The Trauma Professional's Blog

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Emphasis: DVT Subscribe: www.TraumaMedEd.com July 2012

What Is The Origin Of DVT/PE In Trauma Patients?

We have long assumed that pulmonary emboli start as clots in the deep veins of the legs (or pelvis), then break off and float into the branches of the pulmonary artery in the lungs. A huge industry has developed around how best to deal with or prevent this problem, including mechanical devices (sequential compression devices), chemical prophylaxis (heparin products), and physical devices (IVC filters).

The really interesting thing is that less than half of patients who are diagnosed with a pulmonary embolism have identifiable clots in their leg veins. In one study, 26 of 200 patients developed DVT and 4 had a PE. However, none of the DVT patients developed an embolism, and none of the embolism patients had a DVT! How can this kind of disparity be explained?

Researchers at the Massachusetts General Hospital retrospectively looked at the correlation between DVT and PE in trauma patients over a 3 year period. DVT was screened for on a weekly basis by duplex venous ultrsonagraphy. PE was diagnoses exclusively using CT scan of the chest, but also included the pelvic and leg veins to look for a source. A total of 247 patients underwent the CT study for PE and were included in the study.

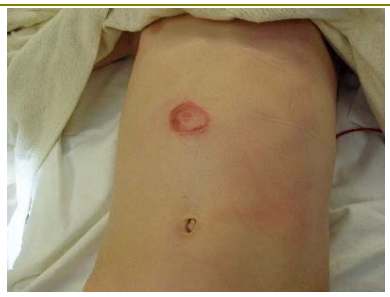
Trauma Calendar of Events

World Trauma Congress
Place: Sulamerica Convention Center
Rio de Janeiro, Brazil
Date: August 22-25, 2012

American Association for the Surgery of Trauma
Place: Grand Hyatt Kauai, Kauai, Hawaii
Date: September 12-15, 2012

IMAGE BANK

Classic Handlebar Injury - Pediatric



A bicycle handlebar struck this child in the epigastrium. You can see an impression from the hole in the plastic covering the handlebar. Pancreatic and duodenal injuries are more likely with this injury pattern.

Inside This Issue

Forty six patients had PE (39% central, 61% peripheral pulmonary arterial branches) and 18 had DVT (16 seen on the PE CT and 2 found by duplex). Of the 46 patients with PE, only 15% had DVT. All patient groups were similar with respect to injuries, injury severity, sex, anticoagulation and lengths of stay. Interestingly, 71% of PE patients with DVT had a central PE, but only 33% of patients without DVT had a central PE.

The authors propose 4 possible explanations for their findings:

1. The diagnostics tools for detecting DVT are not very good. **FALSE**: CT evaluation is probably the "gold

standard", since venography has long since been abandoned

2. Many clots originate in the upper extremities. **FALSE**: most centers do not detect many DVTs in the arms
3. Leg clots do not break off to throw a PE, they dislodge cleanly and completely. **FALSE**: cadaver studies have not show this to be true
4. Some clots may form on their own in the pulmonary artery due to endothelial inflammation or other unknown mechanisms. **POSSIBLE**

An invited critique scrutinizes the study's use of diagnostics and the lack of hard evidence of clot formation in the lungs.

The bottom line: This is a very intriguing study that questions our assumptions about deep venous thrombosis and pulmonary embolism. More work will be done on this question, and I think the result will be a radical change in our use of anticoagulation and IVC filters over the next 3-5 years.

Reference: Pulmonary embolism and deep venous thrombosis in trauma: are they related? Arch Surg. 2009; 144(10):928-932.

Could Deep Venous Thrombosis (DVT) In Trauma Patients Be Caused By Microparticles?

Deep venous thrombosis is commonplace after multiple trauma. A systemic inflammatory process is activated, which leads to an increase in cytokine production. We know that a process called microvesiculation occurs, where cells undergoing apoptosis shed small particles that contain active tissue factor. These types of microparticles have been shown to lead to thrombosis in cancer patients, but the role in trauma patients has not been clear.

Researchers at the University of Rochester performed a simple study looking at injured or burned patients with an Apache II score >20 compared to normal controls. They examined blood drawn after day 2 in the hospital, and looked for microparticles using fluorescent microbeads. They concentrated on differences between 3 trauma patients who did not develop DVT and 2 who did.

Patients who developed DVT had nearly 300% more circulating microparticles than matched controls. It is likely that the majority of those microparticles expressed tissue factor as well.

