Anticoagulation in traumatic brain injury

- Venous thromboembolism prophylaxis in adults with acute traumatic brain injury: a systematic review
- Are isolated linear fractures over major dural venous sinuses a risk factor for sinus thrombosis in mild TBI?
- Thrombosis and Coagulopathy
- Impact of anticoagulant therapy on delayed intracranial haemorrhage after traumatic brain injury: A study on the role of repeat CT scans and extended observation
- Traumatic bilateral deep cerebral venous thrombosis: illustrative case
- A Systematic Review Supporting the Development of the Society for Vascular Surgery Clinical Practice Guidelines on the Management of Blunt Thoracic Aortic Injury
- The Characteristics of Severely Injured Trauma Patients Admitted to a Level I Trauma Center with Pre-Injury Use of Oral Anticoagulation (OAC) or Antiplatelet Therapy (APT)
- Optimizing the Dosing of Heparin for Therapeutic Anticoagulation in Neurocritical Care Patients at High Risk of Bleeding: Report on a Quality Improvement Initiative

Anticoagulation in **traumatic brain injury (TBI)** presents a significant clinical challenge, especially when the injury involves bleeding or hemorrhagic complications, such as **intracranial hemorrhage (ICH)**. The use of anticoagulants in these cases must be carefully considered, as they can exacerbate brain hemorrhage, leading to worsened outcomes. However, anticoagulation may be required for the prevention or treatment of thromboembolic events, such as **pulmonary embolism (PE)**, **deep vein thrombosis (DVT)**, or atrial fibrillation.

Key Considerations for Anticoagulation in TBI:

1. **Risk of Hemorrhage vs. Thromboembolic Risk**:

   1. **Intracranial Hemorrhage (ICH)**: In cases of TBI with suspected or confirmed hemorrhagic lesions (e.g., subdural hematoma, subarachnoid hemorrhage, epidural hematoma), anticoagulation therapy is often contraindicated, as it can worsen bleeding and increase the risk of brain herniation.
   2. **Thromboembolic Risk**: Some TBI patients, especially those who are elderly or have prolonged immobilization, may be at high risk for thromboembolic events like PE or DVT. In such cases, anticoagulation therapy may be needed, but it must be balanced with the risk of bleeding.

2. **Timing of Anticoagulation**:

   1. **Immediate Post-TBI Period**: If the patient presents with significant intracranial hemorrhage, anticoagulation should generally be **avoided** until the hemorrhage is stable or resolving. The **first 24-48 hours** after the injury are critical for monitoring and intervention.
   2. **After Stabilization**: Once the patient's intracranial hemorrhage has stabilized (e.g., no ongoing bleeding or signs of mass effect), and the risk of further hemorrhage is low, anticoagulation therapy may be considered based on the patient's thromboembolic risk.
   3. **Delayed Anticoagulation**: In some cases, anticoagulation can be **delayed for several days**, especially if the patient is at high risk of bleeding. During this time, mechanical prophylaxis (e.g., **sequential compression devices** or **intermittent pneumatic compression (IPC)**) can help reduce the risk of DVT.

3. **Management Strategies in Specific Scenarios**:

1. **Traumatic Brain Injury with Anticoagulant Use Prior to Injury**:
    1. If the patient was on anticoagulation therapy (e.g., warfarin, direct oral anticoagulants [DOACs]), it is essential to reverse the anticoagulation promptly, especially in the presence of brain hemorrhage. The reversal may include:
        1. **Vitamin K** and **fresh frozen plasma (FFP)** for warfarin.
        2. **Prothrombin complex concentrates (PCCs)** or **activated PCC (aPCC)** for DOACs (e.g., rivaroxaban, apixaban).
    2. After stabilization, anticoagulation therapy should be reinitiated with close monitoring.

4. **Use of a Vena Cava Filter (IVC Filter)**:

1. **Inferior vena cava (IVC) filters** may be placed in patients who cannot receive anticoagulation therapy due to an ongoing risk of brain hemorrhage but who are still at high risk for thromboembolic events. This is especially relevant in trauma patients who develop PE or DVT.

5. **Anticoagulation in Mild to Moderate TBI**:

1. In cases where there is no significant hemorrhage or when the hemorrhage is stable, anticoagulation may be initiated cautiously, with close monitoring of intracranial pressure and clinical condition. **Low-molecular-weight heparin (LMWH)** is often preferred for DVT prophylaxis in non-hemorrhagic TBI, as it has a more predictable anticoagulant effect and is less likely to cause complications compared to unfractionated heparin (UFH).

