

Challenges and pitfalls in the forensic assessment of posttraumatic extracerebral fluid collections

Danil Adam^{1,*}

Abstract: The purpose of this article is to analyze the particularities of these collections and to discuss their medical-legal consequences. Different types of collections were presented together as they have similar prognosis and management.

A retrospective analysis of data collected from 46 patients with posttraumatic extra cerebral fluid collections is presented. Asymptomatic patients or those with mild symptoms were conservatively treated (n=27). Surgical evacuation was performed in cases of intracranial hypertension or neurological worsening (n=19). Clinical results are presented using the Glasgow Outcome Scale (GOS). Regarding the group of patients treated conservatively, good results were obtained (GOS score 4 or 5) in 81.4 % of the cases. Regarding the surgical treated group of patients, good results were obtained in 73.6% of the cases. The mortality rate was 18.5%, and 26.3% respectively. Conclusions: posttraumatic extra cerebral fluid collections appear more often in elder patients, often secondary to minor trauma (like falls), but can lead to difficulties in analyzing the case from a medical-legal point of view. The majority of patients whose CT-scans show extra cerebral fluid collections are asymptomatic and the fluid collections are eventually spontaneously resolved. Mortality is in most cases caused by associated cerebral damage or systemic complications.

Key Words: hygroma, hydroma, serous meningitis, posttraumatic extra cerebral fluid collections.

Posttraumatic intracranial fluid collections have rarely been reported in neurosurgical literature before modern imaging techniques emerged.

However with the implementation of modern imagery, their incidence increased significantly. They represent a heterogeneous group of fluid collections that can be localized in the subdural space (hygroma, hydroma) or in the subarachnoid space (serous meningitis). Subdural collections of fluid other than blood are referred to as subdural hygromas or hydromas. The term subdural hydroma was first reported by Mayo [1] in 1894, but subsequently evolved into subdural hygroma or subdural effusion.

Described initially by Payr [2] in 1916, hygroma was separated as a nosologic entity under this name by

Dandy [3] in 1932. Hygroma represents a fluid collection circumscribed in the subdural space, containing a xanthochrome liquid that is encapsulated by a neo-membrane [4]. Hydroma is defined by the presence of a clear liquid and the absence of a membrane. Serous meningitis is defined as a circumscribed or diffuse accumulation of fluid located in the subarachnoid space. It is often confused with external hydrocephalus. Between serous meningitis and hygroma/ hydroma there is only a difference of topography.

The purpose of this article is to analyze the particularities of these collections and to discuss their medical-legal consequences. Different types of collections were presented together as they have similar prognosis and management.

1) Associate Professor, Department of Neurosurgery, "Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

* Corresponding author: Emergency Clinical "St. Pantelimon" Hospital, Șos. Pantelimon 240, Sect.2, Bucharest, Romania, E-mail: adam_danil@yahoo.com

MATERIALS AND METHODS

All patients with posttraumatic extra-cerebral fluid collections diagnosed and treated in our institution were included in this study.

The following clinical information was collected: age, sex, GOS score at admission, mechanism of injury, signs and symptoms and comorbidities.

Investigation protocol included a CT-scan at admission, which was repeated during hospitalization if the clinical symptoms advanced, and also in postoperative period. Were taken into consideration: size of the fluid collection, midline shift and coexisting brain injuries.

Treatment for fluid collections was conservative for asymptomatic patients or for those with mild symptoms, including analgesics and hydration and was administered in the neurosurgery ward or intensive care unit, depending on associated diseases.

Surgery was performed in cases of intracranial hypertension associated with altered consciousness, under general anesthesia by oro-tracheal intubation. The skull opening was performed with a trephine of 4.5 centimeters in diameter. After a star-shape opening of the dura mater and evacuation of the fluid under pressure, spontaneous and by suction, the dura mater was partially closed, the bone flap was restored and an epidural drain was left for 24-48 hours.

The patient's clinical outcome was recorded on GOS scale 3 months later, when he came at a follow-up visit.

RESULTS

Forty-six patients with posttraumatic extra-cerebral fluid collections were diagnosed and treated over a 5- year period. There were 36 males (78.2%) and 10 females (21.8%), aged between 16 and 94 years old, with a mean of 68 years. In most cases the traumatic event was either a fall (22 cases, 47.8%), often associated with older age, or a car accident (7 cases, 15.2%). In a single case the trauma was associated with a blood disorder secondary to thrombocytopenia For details see Table 1.

Irrespective of subdural or subarachnoid topography of fluid, the patients had similar clinical symptoms. The patients were treated either without (Group A, 27 cases) or with surgery (Group B, 19 cases). There were no differences in treatment modalities regarding the topography of fluid.

CT-scan revealed a simple fluid collection, not associated with brain injuries, in 6 cases, whilst the rest of 40 cases had additional traumatic injuries, detailed in Table 2. Sample CT scans of the traumatic injuries are presented in Figures 1, 2.

