]	110 [28]	<0.001	10.43	3.43	31.74	<0.001
Hearing loss type (n=115)	115	8	107					
None	27 [23]	1 [13]	26 [24]	0.459				
CHL	20 [17]	0 [0]	20 [19]	0.998				
SNHL	50 [43]	7 [88]	43 [40]	0.031	10.10	1.17	86.92	0.035
Mixed HL	18 [16]	0 [0]	18 [17]	0.998				
Extent Hearing loss (n=115)	115	8	107					
None	27 [23]	1 [13]	26 [24]	0.459				
Mild	33 [29]	0 [0]	33 [31]	0.998				
Moderate	22 [19]	0 [0]	22 [21]	0.998				
Mod-severe	6 [5]	0 [0]	6 [6]	0.999				
Severe	9 [8]	0 [0]	9 [8]	0.999				
Profound	18 [16]	7 [88]	11 [10]	<0.001	66.86	6.75	662.49	<0.001
Assoc symptoms (n=313)	313	12	301					
Tinnitus	38 [12]	2 [17]	36 [12]	0.626				
Vertigo	38 [12]	5 [42]	33 [11]	<0.001	5.61	1.65	19.01	0.006

The data are expressed as n [%]. OCV, otic capsule violating; OCS, otic capsule sparing; TBI, traumatic brain injury; ICH, intracerebral haemorrhage; BOS, base of skull; BCVI, blunt cerebrovascular injury; FNP, facial nerve palsy; CSF, cerebrospinal fluid; OCD, ossicular chain discontinuity; CN7, facial nerve; CHL, conductive hearing loss; SNHL, sensorineural hearing loss; OR, odds ratio; CI, confidence interval.

this important distinction when patients are intubated and these patients should be considered and managed as an immediate injury (1,13). FNP can be complete or incomplete, with the latter suggesting a neuropraxia and generally having an excellent rate of complete recovery with conservative treatment (9,14,15).

The onset of paralysis is important because as it can indicate the severity of the nerve damage. Immediate onset FNP is due to compression of the nerve from bony fragments or in worst case scenario complete nerve transection. Delayed onset palsy is thought to be due to contusion or perineural haematoma causing neural oedema and compression against the bony canal (6). Previous studies have also found generally good recovery for delayed injuries with conservative management (9,15-18). This usually consists of a 1–2-week course of high dose steroid therapy, if not contraindicated (1). This evidence comes mostly from the management of idiopathic FNP and there is limited evidence for its use in traumatic facial nerve injury, but it is still standard practice (19). All patient with FNP were managed non-surgically with or without steroids, and despite small sample size we found no statistically significant difference in those achieving a good outcome when steroids were administered (P=0.37). Steroids must be used with caution in the severely head injured patient after the result of the CRASH study and Cochrane review finding increased mortality in this cohort (20,21).

We found in total 6 injuries (18.8%) had a poor outcome (HB 4–5) in our study. Only two of these cases had nerve conduction performed and adequate follow-up to draw conclusions. It is unclear whether these cases were offered surgery, but their outcomes suggest a severe facial nerve injury that may have benefited from exploration. Surgical intervention for patients with facial nerve injury remains controversial, as does the timing of surgery. Some authors recommend early decompression within 2–4 weeks after injury (13,22). However, recent studies have suggested there is still benefit from delayed surgery 1–3 months after injury (5,15,23,24). Several authors agree there are considerably worse outcomes after 3 months (15,22).

Patients with immediate or complete FNP are generally considered to have a worse nerve injury consistent with axonotmesis or neurotmesis and it is recommended these patients are assessed with nerve testing using either electroneuronography (ENoG) or electromyography (EMG). The primary benefit of EnoG is for prognostication and it has been generally accepted that EnoG showing >90% degeneration of the affected side in the first 6 days or progressing to >95% within 14 days is associated with a poor recovery and should be an indication for surgery (9,23,25). Other studies have shown that ENOG <90% is associated with a good outcome and can be managed conservatively (14). The decision to perform nerve conduction studies will vary widely between hospitals and countries, depending on availability of testing and surgeon preference. Patients may also be referred late to specialist centres, having missed the relatively short window where these tests are most useful. However, with the global trend for trauma patients to be managed in specialist tertiary centres with multi-disciplinary team input, these tests should be more accessible for early assessment.

