MDT Morbidity & Mortality Peer Review Committee

14 AUG 2023

LITERATURE REVIEW COMPILATION

URETER TRAUMA

TAKE A LOOK

VERONICA BUSTILLO-ARUCA, MD

- Most cases of ureteral injury are secondary to penetrating trauma; 2-5% of GSWs
- It’s rare, 1% of urologic trauma
- It’s not an emergency, you CAN look later
- Delay in diagnosis is common, very common. 38.2%
- 60% of them involve the proximal ureter
- After day 6 we dramatically increase patient morbidity.
- Early diagnosis is the single most important prognostic factor

WHICH ZONE?

1, 2, 3

- It’s not expanding, doesn't matter: take a look!
- Too unstable to look back there:
  - CT pyelogram
  - 2nd Look
  - Bedside cystoscopy with fluro
- Call a friend: urology consultation early on

Literature Review

Timing of DVT Prophylaxis

In Trauma Patients with Solid Organ Injury Undergoing NOM

Eric Raschke, DO

Virginia Mason Franciscan Health

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When is It Safe to Start VTE Prophylaxis After Blunt Solid Organ Injury? A Prospective Study from a Level I Trauma Center

Schellenberg, M., Inaba, K., Biswas, S. *et al.* When is It Safe to Start VTE Prophylaxis After Blunt Solid Organ Injury? A Prospective Study from a Level I Trauma Center. *World J Surg* **43**, 2797–2803 (2019). https://doi.org/10.1007/s00268-019-05096-7

A recent prospective study published in World of Trauma looking at safety and efficacy of starting DVT prophylaxis in blunt abdominal trauma patients with solid organ injury, in attempt to validate retrospective studies performed in the past.

Methods

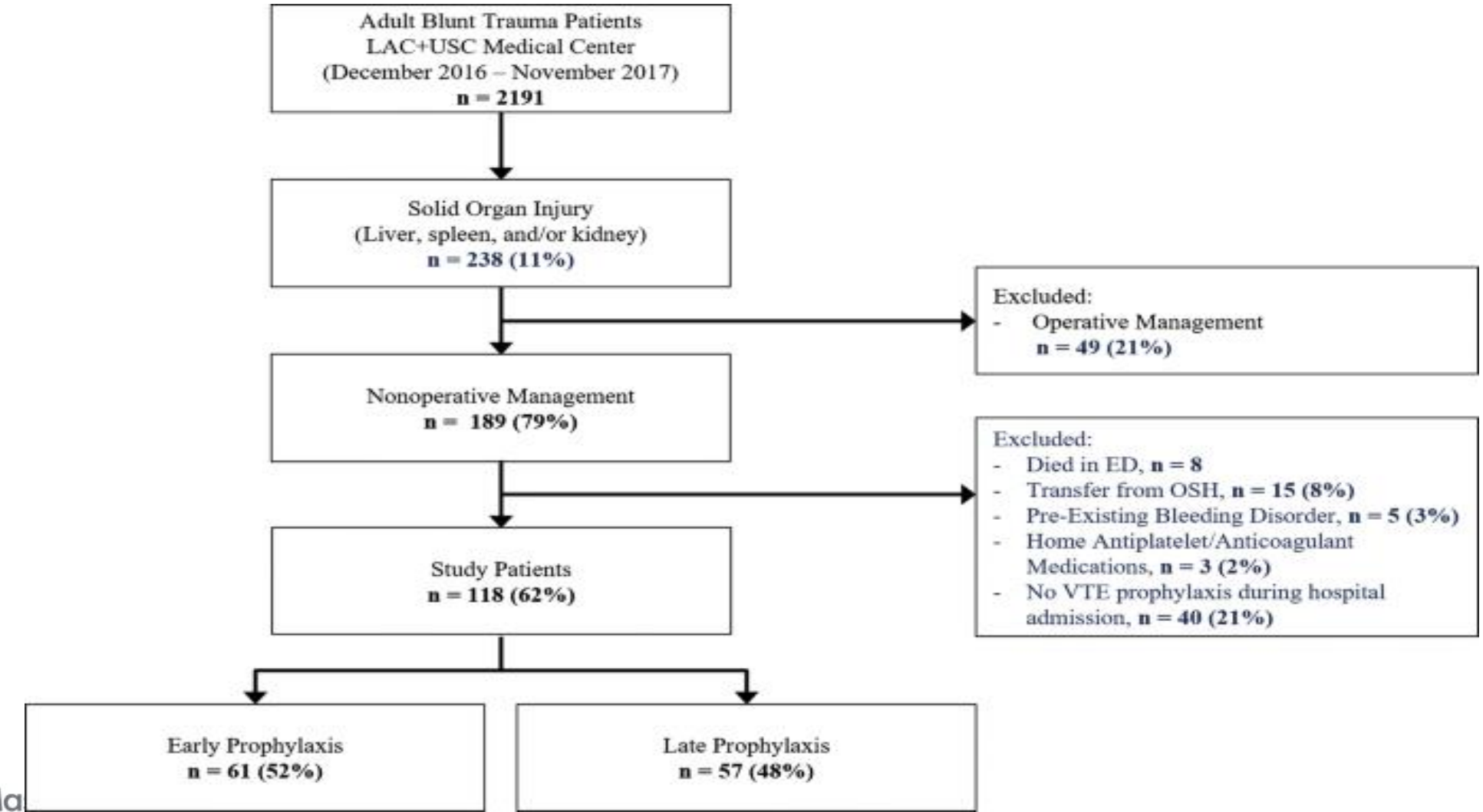
- Inclusion criteria: >15 of age presenting after blunt trauma (12/01/16–11/30/17) were prospectively screened. Patients were included if solid organ injury (liver, spleen, kidney) was diagnosed on admission CT scan and nonoperative management was planned.

