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Acute spinal epidural hematoma, clinical and etiopathogenic diagnostic difficulties – Case presentation and synthesis of the literature

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Abstract

Introduction: Epidural hematoma has a double anatomopathological topography: intracranial and/ or spinal. Its etiology is complex: post-traumatic (spinal trauma, or lumbar puncture), iatrogenic (secondary to an inadequate anticoagulation or antiplatelet treatment), congenital or acquired disorders of coagulation (leukemia, hepatic cirrhosis), secondary to intense Valsalva maneuvers (e.g. during labor, or an intense physical effort), and idiopathic.

The purpose of this article is to present a clinical case of acute spinal epidural hematoma (SEDH) with atypical clinical picture and a puzzled pathophysiological mechanism, and also a brief review of the relevant literature.

Case presentation: An 80-years-old male patient, with locomotor disability (bilateral congenital foot deformity), and multiple cardiovascular comorbidities (chronic atrial fibrillation (AF), dilated cardiomyopathy and contractile dysfunction (chronic heart failure, with left ventricle ejection fraction 40 %), chronically anticoagulated with a vitamin K antagonist (acenocumarol). The elderly submitted a body-level fall without cranial trauma, event followed by a short loss of consciousness (without convulsions or sphincter relaxation). He suffered a low-energy cervical fracture (C7 vertebral injury) and a posterolateral acute SEDH at C3-Th2 vertebral levels.

Decompressive hemilaminectomy at the C4-Th2 levels and evacuation of the SEDH, was performed during the early sub-acute phase. The patient was transferred in our rehabilitation clinic as C4 AIS-C tetraplegia (global motor score 50/100), neurogenic bladder and bowel, with post surgical wound dehiscence (healed per secundam). The subject had a favorable neurological evolution and was discharged as C7 AIS-D tetraplegia (global motor score 81/100).

Discussion: The case particularity consists in a puzzled etiopathogenetic mechanisms and difficulty to accurately indicate the chronological chain of events generating the acute SEDH.

An overdosed anticoagulant therapy might be incriminated as an iatrogenic cause for a “spontaneous” SEDH, but most probably its etiology is complex, probably traumatic, consequence of the cervical spine fracture due the low-intensity biomechanical impact.

The complex predisposing circumstances to accidental fall in our elderly patient were due to the:

- impaired, unstable locomotor function, secondary to his bilateral congenital clubfoot deformity / disability
- chronic AF, contractile dysfunction and hypodiastolic phenomena, with cardiogenic syncope and global brain ischemia or transient ischemic cerebral attack.

Despite the good immediate outcomes, his future functional prognosis might be poor, due to the advanced age, severe cardiovascular pathology and the complex disturbances of the neuro-myo-artro-kinetic apparatus (major impediments of the somatic / **body** functions and structure). This health-related condition had severe repercussions on the subject's **activity** (related to tasks and basic activities of daily living) and **participation**, affecting the outcome of rehabilitation, and his quality of life.

Conclusions: Clinicians should consider the remote risk of SEDH (even with atypical clinical presentation) in patients with AF and anticoagulant medication.

Despite a postponed decompressive intervention (imposed by the severe comorbidities), our patient neurologically improved without recurrence, following a complex neurorehabilitation program.

Key words: *spinal epidural hematoma, elderly, same-level fall, tetraplegia, hemilaminectomy, rehabilitation,*

Introduction Spinal epidural hematoma (SEDH) is a rare but serious condition. Its incidence is one per million inhabitants [1], accounting for less than 1% (0.3% to 0.9%) of the spinal epidural space-occupying lesions [2], [3].

Epidural hematoma has a double anatomopathological topography: intracranial and/or spinal [4]. Any level of the spinal canal may be involved, but highest frequency of SEDH distribution

is in the upper thoracic (Th3-5) and middle to low cervical regions (C4-6) [5], [6].

The majority of hematomas (53%) is located posterior to the spinal cord, 23% are located posterolateral, 18% anterior, whereas 5% are lateral and 1% are circumferential to the cord [6].

