Traumatic Brain Injury Secondary to Distal Ballistic Injury (GSW)

Slide 1

This presentation gives a brief overview of the initial and ongoing management required for TBI as a result of a distal ballistic wound, or gunshot wound, remote from the brain itself. The term ‘bullet’ instead of ‘round’ will be used. Very simple sentences will also used in the audio for ease, and above all accuracy, in translation to French and Arabic.

Slide 2

Much of the ‘evidence’ on the causes and management of TBI appears contradictory. This is however due to the poor quality of some research in even peer reviewed articles. The following slides are based upon American, British and Australian Defence Force statistics and civilian law enforcement trials in the U.S. and Greece.

Slide 3

We begin with a very brief background to ballistics. The basic differences between high and low velocity rounds, or bullets, the different types of wounds they cause and as a result, what to look for. Treatment of the actual distal wound itself will not be discussed in these slides. The focus is on the management in the first hour to 72 hours to avoid the secondary complications that are so common, and to a certain extent avoidable, in such brain related injuries.

Slide 4

Keep it simple. At least as simple as possible.

Every situation is different and it will not always be possible to know the distance to target. It will often be even more difficult to know what type of weapon caused the injury. If however, the approximate distance and the weapon type can be ascertained certain management can be pre-empted.

So what are the basic differences in weapon types and why does it make a difference?

Slide 5

Muzzle velocity, the speed at which the round exits the barrel, applies to both pistols and longarms. In pistol wounds it is most important at close proximity. A pistol wound can cause a complex, aggravating wound from 100 metres away. If the impact point is not to the head or the neck, it is unlikely however to create a pressure wave that will cause secondary traumatic brain injury. As opposed to high powered rifles, there is a rapid decrease in the velocity of a pistol round over a relatively short distance.

Slide 6

Voila a chart showing how rapidly a 9mm round slows during its trajectory. The poor Americans are still using the medieval system of yards and inches but they do know their guns.

Slide 7

The M4/M16 chart shows a NATO standard assault rifle zeroed to 300 metres. This is the approximate equivalent to a civilian .223 round. There will be minor differences due to weapon and round design. The main point is that a distal wound from a high powered longarm can cause TBI at over 300 metres.

Slide 8

Certain pistol rounds at close range and high powered weapons do have the ability to 'punch through' metal and glass of varying thickness. Any such obstacle will normally have a significant effect upon the round before striking human tissue. 'Mushrooming' and/or 'tumbling' of the round are the most common effects. As a result, penetrating power is significantly reduced. As an example, a typical AK- 47 assault rifle round, while still highly lethal, will begin to tumble after striking the outer panel of a normal, unarmoured car door.

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Picture only

Slide 10

For this presentation the basic wound types have been broken down into 4 categories

The first two categories in this slide are wounds caused by high velocity rounds to bone and flesh.

High Velocity

Bone

A high velocity bullet can shatter bone beyond repair. It is this type of injury that is also likely to lead to the transmission of a pressure wave, or shock wave throughout the body. The complex, distal effects referred to in this case is the traumatic injury to the lower part of the brain.

Flesh

The effects of a high velocity bullet on flesh will depend on the bullet type and whether or not it has already struck an obstacle before entering the flesh. If the bullet has struck an obstacle or 'designed' to tumble, the wound is likely to be extensive and complex. If it is a high velocity penetrating round there is the possibility of relatively little damage. High velocity bullets are known to pass straight through flesh in the arms and legs. Some gunshot

wound victims even survive from multiple, high powered bullets that have passed straight through the torso.

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The next 2 categories are low velocity rounds to bone and flesh

Low Velocity

Bone

Low velocity rounds such as the .22 calibre and pistols at greater distances are much less likely to shatter bone and cause the transmission of a pressure wave. As a result, TBI from the wound itself is much less likely.

Flesh

To the flesh however, low velocity bullets, particularly from pistols, can cause extensive damage at the wound site itself. Some tactical groups deliberately use low velocity bullets particularly in close quarters fighting. The objective is for the low velocity bullet to mushroom and stop within the intended target. The risk with high powered bullets is passing through the intended target and striking an innocent victim in close proximity.

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Distance to target is not just academic but an essential, practical consideration.

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Most centrefire, or large calibre rifle bullets, and pistols of the 9mm variety or larger at close range, significantly increase the likelihood of traumatic brain injury from pressure or shock waves alone.

Most handgun bullets at greater distances, over 40 meters for example, can still cause significant damage to flesh. Most handgun bullets at these distances are much less likely to induce a pressure wave from a distant injury that will cause traumatic brain injury.

Flesh wounds from some high velocity bullets may actually cause less damage than low velocity bullets to the same area of flesh.