- Exclusion Criteria: ED deaths, transfers, patients with pre-existing bleeding disorders or home antiplatelet/anticoagulant medications, and those who did not receive VTE prophylaxis were excluded

Methods

- Demographics, injury/clinical data, type/timing of VTE prophylaxis initiation, and outcomes were collected.
- Patients were dichotomized into study groups based on VTE prophylaxis initiation time: Early (≤48 h) vs Late (>48 h after admission)
- The primary study outcome was VTE event rate. Secondary outcomes included hospital length of stay, intensive care unit days, need for and volume of post-prophylaxis blood transfusion, need for delayed (post-prophylaxis) interventional radiology or operative intervention, failure of nonoperative management, and mortality.

Patient Selection



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Demographics

Table 1 Patient demographics, clinical data, and injury data

	All patients (n = 118)	Early prophylaxis (n = 61, 52%)	Late prophylaxis (n = 57, 48%)	p
Demographics				
Age, years	36 [27–55]	36 [27–54]	36 [27–56]	0.631
Male	78 (66%)	39 (64%)	39 (68%)	0.698
Clinical data on admission				
SBP, mmHg	127 [112–146]	126 [105–144]	129 [115–149]	0.250
SBP < 90 mmHg	13 (11%)	8 (13%)	5 (9%)	0.561
HR, bpm	96 [79–108]	95 [79–107]	97 [79–113]	0.465
HR > 120 bpm	17 (14%)	5 (8%)	12 (21%)	0.066
GCS	15 [14–15]	15 [14–15]	14 [13–15]	0.009
Injury severity				
ISS	22 [14–26]	17 [14–22]	22 [17–27]	0.002
AIS head/neck	0 [0–2]	0 [0–0]	0 [0–3]	0.368
AIS face	0 [0–0]	0 [0–0]	0 [0–0]	0.395
AIS chest	2 [0–3]	3 [1–3]	2 [2–3]	0.522
AIS abdomen/pelvis	3 [2–3]	3 [2–3]	2 [2–3]	0.920
AIS extremities	2 [0–2]	2 [0–2]	2 [0–2]	0.101
AIS external	1 [0–1]	1 [0–1]	1 [0–1]	0.689
Solid organ injury				
Liver	57 (48%)	31 (51%)	26 (46%)	0.586
Spleen	43 (36%)	22 (36%)	21 (37%)	1.000
Kidney	34 (29%)	17 (28%)	17 (30%)	0.841
>1 Solid organ injury	19 (16%)	12 (20%)	7 (12%)	0.323
Associated injuries				
TBI	23 (19%)	5 (8%)	18 (32%)	0.002
Pelvic fracture	42 (36%)	22 (36%)	20 (35%)	1.000
LE fracture	26 (22%)	9 (15%)	17 (30%)	0.074
Need for angioembolization	22 (19%)	10 (16%)	12 (21%)	0.637