Acute SEDH is a rare neurosurgical emergency most often associated with acute symptomatic spinal cord compression and potentially permanent

neurologic deficits. Most patients present with sudden (radicular) back pain and paraplegia or tetraplegia (in variable degrees of severity), possibly associating neurogenic bladder [3],[4],[7],[8],[9],[10].

It can cause severe morbidity and mortality in 5.7% of cases [11]. Any age groups can be affected, but most cases are in their 60s or 70s, with a slight predominance (1.4:1) of the male gender [2], [7], [8], [11], [12], [13].

Most cases of acute SEDH have a multifactorial etiology, including old age, uncontrolled arterial hypertension, congenital and acquired coagulopathies, platelet dysfunction or even anti-platelet medication (aspirin, clopidogrel) [14], anticoagulants [15], [16], vascular malformations, tumors, spinal surgery or spinal procedures (lumbar punctures, epidural anesthesia) [10], [17], severe hepato-renal comorbidities (cirrhosis, liver transplantation, hemodialysis) [10], [17], even intense efforts implying sustained Valsalva maneuvers (intense efforts, lifting heavy weights [8], childbirth).

The paraclinical diagnosis is performed mainly with magnetic resonance imaging (MRI) [8].

The management of acute SEDH usually implies a surgical solution: decompressive hemilaminectomy and hematoma evacuation. Conservative treatment is reserved for subjects with minimal neurological impairment or with high risk for surgery [5], [6], [8], [13], [14], [19].

In rapid deterioration of the neurological status the most appropriate management is emergency decompressive hemilaminectomy and hematoma evacuation [17].

Case report This is a retrospective case study of an 80-year-old man who fell from body level on the ischiadic regions and suffered a C7 vertebral fracture, and a painless cervical SEDH at C3-Th2 vertebral levels.

Complex comorbidities were present in his medical history:

- Bilateral congenital foot deformity (fig. 1)
- Dilated cardiomyopathy with decompensated heart failure (NYHA IV then III, left ventricular systolic dysfunction with 40% ejection fraction)
- Permanent AF, chronically anticoagulated with a vitamin K antagonist (VKA, acenocumarol)
- Degenerative, large aortic stenosis

- Stage 2 arterial hypertension and episodes of orthostatic hypotension
- Dyslipidemia
- Cerebral microangiopathy, vertebro-basilar circulatory insufficiency
- Cervical herniated disc C3-C4 (MRI scan performed on 08.12.2017)
- Chronic alcoholism



Fig.1 Bilateral congenital foot deformity

The elderly felt dizzy a few days before, and on 26.11.2018 he had accidentally fallen (from body level) on his ischial tuberosities, without cranial impact with the surrounding objects (e.g. furniture). This episode was followed by a short loss of consciousness (without convulsions or sphincter relaxation). When he recovered consciousness he accused motor limbs deficit, mainly on the left side, and neurogenic bladder.

The patient was first admitted in the Cardiology Clinic of Fundeni Hospital (28.11.2017 – 08.12.2017). CT scan of the brain (29.11.2017) ruled out acute ischemic or hemorrhagic lesions and revealed cerebral microangiopathy (Fig. 2).

The patient was transferred in the neurosurgical spinal department of THEBA (on 08.12.2017, eleven days after the onset of symptoms), as C4 AIS-C tetraplegia. Spinal MRI scan revealed a posterior epidural hematoma in the cervico-thoracic region, compressing the cervical spinal cord, and C7 fracture (body and vertebral arch) - fig.3. MRI angiography revealed hypoplasia of the right vertebral artery.

The patient underwent a surgical decompressive intervention (on 13.12.2017): hemilaminectomy at the C4-Th2 levels, and evacuation of the hematoma (fig.4). The C7 vertebral fracture was conservatory treated (external immobilization with Philadelphia collar).