Slide 14

In addition to the effects of different types of bullets on bone and flesh there are two obvious factors and one less obvious factor that determines the type of wound.

Firstly, the permanent cavity detroyed upon entry of the bullet. This will be affected by the velocity and morphology of the missile.

Secondly, fragmentation of the missile and/or bone. This will not occur in all wounds.

And thirdly, the least obvious and where errors were often made in terms of treatment decisions within the first few hours of GSWs …

Slide 15

The temporary cavity caused by the initial entry of the bullet is essentially caused by the spin of the projectile. For a fraction of a second the fluids around the path of the bullet are put into an outward circular motion spinning away from the bullet path. Certain high velocity projectiles can create a temporary cavity up to 24cm in diameter. The temporary cavity disappears almost as fast as it appears. This does however have serious implications for surgery within the first 3 hours. The vasospasm caused by the temporary cavity will affect the four parameters that are used to decide whether to operate or not. Colour, bleeding, contractility and consistency of the tissue will be severely affected. Past experience has shown a tendency to excise the affected tissue too soon due to these parameters. The initial 3 hours of vasospasm and vasoconstriction is normally followed by a period of hyperaemia, or excessive blood flow. As a result, much of the originally affected tissue will return to normal negating the need for the excessive removal of surrounding tissue.

Slide 16

The temporary cavity is one aspect of a GSW to be aware of. It is however different from TBI as a result of a distal gunshot injury.

Slide17

Instantaneous perturbation of dentate interneuronal networks by a transient pressure wave delivered to the neocortex. There was no easy way to paraphrase that description so it has just been robbed, word for word. In short, it means potential brain damage from the shockwave created by a gunshot wound.

The hippocampus is in the lower central part of the brain. The cells are large, relatively loosely packed and the most susceptible to such pressure waves.

Slide 18

Even though the pressure wave, or waves, may be transmitted all the way to the upper and outer brain, it is the inner, central portion of the brain that is typically the most affected in such injuries.

Slide 19

Another view of the hippocampus. The hypothalamus is positioned above the hippocampus in the brain. This may also be damaged if the pressure wave is strong enough from a high velocity round.

The Blood Brain Barrier is like a Kurdish checkpoint. It lets through anything that's harmless or anything that the brain can benefit from. Everything else, including complex proteins,

bacteria and the like are verboten. The pressure wave is just like a car bomb. It knocks out the defences of the Blood Brain Barrier. One of the culprits to get through is water.

Slide 20

The lateral ventricles in the brain contain choroid plexuses that produce cerebrospinal fluid. A significant pressure wave will cause the overexpression of Aquaporin channels permitting the movement of water into these lateral ventricles. End result, cerebral oedema, or swelling of the brain with water.

Swelling also occurs at the cellular level. Trauma causes a massive release of glutamate, up to 250 per cent above normal levels. Glutamate also affects lactate levels and hence, the type of fluid to be given to trauma victims. i.e. avoid Lactated Ringers, or Hartmanns, in most Resus situations. But that is another subject. Glutamate causes a massive influx of Sodium and Calcium into the brain cells.

Slide 21

This page and the next is for anybody who paid attention in school. For the rest of us, it means one thing. Brain cells swell, leak and die when subjected to such trauma.

Slide 22

For those who can read .....

Slide 23

In most TBI cases, the initial injury is often compounded by a fall, usually involving head impact to complicate matters further.

The next question is how to deal with all of this.

Slide 24

The ABC for the first responder does not change.

Airway, C – Spine protection and haemorrhage control if necessary, are the first priorities over everything else. This is particularly important given the fact that, as mentioned, the primary injury is often excacerbated by a fall involving an impact to the head.

What does vary according to the type of injury and situation is D. Defibrillation is for those with the right equipment and a non-responsive patient with rapid, irregular and/or deteriorating heartbeat (Ventricular tachycardia deteriorating to ventricular fibrillation and potentially asystole).

In slightly less extreme circumstances are the D – Disability factors specific to this type of injury:

Ecchymosis is the pooling of blood under the skin from a rupturd blood vessel and is not the same as a bruise, being the result of direct trauma.

Cerebro Spinal Fluid (CSF) is a clear liquid from the brain and spine. It is produced in the choroid plexuses of the ventricles referred to in Slide 20. If you see this liquid coming from the ears or nose you can be quite certain of trauma to the brain.

Short periods of lucidity are not uncommon in such injuries and are quite deceptive. Such periods can lead to the mistaken belief that the patient has little or nothing wrong with them.

Slide 25

The three main options for dealing with potential TBI and its after affects in the field are fluids, oxygen and Blood Glucose Levels – controlled through insulin. These are for the TBI, not the site of the GSW itself. BGLs and insulin need to be considered once the immediate issues of haemmorrhage, airway, breathing and circulation have been dealt with. Transport to hospital facilities may be delayed and excessive hyperglycaemia will lead to unnecessary complications in the longer term that could have been avoided.

