Compressive Spinal Cord Injury Secondary to Hematoma: Systematic Review and Contrast with Traumatic Spinal Cord Injury

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Background and Objectives: Non-traumatic spinal cord injuries (SCIs) include heterogeneous etiologies often lumped together for research comparisons with traumatic SCI. SCI secondary to hematoma is one such non-traumatic etiology; however, hematomas secondary to procedural complications are typically classified as traumatic by Model Systems criteria. While the compression secondary to a hematoma does exert a mechanical force on the spinal cord and can be considered a trauma, the degree and rate of compression compared to conventional impactacceleration traumatic SCI likely exert different pathophysiologic responses, functional consequences, and expected recovery trajectory / outcomes. We aim to more precisely characterize features unique to SCI secondary to hematomas including subtypes, demographics, risk factors and comorbidities, prognostic factors, and outcomes compared to better-studied traumatic SCI.

Design: Systematic review; PRISMA guidelines. Two investigators completed a comprehensive database search. Pre-determined exclusion criteria included: studies not in English, animal studies, and individual case reports or limited case series. Participants included individuals of all ages with SCI secondary to non-traumatic hematomas. Data collected: population demographics (age, sex, comorbidities/risk factors), unique clinical features, prevalence/severity of functional sequelae (motor/sensory dysfunction, neurogenic bowel/bladder, sexual dysfunction, neuropathic pain, spasticity, syrinx formation, DVT, pressure injury), functional outcomes, and psychosocial outcomes (employment, disposition, mental health).

Results: Twelve studies were included capturing 2,253 cases of hematoma-related SCI, further classified by (spontaneous. pregnancyhematoma subtype procedure-related). Differences in associated demographics and risk factors were observed both within hematoma-induced SCI (between sub-etiologies) and between hematoma-induced and traumatic SCI. Notable gaps in the literature were identified for longterm neurologic sequelae and functional outcomes from hematoma-induced SCI. We present insights into hematoma-related SCI demographics and outcomes and provide rehabilitation-specific and prognostic considerations in contrast with traumatic SCI Full details at right.

Let's talk: Clinical Features

Hematoma-induced SCI have a more insidious onset compared to traumatic SCI

- · 85% present with acute to subacute pain at level of injury
- ~40% have bladder symptoms
- Motor and sensory impairments occur in 60-90%, but are more subtle
- ~5% present atypically with headache, vomiting, cerebral edema, nystagmus, or impaired consciousness (more common in extensive hematoma; 76% span entire spinal canal)
- Location/size: cervical to upper thoracic; mean segment span 3-6 levels

Demographics & Risk Factors

Category	Diagnosis	Age	Sex	Risk Factors (RFs) & Comorbidities	Coagulopathies and Anticoagulation
	Spontaneous	Median: 52 years old	55% male 45% female	HTN 23%, 15.2% other CV RFs 5% underlying vascular malformation Majority of cases: no relevant PMHx.	44% on anticoagulation 5% with coagulopathies
Hematoma	Peri-partum Spinal Epidural	Pregnancy- related: Mean 27.9 Post-partum: Mean 29.75	100% female	 81% had no vascular risk factors or anticoagulation therapy 1 case previous spontaneous spinal epidural hematoma, 1 HTN + preeclampsia,1 case HELLP w/o HTN >50% had spinal or epidural anesthesia 	
	Procedure- related			Spinal procedure	34.5% of cases had coagulopathy
Traumatic		Mean 35.6 Mode: 19	4:1 male to female ratio	EtOH or other substance use; 25% assoc. w/alcohol use	

Outcomes and Prognosis

Diagnosis	Initial Functional Score	Follow-up Functional Score	Probability of Functional Improvement	Factors Predicting Positive Outcome	Factors Predicting Negative Outcome	Probability of Favorable Outcome
Hematoma	Frankel: A 41% B 14% C 32% D 10% E 4% A=complete	40% with recovery ≥ Frankel C; for Frankel C, D, or E, ≥ to 1 grade improvement	Frankel: A 41% B 75% C 91% D 96% E 89%	Paraplegia <24hrs Only cauda involved Location above C6 Location below L1 Frankel B-E Controversial: fewer spinal segments involved	Frankel A (complete) Neurological deficits Requiring surgery (50% unfavorable outcome, vs 18% of non-operative cases) Comorbid coagulopathy or anticoagulation	43% clinically significant recovery, 36.2% mild recovery, 20.8% minimal to no recovery or died of hematoma associated complications
Traumatic	AIS Grade: A 43% B 11% C 19% D 27% A=complete	<u>1 year f/u AIS</u> <u>Grade:</u> A 31% B 11% C 14% D 43% E 1%	A -> D progression in 1yr <5% C -> D progression much higher	Incomplete injury, particularly if motor preservation below Pinprick sparing below level of injury Timely surgical decompression	AIS A (complete) Transection or hemorrhage Negative modifiers: age (>50), obesity, # of comorbidities Level of injury: higher = less likely to be independent or to ambulate	Proportional to AIS grade Modified by comorbidities, support system, psychosocial factors

Functional Consequences

Diagnosis	Sensory Symptoms	Motor Symptoms	Ambulation/Mobility	Neurogenic Bladder
Hematoma	64% experience sensory deficits: 13% hypesthesia/hypalgesia, 45% paresthesia	89% experienced motor symptoms. Of these: 43% paraparesis 46% paraplegia 2% tetraparesis 3% tetraplegia 3% hemiplegia 3% monoparesis	41% achieved independent ambulation w/o devices 18% independent and ambulatory with aid 24% wheelchair users	42.8% experience bladder dysfunction as initial symptom o onset. No data on long-term bladder involvement
Traumatic	quantity of spinal cord ti of motor and/	neurological injury and preserved ssue. All experience some degree or sensory impairment. AlS grades above.	Overall predicted ambulation based on AlS grade: A 3%, B 50%, C 75%, D 95% Ambulation at IPR discharge (old data, longer LOS): A <1%, B 1-15%, C 28-40%, D 67-75%.	Present in 70-84% of patients

No data for: neurogenic bowel, spasticity, neuropathic pain, pressure injuries, sexual dysfunction, syrinx formation, DVT/PE, IPR LOS / functional gains, disposition, or psychosocial outcomes.



When compared to traumatic SCI, hematoma-induced SCI has unique demographics, risk factors, and different clinical presentations, though functional outcomes and prognostic data are lacking. We need to study them more.

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Conclusions

Hematoma-induced SCI is vastly understudied, particularly in areas including: rates of functional sequelae, functional outcomes, and psychosocial outcomes. Published data on the various etiologies are sporadic and imprecise. In addition, efficacy/outcomes of acute inpatient rehabilitation are not reported and rehabilitation-relevant interventions for common SCI sequelae including neurogenic bladder, neurogenic bowel, neuropathic pain, spasticity, and more have not been explored in the hematoma-induced SCI population specifically. Further study is needed to characterize prognostic features, rehabilitation considerations, and intervention efficacies to provide adequate guidance, expectant management, prognostication, and treatment development/targeting for patients who experience this type of SCI.