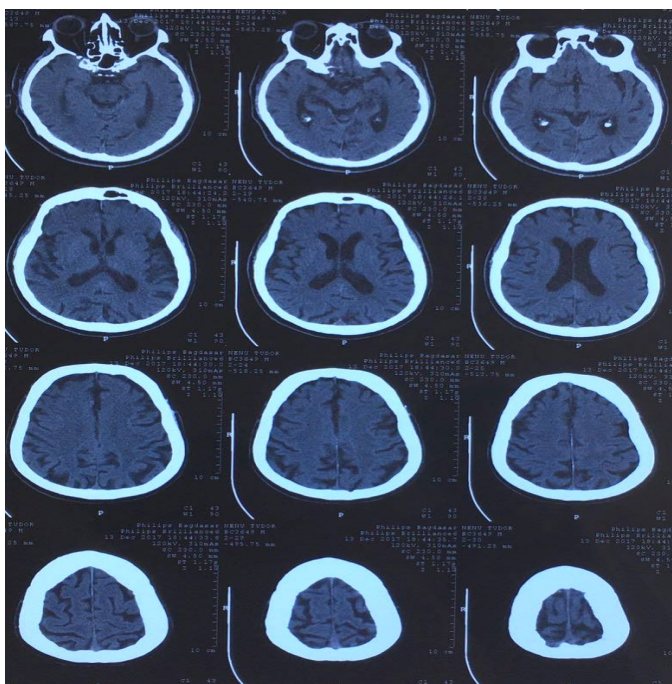


Fig.2 CT cerebral scan. Neither ischemic nor hemorrhagic acute / subacute lesions.

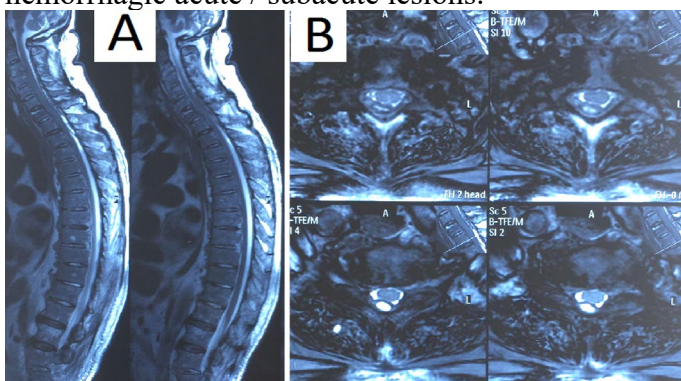


Fig.3 Pre-surgical MRI imagery.(A) Sagittal T2-weighted images: posterior epidural area of increased signal intensity (acute SEDH), compressing the spinal cord at C3-Th2 vertebral levels. (B) Transversal T2-weighted sections: right posterolateral SEDH.

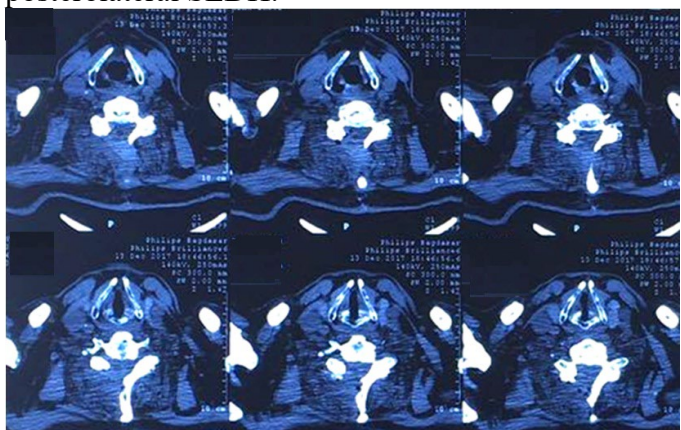


Fig.4 Post-surgical CT spine control, right hemilaminectomy.