CSF leak

It has been reported that up to 78% of traumatic CSF leaks will resolve spontaneously within 7 days with conservative management (9), which involves a period of bed rest and measures to prevent fluctuations in intracranial pressure and blood pressure (such as avoiding nose blowing, straining etc.) (26). This is usually appropriate until day 7–10 at which point further intervention may be required with lumbar puncture (LP) or lumbar drain (1). One study reported a significant reduction in the duration of CSF leak of 3.4 days for patients treated with lumbar drain compared to conservative management alone (27). The average duration of leak in our study was 7 days with 2 patients undergoing LP and 5 patients requiring a lumbar drain. None required specific surgical intervention during the study period emphasising the majority will heal.

One of the well documented risks after CSF leak is the possibility of meningitis. Meningitis is rare in patients without CSF leak and antibiotics are not indicated as prophylaxis for BOS fracture alone (28). In our study 49.1% of patients with a CSF leak were on antibiotics, but only 2 patients (3.8%) with a leak developed meningitis, both having received antibiotic therapy. Previous meta-analyses found conflicting results for the risks of meningitis after traumatic CSF leak (29,30). To address these deficiencies, two Cochrane database meta-analysis performed in 2011 and 2015 which included five randomized controlled trials have since concluded that the evidence does not support the use of prophylactic antibiotics to reduce the risk of meningitis in these patients (28,31). This is increasingly important in the modern era of global antibiotic resistance, and the data from our study tentatively supports this conclusion although patients may require antibiotics for

other injuries. Previous studies have shown the rate of meningitis is increased in prolonged CSF leaks over 7 days so patients should receive active management if persistent beyond this point (9).

Hearing loss

Hearing loss is reported to affect between 24–81% of patients after PTB fracture (8,9). Most patients will complain of some degree of hearing loss which may be transient or permanent. Injury can result in a SNHL, CHL or mixed picture. SNHL implies there has been damage to the delicate structures of the inner ear whereas CHL implies an issue lateral to the cochlea, such as haemotympanum, TM perforation or damage to the ossicular chain (6).

CHL second to haemotympanum is expected to resolve in the weeks after injury and TM perforations should be monitored for resolution. A 30-dB air-bone gap that persists after 6–8 weeks would be suspicious for an ossicular injury and should be considered for middle ear exploration (1). Conservative management of CHL often leads to spontaneous recovery and intervention is rarely needed before 6 months (32). Damage to ossicles is seen more often in longitudinal fractures, with directional classification schemes less predictive of SNHL (6).

There is a stronger association between OCV fractures and profound SNHL from the literature, with OCV fractures up to 25 times more likely to cause SNHL (2,8,9). This is supported by our study whereby OCV fractures were 2 times more likely to result in SNHL (88% *vs.* 40%; P≤0.05) and 4 times more likely to be profound in nature (88% *vs.* 10%; P<0.001). We found no statistically significant association between hearing outcomes and directional classification in our population.

SNHL can be immediately apparent but is usually difficult to assess unless the patient is neurologically stable. If possible, early detection is beneficial and initiation of steroid therapy may help preserve hearing function as shown by Padmakumar *et al.* where early steroids caused a mean improvement in hearing of 12.7 dB (33). However, hearing loss is often not assessable soon after injury and the same cautions with steroids in severe TBI mentioned previously apply.

Other symptoms

There are a number of other sequalae from PTB fracture that were seen in our study that have also been well documented in other series (9,10). Of the cases surviving for follow up (n=313), there were 38 with vertigo (12%), 38 with tinnitus (12%), 11 with anosmia (3.5%) and 13 patients with loss of taste (4.2%). Whilst not the focus of this study, these are symptoms that can negatively impact a patient's quality of life and should be monitored for in the follow up period. Fracture of the tympanic segment and damage to the bony ear canal can lead to canal stenosis, which occurred in 2 patients in this series. Early use of ear wicks or packing can splint the canal to help prevent this complication, as it can be difficult to treat once established and can predispose to later cholesteatoma (1).

