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Tracheal perforation from non-fatal manual strangulation

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ABSTRACT

Non-fatal strangulation is a very common but often underestimated cause of severe injury. In this case, a woman experienced several episodes of manual strangulation in an episode of domestic violence which involved loss of consciousness, confusion and neck pain, particularly over the trachea. CT angiogram of the neck showed a small perforation of the trachea at the level of the thoracic inlet.

The prevalence of non-fatal strangulation in the Australian general population is unknown, but a study in Western Australia measured a prevalence of 7.4% of women presenting to a sexual assault service. A systematic review analysing data from 9 countries in North America and Europe, estimated a lifetime prevalence by an intimate partner of between 3.0% and 9.7% of all women (1). US data suggests that of those experiencing intimate partner violence, prevalence ranges from 27 to 68% (2, 3).

This article considers the limited science known about the injuries sustained from non-fatal manual strangulation, much of which is extrapolated from case reports in the literature.

In this case report, we describe a small tracheal perforation caused by tracheal occlusion during strangulation, on a conscious woman in the context of domestic violence.

Ms AB, a 21 year old woman from remote North Queensland, presented to the Emergency Department after an alleged incident of domestic violence involving 3 episodes of strangulation. The police reported the alleged incident occurred over 20 minutes.

The initial history and exam taken was obtained approximately 3 hours post the alleged incident. She reported being strangled multiple times in different locations including being “pinned against a wall and strangled”, making her feel short of breath and like she was being “choked to death”. She said that she had pain “everywhere”, worse over her anterior neck and under her jaw, and was nauseated. She was unable articulate a clear line of events, had pain on swallowing, a hoarse voice, redness to the base of the throat extending to the front of the shoulders, and tenderness directly over the trachea. Repeated physical assessment at 6 hours post incident found no external signs over her head or neck.

A non-contrast CT was done of the neck and brain (Fig. 1). Results showed a small (19 × 9 × 13mm) gas pocket at the level of the thoracic inlet, on the right-side of the trachea (shown in Fig. 1). The trachea at that level was misshapen and there was a bulge that was likely the tract to the air pocket. This is a small tracheal perforation. No other sign of injury was seen on the CT. Unfortunately, the woman was lost to follow-up.

Non-fatal strangulation is a very common but often underestimated cause of severe injury. Also known as choking, throttling, head-lock and suffocation, much of the evidence surrounding this injury is based on case reports, and small case series with extrapolations from fatal strangulations and hanging injuries.

The prevalence of non-fatal strangulation in the Australian general population is not known. However, a systematic review analysing data from 9 countries in North America and Europe, estimated a lifetime prevalence by an intimate partner of between 3.0% and 9.7% of all women.[1] US data suggests that of those experiencing intimate partner violence, prevalence ranges from 27 to 68%.[2,3] A study of women presenting to a sexual assault service in Western Australia measured a prevalence of non-fatal strangulation of 7.4% which rose to 33.9% in those 30–39 years old who had been assaulted by an intimate partner.[4]

It is generally accepted that strangulation is a form of asphyxia characterised by closure of blood vessels and/or the air passage due to external pressure on the neck. There are 4 main pathways to death:

1. Occlusion of the blood flow to the brain, through occlusion of the carotid arteries. Should the pressure be over the base of the neck, the vertebral arteries may also be occluded. This mechanism is known as anoxic, but all energy substrates including oxygen are denied to the brain.
2. Occlusion of the veins draining the brain resulting in congestion of the blood flow to the brain with rupture of the small vessels resulting in petechial haemorrhages.
3. Occlusion of the airway resulting in hypoxia.
4. Stimulation of the carotid sinus.[6]