He was transferred in the Neurorehabilitation Clinic on 29.01.2018 with severe post-surgical pain and depression. Clinical examination revealed normal vital signs: respiratory rate – 22/min, chronic AF with a 74 bpm heart rate, blood pressure – 140/90 mmHg, indwelling urinary catheter, post-operative wound dehiscence (healed per secundam, fig.6).

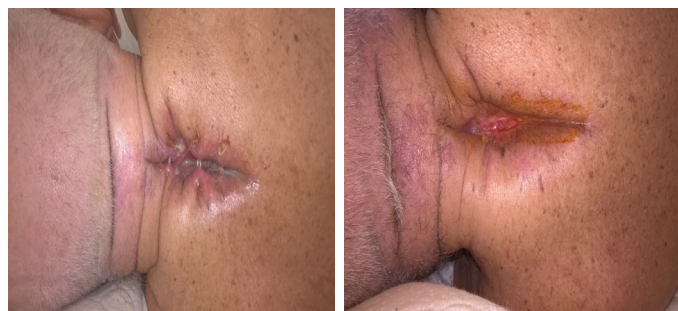


Fig.6 Post-operative wound, healed per secundam (left: at admission in the neurorehabilitation clinic on 31.01.2018; right: at discharge on 19.02.2018)

Neurological evaluation of the extent and severity of the spinal cord lesion, assessment of the rehabilitation targets and objectives, respectively the evolution were and quantified using the American Spinal Injury Association (ASIA) Impairment Scale (AIS).

Post-surgical neurological status at admission on rehabilitation was also C4 AIS-C tetraplegia: the global motor score was 50/100 (upper right limb 8/25, lower right limb 7/25; upper left limb 16/25; lower left limb 19/25): pin prick score was 100/112 (right side 49/56, respectively left side 51/56), and light touch score was 110 /112 (bilaterally 55/56).

Work-up. Usual blood tests revealed a slight anemia Hgb 9.7 g/dL and slight leucocytosis WBC $12.71 \times 10^3 /uL$ (on 13.12.2017), then normalized (on 30.01.2018). Blood glucose, renal and liver panels were within normal parameters (estimated creatinine clearance was 58.53 ml/min 1.73 m²). Coagulation tests were influenced by the use of LMWH, eventually replaced with apixaban.

Post-surgical MRI control revealed the absence of the SEDH, showing only the C7 vertebral fracture and centromedullary edema (extended about 3 cm, fig.5).

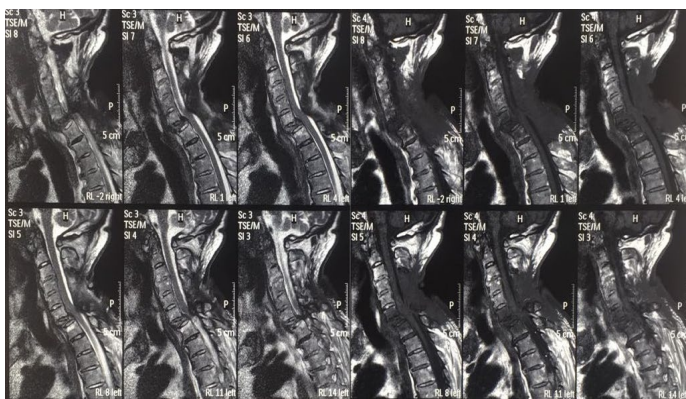


Fig.5 Post-surgical MRI control, sagittal T2-weighted images (05.02.2018): C7 compression vertebral fracture and centromedullary edema.

The rehabilitation program was individualized and adapted to the current clinical-biological status; the objectives and methods, physical and occupational therapy approaches were focused on improvement of ADL abilities, cardio-respiratory endurance, bladder training program and avoiding complications.

Medication consisted in a synergic association of vitamins, neurotrophic supplements, analgesics, antidepressants, urinary disinfectants, and antibiotics.

During admission in the surgical ward the vitamin K antagonist (VKA) acenocumarol was replaced with a low-molecular-weight heparin (LMWH) Nadroparine /Fraxiparine 6000 U, subcutaneously.

