SUSPECTED SNAKEBITE: CLINICAL PATHWAY

SUSPECTED AND CONFIRMED SNAKE BITE: all cases should be observed with serial blood testing for 12 hours to exclude severe envenoming using the following pathway.

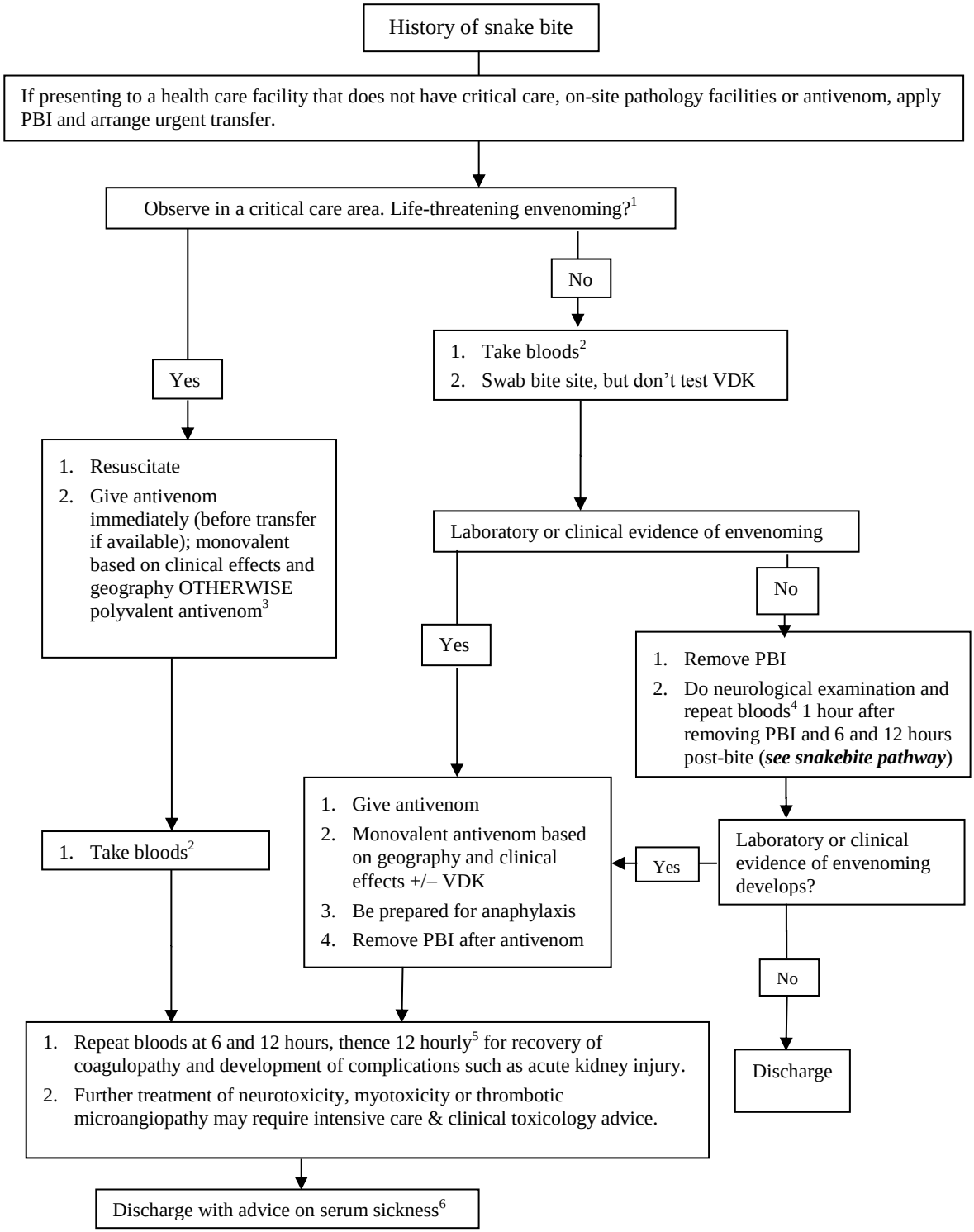
Date_____ MRN: _____

Initial for **YES**

INTERVENTION /OUTCOME	**INITIAL**
Patient presented at _____hrs. Pressure bandage with immobilisation (PBI) in situ.	
Pathology taken on admission for: Coagulation tests (INR[1], aPTT, quantitative D-Dimer), FBC,UEC,CK,VDK[2]	
Pathology results reviewed within one hour and are within normal limits. The patient has no signs of neurotoxicity (ptosis, bulbar, respiratory or distal paralysis)[3] **IF** pathology results are abnormal, OR neurotoxicity develops, ***exit pathway, admit patient and treat; see guidelines***[4]	
Remove pressure bandage and immobilisation; observe for any clinical evidence of envenoming.	
Repeat bloods 1 hour post- bandage removal : INR, aPTT and CK	
Pathology results are within normal limits. The patient has no signs of neurotoxicity (ptosis, bulbar, respiratory or distal paralysis)** **IF** pathology results are abnormal OR neurotoxicity develops, ***exit pathway, admit patient and treat; see guidelines***	
Repeat bloods 6 hours post-bite (unless already >6h): INR, aPTT and CK	
Pathology results are within normal limits. The patient has no signs of neurotoxicity. **IF** pathology results are abnormal OR neurotoxicity develops***, exit pathway, admit patient and treat; see guidelines***	
Final Bloods at 12 hours post-bite[5]: INR, aPTT and CK	
Pathology results are within normal limits. The patient has no signs of neurotoxicity. Patient can be discharged. **IF** pathology results are abnormal OR neurotoxicity develops***, exit pathway, admit patient and treat; see guidelines***	

[1] Only laboratory based INR should be done, point of care testing is unreliable and gives false negatives.
[2] A bite swab may be collected and stored; only test if there are any signs of envenoming
[3] Neurotoxicity can be subtle and it is important to include both looking for ptosis and assessing for fatigue (eyelid droop from failure to maintain an upward gaze)
[4] Consult treatment guidelines (e.g. Therapeutic Guidelines) or ***call Poisons Information Centre (131126)***
[5] For the unusual circumstances where the PBI remains on for > 6h, a final set of bloods and neurological examination should be done 6 hours after PBI removal.

Summary of snake bite management



[1] Cardiac arrest, respiratory failure secondary to paralysis, major haemorrhage (intracranial, major gastrointestinal or other life-threatening bleeding).