6. **Monitoring During Anticoagulation**:

1. Once anticoagulation is reinitiated, **frequent monitoring** is essential to ensure appropriate therapeutic levels while minimizing bleeding risks.
    1. **INR monitoring** for warfarin.
    2. **Anti-Xa levels** for LMWH.
    3. **Thromboelastography (TEG)** or **rotational thromboelastometry (ROTEM)** may also be used to assess coagulation status, especially in the acute setting.

7. **Multidisciplinary Approach**:

1. Management of anticoagulation in TBI should involve a **multidisciplinary team**, including trauma surgeons, neurologists, hematologists, and intensivists, to ensure appropriate decisions are made based on the patient's clinical status, hemorrhagic risk, and thromboembolic risk.

8. **Surgical Intervention**:

1. In some cases, surgical intervention (e.g., craniotomy) may be required to address significant brain injury. If anticoagulation is needed, **bridging therapy** with short-acting anticoagulants, such as LMWH or heparin, may be considered after surgical stabilization to avoid further thromboembolic events.

Strategies for Specific Types of Anticoagulation:

1. **Warfarin**:

1. **Reversal**: Vitamin K, FFP, or PCC.

2. **Restarting**: Anticoagulation should not be resumed until the patient is neurologically stable, and brain hemorrhage has resolved or improved.

2. **Direct Oral Anticoagulants (DOACs)**:

1. **Reversal**: Specific reversal agents like **idarucizumab** for dabigatran or **andexanet alfa** for factor Xa inhibitors (e.g., rivaroxaban, apixaban).
2. **Restarting**: Similar to warfarin, DOAC therapy should be restarted cautiously, taking into account the risk of brain hemorrhage and the patient's thromboembolic risk.

3. **Low-Molecular-Weight Heparin (LMWH)**:

1. **Use for DVT Prophylaxis**: If the brain injury is stable, LMWH is preferred for thromboprophylaxis due to its more predictable anticoagulation effects and lower bleeding risk compared to UFH.
2. **Restarting**: LMWH can often be restarted sooner than warfarin or DOACs but must be carefully monitored.

4. **Unfractionated Heparin (UFH)**:

1. **Use in ICU/High-Risk Patients**: Heparin may be used in critically ill patients for venous thromboembolism prophylaxis or treatment, but careful monitoring of aPTT levels is necessary.

Conclusion: Anticoagulation in patients with traumatic brain injury requires a **careful, individualized approach** based on the type and severity of the brain injury, the presence of other medical conditions (e.g., PE or DVT), and the patient’s overall clinical stability. The key is to balance the need for **anticoagulation** to prevent thromboembolic events with the **risk of further bleeding** in the brain. Early intervention to reverse any prior anticoagulation, use of **mechanical thromboprophylaxis**, and **timely reinitiation** of anticoagulation (once the risk of bleeding has diminished) are essential to optimizing outcomes.

Anticoagulation are proven risk factors for intracranial hemorrhage (ICH) in traumatic brain injury (TBI).

Standard diagnosis and treatment algorithms for patients who sustained mild traumatic brain injury are only partially applicable to anticoagulated patients [1].

Controversy exists regarding the utility of head computed tomography (CT) in allowing safe discharge dispositions for anticoagulated patients suffering minor head injury.

What is the risk of delayed intracranial hemorrhage in anticoagulated patients with minor head injury and a normal initial head CT scan?

Four observational studies were reviewed that investigated the outcomes of anticoagulated patients who presented after minor head injury.

Overall incidence of death or neurosurgical intervention ranged from 0 to 1.1% among the patients investigated. The studies did not clarify which patients were at highest risk.

The literature does not support mandatory admission for all anticoagulated patients after minor head injury, but further studies are needed to identify the higher-risk patients for delayed bleeding to

determine appropriate management [2].

A total of 1606 patients with blunt TBI charts were reviewed of whom 508 patients had intracranial bleeding on initial CT scan and 72 were on prehospital anticoagulation therapy. Anticoagulation patients were older (P < 0.001), had higher Injury Severity Score and head Abbreviated Injury Scores on admission (P < 0.001), were more likely to present with an abnormal neurologic examination (P = 0.004), and had higher hospital and intensive care unit lengths of stay (P < 0.005). Eighty-four per cent of patients were on antiplatelet therapy and 27 per cent were on warfarin. The Anticoagulation patients have a threefold increase in the rate of worsening repeat head CT (26 vs 9%, P < 0.05). Prehospital Anticoagulation therapy is high risk for progression of bleeding on repeat head CT. Routine repeat head CT remains an important component in this patient population and can provide useful information [3].

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3)
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