After a careful analysis of risks and benefits, considering CHADS2VASc = 6 as a high risk score for stroke in our patient with AF [20], and Has-Bled Score = 6 as a major high bleeding risk [21], we reconsidered the injectable anti-factor Xa (LMWH), and prescribed a novel / direct oral anticoagulant (apixabanum, 2 x 2.5 mg BID) for a “long cruise” anticoagulant therapy, to prevent AF-related stroke.

The subject had a favorable neurological evolution and was discharged as C7 AIS-D tetraplegia (global motor score 81/100: upper right limb 14/25, lower right limb 20/25 ; upper left limb 22/25; lower left limb 25/25); pin prick score was 103 (right side 49/56, respectively left side 54/56), and light touch score was 112 /112.

At discharge he was capable to walk a few steps, with lateral assistance and support. He was recommended a wheel chair and sterile pre-lubricated

Nelaton catheters, for intermittent urinary program of bladder rehabilitation (both items being freely provided by the National Insurance House).

Discussion: Spinal epidural hematoma (SEDH) is a rare but serious condition. Old age, hypertension, and anticoagulant therapy are the main risk factors for SEDH. Any age groups can be affected, but most cases were in their 60s or 70s, with a slight predominance of the masculine gender [12], [22].

Summing data published between 2004-2016 one identified about 700 cases [8]. A new personal literature search found another 35 papers describing 40 new case reports published in 2017, respectively 8 case reports published in 2018.

Messerer (2012) [10] proposed a practical classification of the spinal epidural hematomas according to the most probable etiology (whatever the associated factors), in six groups: spontaneous, secondary, iatrogenic, traumatic, recurrent, and idiopathic. Spontaneous hematoma is most often defined as a condition occurring in the absence of any traumatic event or iatrogenic procedure.

The present case has some clinical problems to be considered. Its main particularities consist in puzzled etiopathogenetic mechanisms and the difficulty to accurately indicate the chronological chain of events that generated the acute SEDH.

Literature data emphasize that etiology remains unclear in approximately 40% (up to 50-75%) cases of epidural hematoma, [3], [10], [17]. In our subject the exact underlying etiopathogenetic mechanism remained uncertain.

The patient was chronically anticoagulated with a VKA (acenocumarol), and daily ingested alcohol (alcohol consumption representing a factor incriminated in INR modification). Data referring to a retrospective estimation of how long the patient was efficiently anticoagulated (therapeutic time in range, TTR%) were not available.

Main risk factors incriminated as iatrogenic causes for a “spontaneous” epidural hematoma are: poorly managed arterial hypertension [14], [15], [16], anticoagulant therapy (warfarin [22], [23], acenocumarol or even novel anticoagulants such as rivaroxaban [16], [24], [25]), or in very rare situations, low-dose aspirin (75-100 mg daily) [8], [14], [26], [27] and/ or other antiplatelet medication (clopidogrel [26], [28]).

Spontaneous SEDH is a rare cause of spinal cord compression in adults, especially in the elderly [27]. During the acute neurosurgical episode, differential diagnosis was made with other epidural space-occupying lesions: traumatic disc hernia, other hematomas associated with congenital or acquired vascular lesions (arteriovenous spinal malformations), hemorrhagic tumors, hemophilia.

Typically an epidural hematoma is preceded / accompanied by local pain in the spine, [3], [9], [10]. Sometimes spontaneous cervical epidural hematoma is not always an obvious diagnosis [10], because in some elderly patients, back or neck pain might be absent [29]. In our case, the typical sequential evolutive paradigm (characterized by sudden onset with spinal radicular pain, followed by progressive neurological deficits due to spinal cord compression) was absent.

Accumulation of blood in the epidural space compressed the spinal cord and generated neurologic deficits. Added to the pre-existent congenital feet disability, these impairments predisposed to same-level fall and subsequently to vertebral fracture.