Continuous variables presented as median [interquartile range] and compared using the Mann–Whitney U test. Categorical variables presented as n (%) and compared using the Fisher's exact test

SBP systolic blood pressure, *HR* heart rate, *Bpm* beats per minute, *GCS* Glasgow coma scale score, *ISS* injury severity score, *AIS* abbreviated injury scale, *TBI* traumatic brain injury, *LE* lower extremity

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Outcomes

	All patients (n = 118)	Early prophylaxis (n = 61, 52%)	Late prophylaxis (n = 57, 48%)	p
VTE*	8 (7%)	2 (3%)	6 (11%)	0.153
DVT*	5 (4%)	0 (0%)	5 (9%)	0.024
PE*	5 (4%)	2 (3%)	3 (5%)	0.672
Hospital LOS	9 [5–21]	6 [4–11]	14 [7–35]	<0.001
Need for ICU admission	104 (88%)	52 (85%)	52 (91%)	0.398
ICU LOS	4 [3–9]	3 [2–6]	7 [4–12]	<0.001
Mortality	3 (3%)	2 (3%)	1 (2%)	1.000
Need for post-prophylaxis transfusion	31 (26%)	13 (21%)	18 (31%)	0.058
Volume of post-prophylaxis transfusion	0 [0–0]	0 [0–0]	0 [0–0]	0.180

Continuous variables presented as median [interquartile range] and compared using the Mann–Whitney U test. Categorical variables presented as n (%) and compared using the Fisher's exact test

VTE venous thromboembolic event (DVT and PE), *DVT* deep vein thrombosis, *PE* pulmonary embolism, *LOS* length of stay (days), *ICU* intensive care unit

*There were 10 VTEs in 8 patients

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Conclusion

- In patients with nonoperative blunt solid organ injuries, early initiation of VTE prophylaxis resulted in a lower incidence of DVTs without an associated increase in bleeding or need for intervention. Although no difference in PE was noted, early initiation of VTE prophylaxis is likely to be safe and beneficial for patients with blunt solid organ injury.

Literature Review

HYPONATREMIA IN HEAD INJURY

IMPULSIVITY IN TBI

COMPLIANCE/CAPACITY ISSUES IN ACUTE COGNATIVE IMPAIRMENT

Dr Teresa Bell

Literature Review

"...hyponatremia in head injury..."

July 2020: "Hypo-Na in the Neurologically Ill Pt"

- Review article. Includes many types of brain injury – TBI, stroke, seizures, aneurysm, etc.
- Mild: 130-134; Mod. 120-129; Severe <120
  - Acute sx: N/V; HA; Szr; resp. arrest; coma / death.
  - Chronic sx: N/V; fatigue; gait / fall issues; attention deficits
- SIADH: hypervolemic. Increased ADH → decreased renal excretion of water. Five mechanisms! Urine Na and Osm will be high (theoretically).
- Cerebral Salt Wasting: hypovolemic → failure to tx appropriately can increase cerebral vasoconstriction. Unclear mechanisms. High urine Osm & Na.
- Treat to symptoms, rather than a Na level.

Differentiating ***Cerebral Salt Wasting*** and
Syndrome of Inappropriate Antidiuretic Hormone.

	Cerebral Salt Wasting	**SIADH**
Extracell. volume status	Decreased	Unchanged
Heart rate	Unchanged to increased	Unchanged
Body weight	Decreased	Unchanged
Urine output	Unchanged to increased	Unchanged to decreased
Hematocrit	Increased (relative to baseline)	Unchanged
Blood urea nitrogen	Increased	Unchanged
Serum bicarbonate	Increased	Unchanged
Serum urate	Unchanged to decreased	Unchanged
Urine osmolality	>300 mOsm/kg	>100 mOsm/kg
Urine Na excretion	>40 mmol/L	>40 mmol/L

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Literature Review

“...hyponatremia in head injury...”