Slide 26

A simple, general rule for fluids, oxygen and BGLs in a field situation is that it is better to have a little more than a little less.

Fluids are essential to maintain blood pressure and cerebral perfusion as protecting the brain is the primary goal. Overloading is to be avoided however as delayed hyperaemia can combine with the fluid infusion to contribute to cerebral oedema and increased intracranial pressure (ICP).

There is a lot of conflicting evidence regarding the type of IV fluids to be used in different situations. The goal however is to simplify the situation as much as possible and not complicate it. For this reason, Hartmann's, or Lactated Ringers, should be avoided in most trauma situations. Before pathology results can be obtained it should be assumed that the patient will have increased lactate levels. For this reason, commence with Normal Saline, not Lactated Ringers, as the initial infusion.

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As important as IV fluids are they do not contribute to O2 carrying capacity and blood will have to be given at some point in severe cases of haemorrhage.

Slide 28

Initial high flow oxygen is administered in many cases of trauma. Evidence is however changing practice to reduce the iniital amount of O2 if the patients O2 saturations can be confirmed. This is easy enough to do with ear/finger saturation probes.

The excessive 'flushing' of CO2 with prolonged high flow O2 contributes to vasoconstriction, reduced blood flow and will actually reduce blood flow to certain areas of the brain.

In short, monitor O2 flow and haemoglobin saturations i.e. don't leave the patient on 10 – 15 L/min of oxygen indefinitely until further help arrives as this does lead to higher mortality rates in TBI patients.

Slide 29

Transient hypoxic periods are common in TBI patients and can actually cause a lowered HR, another reason for the close monitoring of O2 flows and blood saturations.

Slide 30

Trauma patients typically have increased BGL levels as a natural stress response. While this does suit our purposes for running away from sabre toothed tigers we have not evolved to withstand high BGL levels over a sustained period of time.

Both high and low BGLs can aggravate brain injury. Excessively low BGLs can have a devastating effect in a very short period of time, potentially and very quickly leading to death. The goal is to be conservative and gradually bring down excessive BGL levels without causing the patient to 'crash'.

While meta-analysis does not support Intensive Insulin Therapy. What they are really saying is that IIT is essential for these patients but you barbarians in the field can't be trusted to do it properly. And in some hospitals as well for that matter.

Once the BGL has been checked, a slow background dose of of slow acting insulin such as Lantus or Levemir is a good way to initiate the process. Short to medium acting insulin doses with close BGL monitoring could be commenced with properly experienced personnel only. The most important thing is to maintain the BGLs above the 8 mmol range as a margin of safety. In chaotic situations, bringing the BGL toward 15mmol is more than sufficient in the short term.

Slide 31

The following slides are more relevant to hospital based treatment and so shall be kept brief.

Mild hypothermia is proving to be beneficial in the ongoing management of TBI patients preventing the massive glutamate releases mentioned before.

Slide 32

Mannitol improves the brain bio-availability of magnesium sulphate which in turn reduces cerebral oedema, principally in the ventricles of the brain.

Clinical trials stating that MgSO4 was of no benefit in TBI patients were discredited when the trials were analysed. The patients were in such a critical state that they required intracranial brain surgery within 8 hours and/or had the GCS of a rocking chair.

Cyclosporine A has proven to be very effective in reducing cellular oedema by preventing the opening of the mitochondrial 'megachannel'.

Slide 33

Very basic Deep Vein Thrombosis and Pulmonary Embolism Prevention

Compression Stockings or TEDs

Hold the Low Molecular Weight Heparin or low dose unfractionated heparin until 48 – 72 hours after admission. Common sense really. Further uncontrolled bleeds are the last thing that are needed with TBI patients.

In severe TBI cases a PPI infusion of Pantoprazole is the most effective stress ulcer prophylaxis.

Slide 34

In TBI management avoid calcium channel blockers that reduce systemic blood pressure and as a result cerebral perfusion pressure (CPP)

Also avoid corticosteroids that contribute to bleeding, hyperglycaemia, increase in cerebral metabolic rate and fluid retention.

Slide 35

Nothing to say with this slide.

Slide 36

The only point to make on this slide is that we may move one day from the common use of MgSO4 to MgCl2, all because of one less water molecule that may reduce toxicity. The points about Substance P inhibitors, oestrogen, progesterone and Mg combinations will be left to the boffins in the labs to prove their efficacy one way or another.

Slide 37

This last slide is more for the civilian cases of TBI that arrive in Emergency Departments. We need to realise that our job is not the be all and all in the process of helping TBI patients. As much evidence as possible needs to be preserved for the police.

Matt Quade RN

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