Falls are the leading mechanism of injury for patients older than age 65, elderly being predisposed to same-level fall (cause of injury, disability, and even death), resulting in cumulated chronic health problems [30], [31], [32]. Predisposing risk factors include: increasing age, gender (78% male predominance), chronic alcoholism, vertebral degeneration, polypragmasia, sensory deficits, comorbidities, disability of the lower extremities, foot problems, cognitive impairment [32], [33], [34]. Cervical spine fractures due to ground level fall are relatively common in the elderly [35].

The physiopathology of our patient's SEDH was probably mixed – iatrogenic and traumatic, consequence of the cervical spine fracture, after ground to floor low-intensity biomechanical impact.

Another particularity of this case report is represented by the type and level of the vertebral fracture. The spinal fracture type is explained by the biomechanical dynamic proprieties of the head. The vertebral injury incurred is determined predominantly by the force vector applied during impact and the intrinsic strength and anatomy of C2 vertebral body and its surrounding spinal elements [36]. In neutral, vertical position, the average weight of human head is around 4.5 to 5 kg at rest. Our patient had accidentally fallen on ischial tuberosities and then on

his back, without cranial impact with the surrounding objects (e.g. furniture). Typically, this type of level fall causes fractures of the atlanto-axial vertebral complex (C1-2), different from the current reported case (C7 compression vertebral fracture). As the head is bending forward, its weight increases from 13 kg (at 15°), to 20 kg (at 30°), 24,5 kg (at 45°), 30 kg (at 60°) [37]. This biomechanical variables might explain the structural wedge deformity of the C7 vertebral fracture compression in our patient (fig.5).

Another puzzling clinical aspect is represented by the short episode of loss of consciousness after falling, possibly owed to a cardiogenic syncope (chronic AF, hypodiastolic phenomena with global cerebral circulatory insufficiency) or to a transient ischemic attack.

Due to the severe cardiac pathology, the surgical decompressive intervention was postponed (for about two weeks); although surgery was performed during the early sub-acute phase, our patient had a good neurologic evolution during rehabilitation.

Despite the immediate good neurologic evolution during rehabilitation, the patient's functional prognosis might be poor, due to the advanced age and complex association of disabilities: the severe cardiovascular pathology and the disturbances of the neuro-myo-atrokinetic apparatus. Somatic **Body** functions and structural impairments at discharge consisted in: previous disabling congenital biomechanical feet condition, the poor hand prehension capacity, impeding a safe utilization of the walking frame or crutches, and incomplete recovery of force and coordination in the lower limbs. This health-related precarious condition had severe repercussions on the subject's **Activity** (related to tasks and basic activities of daily living) and **Participation**, affecting the outcome of rehabilitation, and his quality of life.

Our patient's vital prognosis depends on prevention of most common complications encountered in SCI (such as urinary tract infections, bowel problems, pressure sores, pulmonary infections) [38], in the context of the associated severe cardiac and neurological co-morbidities.

At discharge he was recommended apixabanum as a "long cruise" anticoagulant therapy, in order to provide secondary and tertiary prophylaxis of thromboembolic events (mainly cardio-embolic stroke), as well to diminish the hemorrhagic risk

(nevraxial recurrence or other general severe bleeding). Due to the favourable pharmacokinetic properties (simple BID administration in fixed doses, low inter and / or intra-subject variability, multiple elimination pathways, not requiring laboratory monitoring – all important aspects for boosting compliance [39], [40], [41]) the patient had a good psychological adherence to treatment.

Conclusions Clinicians should consider the remote risk of SEDH (even with atypical clinical presentation) in patients with AF and anticoagulant medication. The clinical case underlies the importance of an individualized therapeutic approach (after careful assessment of benefits against risks, efficacy and safety profile).

Despite a postponed decompressive intervention (imposed by the severe comorbidities), our patient neurologically improved without recurrence, following a complex neurorehabilitation program.

Conflict of interest: Authors have no conflicts of interest to declare.

The paper has the approval of the Ethical Committee of the Hospital.

Written consent was obtained from the patient and next of kin.

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