Imaging

The recommended imaging for accurate detection of PTB fracture is multidetector CT temporal bone (CT-TB) with multiplanar reconstructions at a section thickness of 1mm or less. It is generally recommended that patients with complex radiological fractures or clinical complications from their PTB fracture should have dedicated fine slice imaging to assess their need for intervention (34). Not all PTB fractures may be seen on initial imaging studies and in trauma patients with occult signs of fracture, these can again be further assessed with dedicated CT-TB imaging. However, several studies have suggested that dedicated imaging may not be required for all patients. The combined use of head, maxillofacial and cervical spine CT, which is a common imaging series in head trauma, has been shown to be sufficient to diagnose 99.0% of PTB fractures compared to dedicated CT-TB (4). The negative predictive value (NPV) of maxillofacial CT alone *vs.* CT-TB to diagnose carotid canal involvement was reported as >95% (35).

Temporal bone reformats were requested in 80 patients (19.1%) in this study, with only 17 patients (4.1%) having a dedicated fine slice CT temporal bone. A recent analysis by Szczupak *et al.* showed that in low-risk patients (without involvement of major temporal bone structures or clinically apparent complications) the standard head trauma CT protocol (as performed at our centre) had a NPV of 100% for OCV or CN7 injury compared to dedicated CT-TB, and NPV of 96% and 99% for carotid canal involvement and OCD respectively (36). They suggested that in this patient group standard head trauma CT protocols are sufficient to diagnose PTB fractures and

exclude major complications. This also limits exposure to unnecessary ionising radiation, reduces radiology burden and in-hospital transport of critically ill patients and provides a significant cost saving to the health service (36). We would advocate that reformatted PTB images from the initial head trauma CT series are sufficient for most patents unless they have clinical complications from their temporal bone injury that should then be further assessed with dedicated CT-TB or if complications become apparent on follow-up.

This low rate of dedicated imaging is due to all trauma patients at our centre receiving 1mm slices through the BOS during initial trauma imaging, and in part related to logistic difficulties of transporting patients from ICU and radiology access in a busy tertiary trauma centre. With low patient numbers we were unable to make comparisons between the imaging modalities.

Limitations

This study had some limitations primarily in its retrospective design and accessing information from medical records that at times can be incomplete. Patient selection from the trauma registry at a major tertiary referral centre, which is the second busiest in Australia, means almost all patients had other critical injuries that may factor to overestimate mortality rates. Only patients meeting the trauma registry criteria were recorded in the registry with a complete dataset allowing them to be included in this study, again possibly overestimating the incidence of PTB fracture amongst the general head injury population. Conversely, due to the relatively high in-patient mortality rate and incomplete audiology follow up, not all patients could be properly assessed for complications, so these may be underestimated in our study. Modern understanding of TBI suggests an evolving model of injury including duration of post traumatic amnesia to diagnose severity. We used the original definition of severe TBI (GCS <9) and this may under- or over-estimate this group.

Clinical practice guideline

Using the findings published in this study and published international literature we developed an evidence-based clinical practice guideline for the management of temporal bone fractures at our centre that we think is suitable for other centres that may not have their own guideline (*Figure 2*).

Conclusions

PTB fractures usually occur after significant head trauma, however, should still be suspected in 'lower' mechanism injuries such as a fall from standing height, particularly in the elderly. They often occur in poly-trauma patients and are associated with severe TBI and the initial management strategies should focus on stabilisation of these other injuries. Early ENT input should be sought in cases of FNP and CSF leak to facilitate specialised investigation and intervention. Most other injuries including hearing loss can be managed on an outpatient basis. Standard head trauma CT imaging with reformatted PTB images should be adequate in most situations unless there is an OCV fracture or PTB specific complications, in which case dedicated CT-TB imaging should be sought. We advocate that only the otic capsule classification system be used in future clinical practice and research as it better predicts clinical outcomes. We introduce an evidence-based clinical practice guideline for their management at our centre, which could be used and any similar-level trauma centres around the world.