The fluid collection was unilateral in 21 cases and bilateral in 25 and it appeared at variable time after the traumatic event, from 24 hours to 50 days. In patients without surgical treatment the size of the fluid collection

in the axial plane was between 4 and 14 mm and midline shift was 0 mm (7 patients), 3 mm (4 patients), 4 mm (7 patients), and 7 mm (1 patient); 13 cases had bilateral fluid collection and 14 - unilateral. In patients with surgical treatment (19 cases) the size of the fluid collection in the axial plane was between 6 and 18 mm; in 12 cases the fluid collection was bilateral. The decision

Table 1. Patients characteristics.

Characteristic	No	%	
Mechanism of injury			
fall from the same level	22	47.8	
fall from another level	4	8.7	
car accident	7	15.2	
traumatic brain injury secondary to seizures	2	4.3	
unknown	11	23.9	
Signs and symptoms at admission			
headache and dizziness	20	43.4	
psychomotor agitation	10	21.7	
loss of consciousness for short period of time	10	21.7	
hemiparesis	8	17.3	
dysarthria	7	15.2	
temporo-spatial disorientation	7	15.2	
balance disorders	6	13	
ear bleeding	6	13	
GCS	15-13	19	41.3
	12-19	7	15.2
	8-7	4	8.7
	unspecified	16	34.7
Comorbidities			
arterial hypertension	16	34.7	
chronic alcoholism and smoking	15	32.6	
chronic ischemic heart disease	7	15.2	
CPOD	5	10.8	
ischemic stroke	5	10.8	
CAS	3	6.5	
diabetes	4	8.7	
cirrhosis	4	8.4	
seizures	2	4.3	
hemorrhagic stroke	1	2.2	

GCS-Glasgow Coma Scale, CPOD- Chronic Pulmonary Obstructive Disease, CAS-Cerebral Athero-Sclerosis

Table 2. Brain injuries associated with fluid collections.

Associated lesions	No.	%
hemorrhagic contusions	25	54.3
skull fracture	12	26
cerebral atrophy	11	23.9
epidural hematoma	2	4.3
intraparenchymatous hematoma	4	8.7
subdural hematoma		
acute	3	6.5
chronic	9	19.6

Table 3. Outcome according to GOS (Glasgow Outcome Scale).

GOS characteristics	Group A		Group B	
	No.	%	No.	%
Good recovery	19	70.3	12	63.1
Moderate disability	3	11.1	2	10.5
Severe disability	-		-	
Vegetative state	-		-	
Death	5	18.5	5	26.3

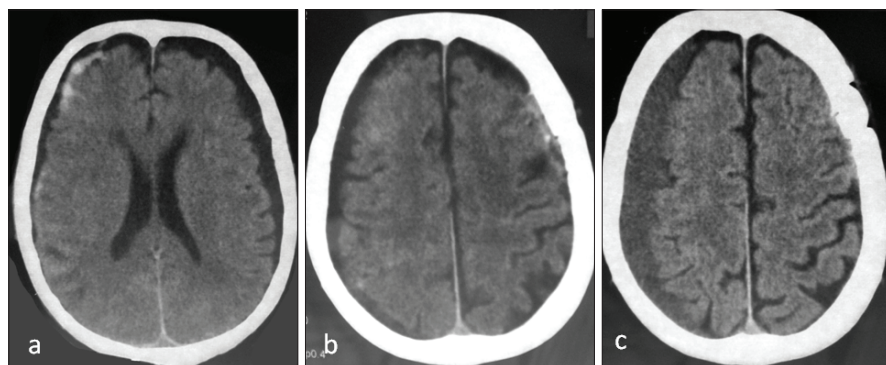


Figure 1. Male, 74, with head trauma caused by accidental 2 meter fall on the stairs, with bilateral hygroma right > left (a). A week after the evacuation of the hygroma in the left side a growth of extra cerebral collection in the right side is observed (b); after 3 weeks it transformed into a chronic subdural hematoma 18 mm thick; the midline shifted 4 mm towards the left side (c). Note that the hygroma from the right side was associated with hemorrhagic cortical contusions (complex hygroma).

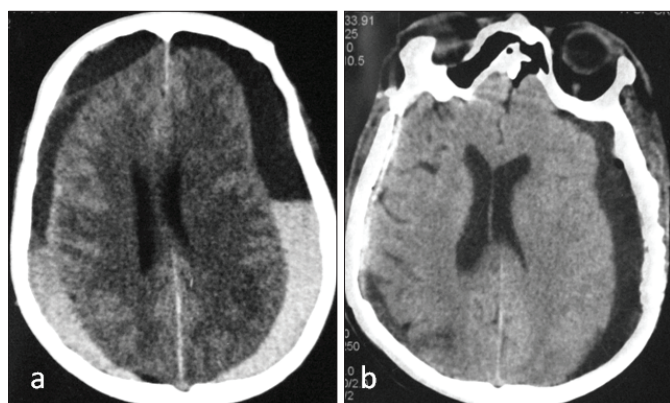


Figure 2. Male, 81, with head trauma caused by fall from the same level. Cerebral CT scan in axial plane highlights: bilateral subdural hematoma (a). Two weeks after surgery it is observed that the collection on the right side disappeared and that a hygroma developed on the left side (b).

to operate was made when neurological deterioration occurred. Among these, in 12 patients the fluid evacuated after dura incision was citrine, in 2 cases it was clear and in 5 cases the color was not mentioned. In 7 patients was identified vascularized parietal subdural membrane, having a variable thickness, without the presence of a visceral membrane.