[2] Blood tests–coagulation screen (INR, aPTT, D-dimer, fibrinogen); FBC & blood film; EUC, CK, LDH
[3] In some regions brown + tiger snake monovalent is sufficient to cover all snakes.
[4] Serial blood tests in non-envenomed patients: INR (or PT), aPTT, CK.
[5] Serial blood tests in envenomed patients: INR (or PT), aPTT, CK, FBC, EUC.

[6] Any patient receiving antivenom should receive advice on discharge about possibility of serum sickness occurring 4 to 14 days later.

Clinical syndromes from Australian snake envenoming

Clinical Syndrome	Characteristics
Venom-induced consumption coagulopathy (VICC)	Activation of the clotting pathway by prothrombin activator toxins and consumption of fibrinogen, factor V and factor VIII leading to a consumptive coagulopathy. • INR and aPTT are prolonged or unrecordable • Fibrinogen is low or undetectable • D-Dimer is very high (at least 10x and up to 1000x normal) *Complete or severe VICC* is defined as: • Undetectable fibrinogen, unrecordable INR and aPTT, and very high d-Dimer (10-1000 times the assay cut-off). *Partial VICC* (less severe changes) is defined as: • Low but detectable fibrinogen, and an abnormal INR <3.0
Neurotoxicity	Descending flaccid paralysis which first involves the eye muscles (ptosis; diplopia and blurred vision due to external ophthalmoplegia), followed by bulbar muscles, respiratory muscle paralysis and limb paralysis.
Myotoxicity	Local or generalised myalgia and/or muscle tenderness: • The CK is usually normal on admission and then rapidly increases over 24 to 48 hours. The CK ranges from >1000 U/L in mild cases to >100,000 U/L in severe cases and may take days to resolve. • Renal impairment and an elevated potassium may develop in severe cases
Sudden collapse	Collapse or syncope that occurs within 1 hour of the bite: • Collapse is associated with hypotension and loss of consciousness. • Spontaneous recovery usually occurs within 5 to 15 minutes • A minority of patients will have a cardiac arrest or seizure.
Systemic symptoms	Non-specific systemic symptoms include nausea, vomiting, headache, abdominal pain and diarrhoea.
Anticoagulant coagulopathy	The aPTT is elevated although some aPTT assays may be insensitive to this coagulopathy. There may be a mild elevation of the INR, but normal fibrinogen. This coagulopathy is not clinically important but does provide a good marker of envenoming by black snakes, including mulga snakes.
Thrombotic microangiopathy (TMA)	Presence of fragmented red blood cells on blood film (microangipathic haemolytic anaemia), thrombocytopenia and an abnormal creatinine, which may lead to acute kidney injury requiring dialysis.