June 2008: “Disorders of Na Balance after Brain Injury”

- Review article. Includes many types of brain injury – TBI, stroke, seizures, aneurysm, etc.
- SIADH: hypervolemic. Increased ADH → decreased renal excretion of water. Urine Na and Osm will be high.
  - Usually self-limited. Fluid restrict. Salt load. “Vaptans.”
- Cerebral Salt Wasting: hypovolemic. Renal Na wasting with polyuria. Frequently dx'd as SIADH.
  - 2-4 weeks duration. Volume load and Salt load. Fludrocortisone.

Common causes of hypotonic hyponatraemia

Hypovolaemic hyponatraemia	Normovolaemic hyponatraemia	Hypervolaemic hyponatraemia
CSWS	SIADH	SIADH
Diuretics (including osmotic)	Traumatic brain injury	Congestive cardiac failure
Ketonuria	Subarachnoid hemorrhage	Nephrotic syndrome
Diarrhoea/vomiting	Other CNS pathology	Cirrhosis
Sweating	Drug induced	Acute renal failure
Blood loss	Pulmonary pathology	Iatrogenic
Adrenal insufficiency	Thiazide diuretics	
	Adrenal insufficiency	
	Hypothyroidism	
	Iatrogenic	

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Literature Review

"...hyponatremia in head injury..."

June 2022: "Determinants of Hypo-Na after TBI"

- TBI pts over 1y, excluding chronic hypo-Na and death <72h.
- 30% (of 283 pts); increased with greater age, worse CT scan esp. "diffuse pattern."
- "Significant" ("2pts < normal, more than 2d") spiked from 7 → 11 days post-injury; "Borderline" (1-2pts < normal, 1-2d duration") had earlier onset and longer course.

Literature Review

“...hyponatremia in head injury...”

Sept. 2011: “Hypo-Na in pts with TBI”

- 40 consecutive TBI pts with “mod. to severe” CHI.
- Daily Na x14d, CVP measurements, FEUA.
- 27% hypo-Na, 6/9 in <7d; 5 c inc. CVP, 3 c low CVP. FEUA did not correlate
- CT scoring was more predictive of hyponatremia than GCS.

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“...hyponatremia in head injury...”

Dec 2017: “Practical Protocol”

- Retrospective, 1500 consecutive pts c TBI. “Hypo-Na = <135, natriuresis = >40mEq/L.
- 13% of pts had hypo-Na; SAH was most common CT finding.
- Early treatment with fludrocortisone decreased hosp. LOS – and eliminated need to differentiate SIADH and CSW

Literature Review

Available treatments

- Volume restriction – or volume replacement!
- Loop diurectics
- Increased solute load: Na, urea, protein
  - 3% NaCl 100cc / 10m, up to 3x for sx
  - Correct no more than 12mEq/L per 24h.
- “Vaptans” - iv conivaptan (esp. if CHF a factor; has TBI indication); oral tolavaptan.
- Fludrocortisone will increase Na... but not yet shown to improve outcomes. ***(01-0.4mg, daily)***

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Literature Review

Traumatic dislocations of the knee

Chrystal Buchanan, PA-C

Traumatic dislocations of the knee

- Determine the Mechanism of injury – high velocity vs low velocity (vascular injury 5% and nerve injury is about 20%)
- There are 5 types of dislocation: anterior (31%), posterior (25%), lateral (13%), medial (13%), rotary (4%)
- Anterior dislocations usually occur from hyperextension of the knee and often the PCL and ACL will be torn
- Posterior dislocation usually has disruption of both cruciate ligaments

Literature Review

- Clinical findings:
  - Popliteal artery and vein injury is common
  - Peroneal nerve injury occurs in 20-40% of knee dislocations. Usually both cruciates and at least one collateral ligament are disrupted. If there is a nerve injury, be concerned for a possible vascular injury

Literature Review

- Subluxation/Dislocation of the Patella
  - Mechanism of injury: usually on twisting injury with the knee extended and foot externally rotated. Or direct blow to knee.
  - Symptoms: knee pain, complaints of instability

- Imaging: Xrays to rule out fracture or loose body. AP, lateral, sunrise views. Other imaging CT and MRI to further rule out loose bodies

Literature Review

- Treatment is nonoperative with bracing for first time dislocation without bony avulsion or presence of articular loose bodies.
  - This is followed by immobilization
  - Recurrent dislocation with non-op treatment is between 15-50% at 2-5 years.

- Operative management is indicated for chronic and recurrent patellar instability.

Literature Review

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