Bottom line: This exciting work may help explain why trauma patients have a higher DVT rate. Additionally, it may eventually provide us with a blood test that will help pinpoint patients at high risk so we can provide more intensive surveillance and/or more aggressive prophylaxis or prevention.

Reference: Multisystem trauma patients who develop venous thromboembolism have increased numbers of circulating microparticles. Marlene Mathews MD et al. Presented at the 34th Annual Resident Trauma Paper Competition at the AmericanCollege of Surgeons Spring Meeting, Washington DC, 2011.

Does Interrupting DVT Prophylaxis Increase Risk for DVT/PE?

Deep venous thrombosis is a common concern in trauma care. Most trauma centers have well defined protocols for prophylaxis and surveillance. Ongoing use of pharmacologic thromboprophylaxis (PTP) in patients with traumatic brain injury (TBI), or in patients who need surgical procedures is controversial. We have all experienced some form of "prophylaxis interruptus", where our orthopedic or neurosurgical colleagues want us to forego or interrupt ongoing administration of heparin products. Does this create new problems?

A trial was conducted at two Denver trauma centers, trying to clarify the optimal administration of PTP in patients with stable TBI. One cohort received PTP, the other did not (either not indicated, short stay, or already on blood thinners). The group receiving PTP was also stratified into those who received it continuously and those who had interruptions in treatment.

They found that the incidence of DVT and PE was similar for patients receiving PTP vs those not receiving it. The two groups were very different, though, because the ones who did not receive it had less severe injuries and were more likely to be ambulating by discharge. The most interesting finding was that being started on PTP and then interrupting it increased the incidence of DVT fourfold.

What is it about prophylaxis interruptus that is so risky? First, there were only 480 patients in this study, so statistical anomalies could be present. Could it be that the conditions (TBI) and operations that cause it to be interrupted greatly increase the risk? Unfortunately this study can't answer those questions.

The bottom line: DVT and its prophylaxis is still a muddy concept. What we really need to do is to find out if PTP is really necessary in all the patients in whom we are using it. It would also be helpful if we knew how harmful it really is in patients with significant bleeding in their head, or in patients who need to undergo surgery. One alternative, if this paper pans out, is to begin with mechanical prophylaxis until cleared by neurosurgery and all operations are completed. For now, it's not yet appropriate to change your existing practice and procedures.

Reference: Interrupted pharmocologic prophylaxis increases venous thromboembolism in traumatic brain injury. J Trauma 70(1):19-26, 2011.The term "prophylaxis interruptus" was coined by Tom Esposito in his discussion of this paper.

Brain Injury and Chemical Prophylaxis for DVT

Deep venous thrombosis (DVT) and its complications are recognized and common problems in trauma patients, particularly those with traumatic brain injury (TBI). We know that giving chemical prophylaxis like heparin and low molecular weight heparin (LMWH) reduces the risk. Unfortunately, trauma professionals (and neurosurgeons in particular) are reluctant to give it after acute TBI for fear of making intracranial hemorrhage worse.

Froedtert Hospital in Milwaukee modified their protocol for TBI patients to allow chemical prophylaxis to start 24 to 48 hours after a 24 hour followup CT that showed no progression of any bleeding. Therefore, prophylaxis could be started 48 to 72 hours after injury. They used subcutaneous heparin three times daily, or LMWH twice daily. All others received mechanical prophylaxis and were screened twice weekly by duplex ultrasound. The chemical prophylaxis group was not screened routinely.

A total of 812 patients were studied, half of whom received early prophylaxis per protocol. The average Abbreviated Injury Score for the head in these patients was 3.4, which represents fairly serious injury. There was a significant decrease in the incidence of DVT in the chemical prophylaxis group (1% vs 3%). More intriguing, there was a lower rate of injury progression in this group as well (3% vs 6%), although not quite statistically significant.

Bottom line: Although this is a small and retrospective study, it was well designed and relatively large compared to most other similar work. It shows that use of chemical prophylaxis works in patients with serious TBI, and appears to be safe. Similar protocols should be considered by trauma program multidisciplinary operations committees to further systematize this process.

Reference: Safety and efficacy of prophylactic anticoagulation in patients with traumatic brain injury. J Am Coll Surg 213:148-154, 2011.

Brain Injury and DVT Prophylaxis Part II

Shortly after the previous work was published, another article now helps to refine the selection of the heparin product to use. A retrospective review looked at 386 ICU patients with a head Abbreviated Injury Score (AIS) > 2. A total of 57 received mechanical prophylaxis, the remainder received heparin products. Chemical prophylaxis consisted of subcutaneous enoxaparin 30mg bid or unfractionated heparin 5000u tid, at the whim of the attending neurosurgeon.