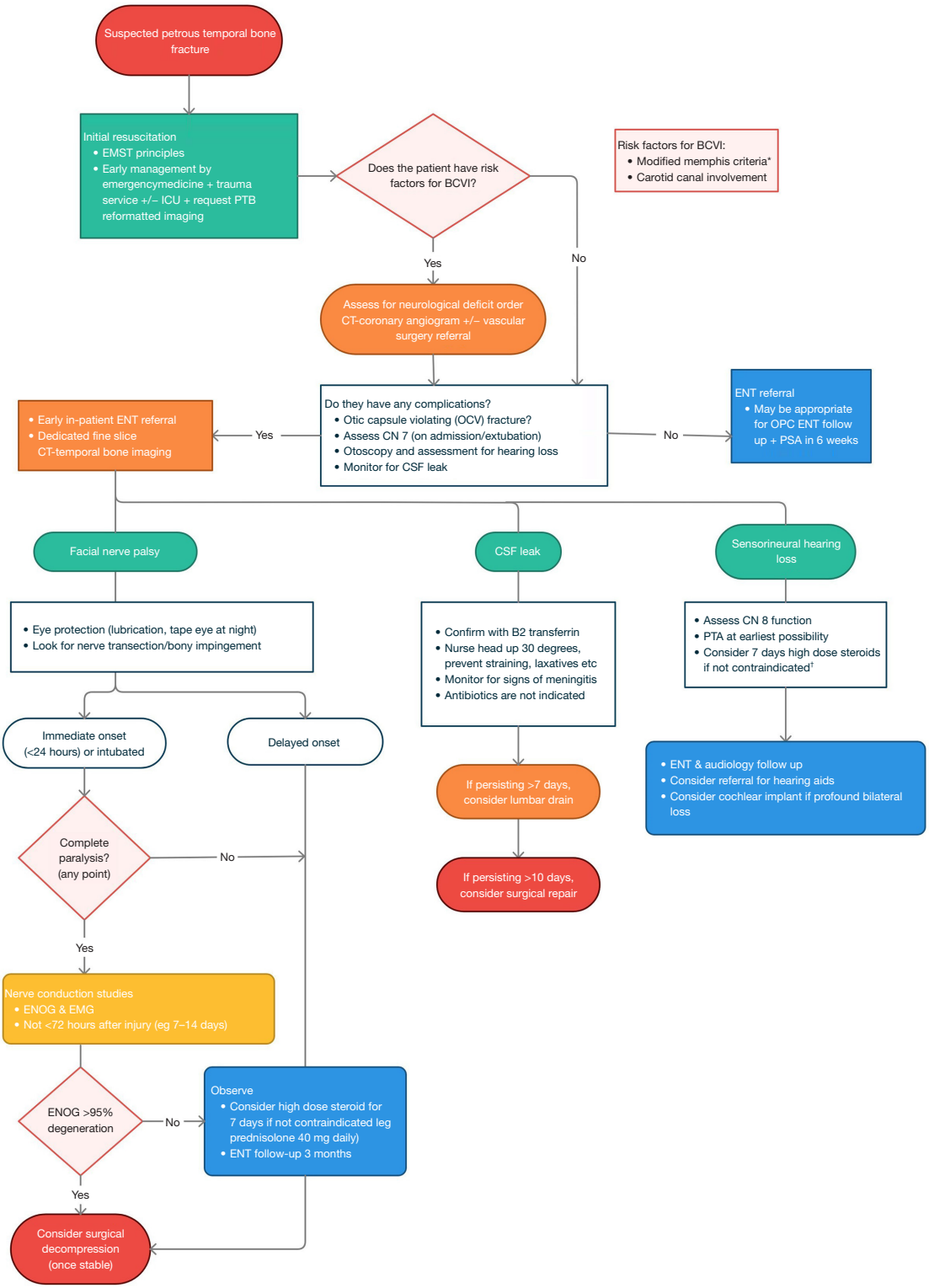

Figure 2 PTB fracture clinical practice guideline (separate file). *, please see full modified Memphis criteria; †, steroids are contraindicated in TBI. EMST, Early Management of Severe Trauma; BCVI, blunt cerebrovascular injury; ENT, Ear, Nose & Throat; CN7, facial nerve; CSF, cerebrospinal fluid; OPC, Out-Patient Clinic; CN8, vestibulocochlear nerve; PTA, pure tone audiogram; ENOG, electroneuronography; EMG, electromyography; PTB, petrous temporal bone.

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Footnote

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by local institutional ethics committee (QA2012012) and individual consent for this retrospective analysis was waived.

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