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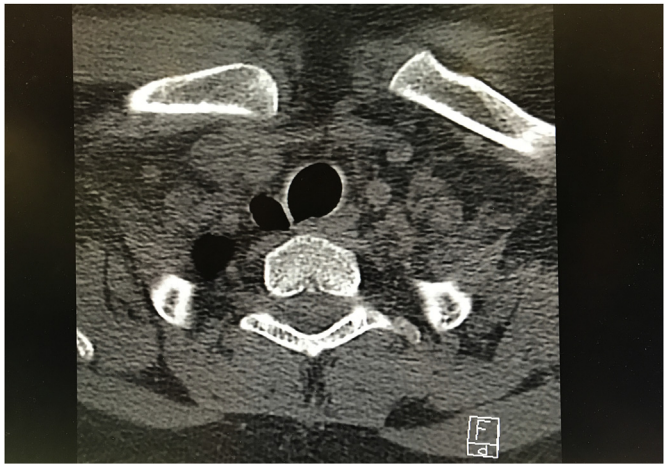

Fig. 1. An axial cross section at the level of the thoracic inlet by non-contrast CT. An arrow points to the gas pocket.

The pressures required to achieve this are variable, and depend on the amount of area compressed, the duration and location of the compression, and how superficial the structures are.[6] However, 2kg for venous compression and 3.5kg have been quoted.[7]

In the anoxic progression, no memories are formed from the point of arterial occlusion as the neurons are deprived of all energy substrates, including oxygen. Time to loss of consciousness varies from 6 to 13 seconds, followed by a short-lived anoxic seizure of 6–8 seconds. Loss of bladder control has been reported after 15 seconds, and loss of bowel control after 30 seconds.[8] Decerebrate rigidity occurs in approximately 20 seconds, with decorticate rigidity in 40 seconds.[9] Brain death is variable from 1 to 6 minutes.[9,10]

In comparison, the hypoxic progression depends on such things as the amount of oxygen in the lungs and blood stream, as well as the physical condition of the body. Memories may continue to be formed while the person is conscious.[11] During the ensuing period of 'panic' or 'struggle',[11] there may be large changes in intrathoracic and airway pressure as the person attempts to breathe against the obstruction.[12–14] Pulmonary oedema may occur.[12] Eventually, the brain has insufficient oxygen to maintain its function and the subsequent events are similar to anoxia.

Injuries to the airway and bronchial tree are well described in relation to intubation, tracheostomy and blunt force trauma.[15] Traditionally, tracheal injuries have been associated with a very high mortality rate due to their association with high mortality events (one series of injuries found MVA 59% and crush injuries 25%[16]). The mortality also depends on the location of the injury, with tracheal injuries (26%) having a higher mortality than bronchial injuries (16% right, 8% left).[15,17] In keeping with the emphasis on severe injury, a 2001 study showed that surgical repair compared to conservative management reduced mortality 10 fold (6% compared to 66%).[18] However, it is unclear how applicable this is to smaller or more isolated injuries from strangulation. The complication rate in the short to long term from tracheal injuries is also unclear. A 2015 paediatric small case series of tracheal injury had a stenosis rate of 62%, over half of which 37.5% required surgical intervention.[19] Given however, that flow is proportional to the radius to the power of 4, a small difference on an already small trachea would lead to a higher intervention rate. A case series of adults with subglottic stenosis identified strangulation as a significant cause,[20] but the complication rate from small injuries does not seem to have been measured.

The pain and difficulty swallowing that she experienced, while from tissue inflammation insufficient to be seen on CT in this case, prompted consideration of potential hyoid, and laryngeal fractures.[21–25] Laryngeal trauma may take several hours to become apparent and carries a complication rate of 15%.[22] One case report describes airway compromise 51 hours after an initially asymptomatic laryngeal injury.[26,27] Fractures to the larynx or the hyoid bone may have no symptoms until palpated or speaking or swallowing.[21,28] Such injuries may have important consequences for airway and voice.[21,22,24–26,29] Fractures of the hyoid bone require pressure above the larynx and are fractured in up to 1/3 of fatal cases but are uncommon among survivors.[28,30–33] Pressure from surrounding traumatic oedema can also compromise airway structures up to 36 hours later.[34]