The heparin group had a slightly but significantly higher Head AIS (4.1 vs 3.8). The drugs were started at the same time post-injury, about 48 hours from admission. Unfractionated heparin was found to be inferior to enoxaparin. The unfractionated heparin patients had both a higher rate of pulmonary embolism, and were more likely to have progression of any intracranial hemorrhage (12% vs 5%). The authors claim a significantly lower DVT rate, but information in their data tables do not support this. Additionally, their overall DVT rate is very low, most likely because they did not routinely screen for it.

Bottom line: The head injury / DVT prophylaxis literature is expanding rapidly. It's time to start working with your neurosurgeons to initiate chemoprophylaxis early (within 48 to 72 hours

from injury once any intracranial bleeding is stable). And it looks like the drug of choice is enoxaparin, not unfractionated heparin.

Reference: Safety and efficacy of heparin or enoxaparin prophylaxis in blunt trauma patients with a head abbreviated injury severity score >2. J Trauma 71(2):396-400, 2011.

DVT In Children

Deep venous thrombosis has been a problem in adult trauma patients for some time. Turns out, it's a problem in injured children as well although much less common (<1%). However, the subset of kids admitted to the ICU for trauma have a much higher rate if not given prophylaxis (approx. 6%). Most trauma centers have protocols for chemical prophylaxis of adult patients, but not many have similar protocols for children.

The Medical College of Wisconsin looked at trends prior to and after implementation of a DVT protocol for patients < 19 years old. They used the protocol below to assess risk in patients admitted to the PICU and to determine what type of prophylaxis was warranted.

The need for and type of prophylaxis was balanced against the risk for significant bleeding, and this was accounted for in the protocol. The following significant findings were noted:

- The overall incidence of DVT decreased significantly (65%) after the protocol was introduced, from 5.2% to 1.8%
- The 1.8% incidence after protocol use is still higher than most other non-trauma pediatric populations
- After the protocol was used, all DVT was detected via screening. Suspicion based on clinical findings (edema, pain) only occurred pre-implementation.
- Use of the protocol did not increase use of anticoagulation, it standardized management in pediatric patients

Bottom line: DVT does occur in injured children, particularly in severely injured ones who require admission to the ICU. Implementation of a regimented system of monitoring and prophylaxis decreases the overall DVT rate and standardizes care in this group of patients. This is another example of how the use of a well thought out protocol can benefit our patients and provide a more uniform way of managing them.

Reference: Effectiveness of clinical guidelines for deep vein thrombosis prophylaxis in reducing the incidence of venous thromboembolism in critically ill children after trauma. J Trauma 72(5):1292-1297, 2012.

VTE Prophylaxis Guidelines

For patients at **high risk of VTE**[1] with **low risk of bleeding**[2]:
- anticoagulate with low molecular weight heparin at 0.5mg/kg subcutaneous, twi daily until hospital discharge

For patients at **high risk of VTE**[1] with **high risk of bleeding**[3]:
- apply sequential compression devices
- on PICU day 7 obtain screening ultrasound of bilateral lower extremities, and upper extremity if CVL is present

For patients at **low risk of VTE**[4]:
- no anticoagulation or other clinical intervention indicated

Risk Factors for VTE:
- projected immobility > 5 days
- Glasgow Coma Scale less than 9
- presence of CVL
- spinal cord injury
- complex lower extremity fracture
- operative pelvic fracture
- use of inotropes
- CPR during resuscitation
- exogenous estrogen
- chronic inflammatory state
- history of previous clot
- known thrombophilia
- current malignancy

Risk Factors for Bleeding:
- intracranial bleed
- solid organ injury
- planned surgical intervention or invasive procedure in the next 24 hours
- heparin allergy
- high risk of severe bleeding
- renal failure

[1]High risk of VTE defined as age greater than 13 years OR age less than 13 years with four or more risk factors for VTE.
[2]Low risk of bleeding defined as no risk factors for bleeding.
[3]High risk of bleeding defined as one or more risk factors for bleeding.
[4]Low risk of VTE defined as age less than 13 years AND three or fewer risk factors for VTE.

VTE = venous thromboembolism; PICU = pediatric intensive care unit; CVL = central venous line; CPR = cardiopulmonary resuscitation

http://bit.ly/OafsVd
Pediatric DVT Prophylaxis Protocol
Download by scanning the QR code or entering the link exactly in your browser.

www.TheTraumaPro.com	@regionstrauma	Michael.D.McGonigal@HealthPartners.com	Michael.D.McGonigal