INR – international normalized ratio; aPTT – activated partial thromboplastin time; CK – creatine kinase.

Summary of the features of clinically important venomous Australian snakes.

Snake	Coagulopathy	Neurotoxicity	Myotoxicity	Systemic symptoms	Thrombotic microangiopathy	Cardiovascular effects	Antivenom
Brown Snake	VICC[1]	Rare and mild	-	<50%	10%	Collapse (33%); Cardiac arrest (5%)	Brown snake
Tiger Snake Group							
Tiger snake	VICC[1]	Uncommon	Uncommon	Common	5%	Rare	Tiger snake
Rough-scale snake	VICC[1]	Uncommon	Uncommon	Common	<5%	Rare	Tiger snake
***Hoplocephalus* sp.**[2]	VICC[1]	-	-	< 50%	<5%	Rare	Tiger or Brown snake
Death Adder	-	Common	-	Common	-	-	Death Adder[3]
Taipan	VICC[1]	Common	Rare	Common	5%	Uncommon	Taipan[3]
Black snakes							
Mulga snake	Anti-coagulant	-	Common	Common	-	-	Black snake[3]
Red-bellied black snake	Anti-coagulant	-	Common	Common	-	-	Tiger snake
Sea Snakes	-	Uncommon	Common	Common	-	-	Sea snake[4]

[1] Partial venom induced consumption coagulopathy (VICC) occurs in 20 to 30% of cases for all snakes that cause VICC, except taipan envenoming where partial VICC occurs in almost 50% of cases [2] The *Hoplocephalus* genus/group includes Stephen's banded snake (*H. stephensi*), the broad headed snake (*H. bungaroides*) and the pale-headed snake (*H. bitorquatus*); [3] Polyvalent antivenom can be substituted for these large volume monovalent antivenom with no increase in risk or cost.; [4] Polyvalent or tiger snake antivenom cannot be used for sea snake envenoming.

Absolute and relative indications for antivenom.

Absolute indications: • History of sudden collapse, seizure or cardiac arrest • Abnormal INR • Any evidence of paralysis with ptosis and/or ophthalmoplegia being the earliest signs
Relative Indications: • Systemic symptoms (vomiting, headache, abdominal pain, diarrhoea) • Leukocytosis (may be associated with lymphopenia) • Abnormal aPTT[1] • CK > 1000U/L[2]

INR – international normalized ratio (Normal range: 0.9 to 1.3); aPTT – activated partial thromboplastin time; CK – creatine kinase; [1] Although an abnormal aPTT may not be clinically significant it is often an early marker of envenoming; [2] The CK may be abnormal on admission, usually between 250 and 1000U/L which is unlikely to be associated with myotoxicity. An increasing CK or abnormal CK rises more than 6 hours post-bite are more likely to be due to myotoxicity

Management of systemic hypersensitivity reactions to antivenom.

1.	**Stop antivenom infusion** *Many reactions resolve with stopping antivenom. It can then be restarted at a slower rate*
2.	**Lie patient flat, start high flow O_2, support airway/ventilation if required**
3.	**For hypotension, give rapid infusion of 1L normal saline (20ml/kg in children)** *Severe antivenom reactions with hypotension will have reduced venous return, so a supine posture and fluid resuscitation are essential.*
4.	**For hypotension, hypoxaemia, wheeze or upper airway obstruction, give adrenaline <u>intramuscularly</u>, 0.01 mg/kg to a maximum of 0.5 mg.** *Alternatively, those experienced with IV infusions of adrenaline may go straight to step 5*
5.	**Consider cautious IV infusion of adrenaline – avoid BP surges** – *Envenomed patients may be severely coagulopathic and high BP may cause or worsen intracerebral haemorrhage. Some patients can have exaggerated hypertensive responses to IM adrenaline, especially to second doses.* – *If no response to steps 1-4, consider starting a cautious and closely monitored IV infusion of adrenaline, which can be reduced as soon as BP starts to recover, preventing BP surges.* – *Use 1mg in 100mL by infusion pump: Start at 0.5 mL/kg/hour and titrate according to response; monitor BP every 3-5 minutes (using the arm opposite to the infusion); beware that as the reaction resolves adrenaline requirements will fall, the BP will rise and the infusion rate will need to be rapidly reduced.*
6.	**For persistent hypotension, repeat normal saline bolus**
7.	**For bronchospasm, consider nebulized salbutamol**
8.	**For upper airway obstruction, consider nebulized adrenaline**
9.	**Seek further advice from a clinical toxicologist or Poison Centre**

A modification of standard anaphylaxis guidelines is recommended due to the risk of coagulopathy.