Strangulation can lead to pulmonary complications. Pulmonary oedema and pneumonia have been implicated in most in-hospital deaths.[12] The mechanism for pulmonary oedema may be both neurogenic and/or secondary to the large changes in intrathoracic pressure.[12] The changes can result in shortness of breath, and pneumonia several days later.[12,29,35,36]

The neurological sequelae of anoxic brain injury include non-specific symptoms such as loss of consciousness, headache, nausea, confusion and amnesia, as well strokes (ischaemic and haemorrhagic) and other focal neurological deficits.[37–40] In addition, the severity of neurological symptoms has been documented to increase with the number of episodes of strangulation.[41] Delayed anoxic encephalopathy, which can present with blindness, choreathetosis, dystopia, pseudobulbar paralysis can occur days to weeks later, where the victim was initially assessed as normal after the strangulation event.[42,43] Differing susceptibility to anoxia of different regions of the brain is thought to be responsible.[38] Although there are many possible explanations for the nausea, amnesia and confusion in this case, these symptoms could also be explained by anoxic brain injury.

An Australian study of non-fatal strangulation in women presenting to a Western Australian sexual assault service recorded a rate of non-petechial bruising of 18% and a rate of petechial bruising of 22%. A 2013 case series on fatal strangulation recorded a rate of 74% of petechiae of eyes, 36% facial petechiae.[30] It is general accepted that petechiae can be a result of no or poor flow in the veins, with patent arteries resulting in congestion of the small vessels.[44] The decreased flow may result from venous compression during the strangulation but also from high intrathoracic pressures resulting in decreased venous return, as with chest compression or vigorous vomiting. In this case, where the intrathoracic pressures were sufficiently high to cause a tracheal perforation, although petechiae were not found, had they been, it would have been difficult to say whether venous flow was obstructed at the neck, or whether they were a result of the high intrathoracic pressures generated.

Injury to the circulation is a rare but potentially catastrophic complication of strangulation, with unilateral and bilateral carotid artery dissections, stenoses and occlusion having been described, both immediately and with delay of potentially years.[7,29,45–48] Preexisting atherosclerotic lesions may also be damaged, resulting in emboli strokes. Hypotheses regarding why the damage occurs (compared to arterial compressions such as those caused by blood pressure cuffs, generally accepted as harmless) include the possibility that strangulation may be part of a struggle and the pressure may be intermittently applied, with shearing force.[29] The tears to the intima occur as the vessel is compressed between the external force and the transverse processes of the vertebrae. Damage to the arteries is uncommon at a rate 1% from non-fatal strangulation,[10,45] compared to a rate of 5% from hanging,[29] but the results may be lethal. It is worth noting that up to 20% of strokes in people less than 45 years old are caused by extracranial carotid artery dissection, of which trauma is an important cause.[49]

In this case, the woman described knowing that she was strangled, with periods of difficulty breathing, and pain directly over the trachea. She was able to generate sufficient pressure in her trachea to perforate it. This does not fit with being unconscious at that point - transient loss of the airway while unconscious is not known to lead to perforations and when high pressures are generated with a valsalvoer manoeuvre,

the GIT tract acts as a pressure valve. With direct pressure on her trachea, there was no pressure release other than perforation when the pressure was greater than the strength of the trachea. The pressure required to do this is unknown. She also experienced neck pain, dysphonia, dysphagia, confusion, amnesia, nausea and potentially lost consciousness. This description and the signs on physical examination could be explained by a dynamic process that likely had periods of anoxic and hypoxic strangulation. The neurological symptoms (confusion, amnesia and potential loss of consciousness) experienced by this woman may have causes other than strangulation injury (such as a head injury). However, the perforated trachea suggests that she experienced at least airway occlusion. It is unfortunate that she was lost to follow up as there is considerable potential for delayed sequelae.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jflm.2019.05.020.

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