The clinical outcome of the patients with intracranial fluid collections is presented in Table 3.

Ten patients died, of which five were in Group A the causes of death being liver failure associated with decompensated diabetes (1 case), exacerbation of chronic kidney disease (1 case), bronchopneumonia (1 case), and severe traumatic brain injury (2 cases), and five in Group B, the causes of death being pulmonary complications (2 cases) ischemic stroke (one case) and hygroma associated with a subdural hematoma (2 cases).

DISCUSSIONS

Subdural hygroma is often considered as an epiphenomenon of a head injury, appearing as early

as a few hours after the traumatic event. Traumatic hygroma is often bilateral, located at the top of the Head if the patient is in supine position, suggesting that gravity and cranial posture are involved in its development.

If a subdural hygroma develops secondary to acute trauma, it can mimic an acute subdural hematoma, often requiring surgical intervention for its evacuation, as were the cases included in Group B. The medical-legal consequences of these lesions are similar to those of any subdural hemorrhage and, subsequently, the assessment of their penal gravity should be similar [4].

The location of the subdural hygroma cannot however predict the location of injury as its development is influenced by posture and gravitation. Lee for example, by studying 47 patients with traumatic subdural hygroma found that its development and location is dependent upon the symmetry of the cranium (being more frequently bilateral in patients with symmetrical cranium and more frequently unilateral in those with an asymmetrical cranium), type of imaging examination (were more oblique in MRI compared to CT, most likely caused by the prolonged time of examination in MRI), gravity, and posture (traumatic subdural hygroma develops in the area of least pressure in the cranium as a lesion of ex-vacuo) [5]. Therefore, by identifying a subdural traumatic hygroma the legal-medicine physician cannot determine the location, direction, and intensity of the traumatic force vector causing it.

Traumatic subdural hygroma may cause chronic subdural hematoma, up to six years after the traumatic event. Park et al described, on a series of 145 patients, 13 cases in which a chronic subdural hematoma developed on the site of a previously known traumatic subdural hygroma [6]. The authors could not find specific elements which to suggest the possibility of the development of the chronic subdural hematoma [6]. Therefore its presence should be noted and, if a chronic subdural hematoma is discovered later, on the same site as the initial hygroma, the causal chain should not be broken, even if there is a symptom free period of up to six years [6].

Although the fluid collections were identified at almost any age, most cases involved patients ages over 60. The youngest patient in our series was 16 years old the fluid collection being caused by a car accident. Mayo reported the case of a 12 year-old boy who, secondary to head trauma, developed a fluid collection overlying the left cerebral hemisphere [1]. This late onset, associated with the possible presence of various types of age related neurological disorders, and the fact that

reoccurrence can be identified after a long symptom-free period[7-10], suggest the need for repeated reevaluations of these patients. Also, if any acute onset mental decay is suspected, the patients should be carefully checked for possible reoccurrences or the development of a secondary chronic subdural hematoma. Hygroma is known to be associated with physical abuse, especially in children; therefore its presence in this population group should lead the physician toward an in depth search of other signs of abuse[12-13].

In our series the cause of death was more likely caused by other underlying conditions or concomitant head trauma than to the posttraumatic fluid collection

per se. However its presence may increase the severity of cerebral edema or other head lesions and may be a favoring circumstance for the death of the patient.

In conclusion, posttraumatic extra cerebral fluid collections appear more often in elder patients, often secondary to minor trauma (like falls), but can lead to difficulties in analyzing the case from a medical-legal point of view. The majority of patients whose CT-scans show extra cerebral fluid collections are asymptomatic and the fluid collections are eventually spontaneously resolved. Mortality is in most cases caused by associated cerebral damage or systemic complications.

References

1. Mayo CH. A brain cyst: The results injury causing aphasia, hemiplegia, etc. Evacuation; complete recovery. NY Med J. 1894; 59:434,.
2. Payr E. Meningitis serosa bei und nach Schadel Verletzungen (traumatica). Med Klin. 1916; 12: 841-846.
3. Dandy WE. Chronic subdural hydroma and serous meningitis (pachymeningitis serosa; localise external hydrocephalus). In: Lewis D (ed): Practice of Surgery, Hagerstom, Maryland W. F. Prior 1932, p. 306-314.
4. Granacher RP. Traumatic brain injury : methods for clinical and forensic neuropsychiatric assessment. Boca Raton: CRC Press/Taylor & Francis Group, 2008.
5. Lee KS, Bae WK, Yoon SM, Bae HG, Yun IG. Location of the traumatic subdural hygroma: role of gravity and cranial morphology. Brain Injury. 2000; 14: 355-61.
6. Park CK, Choi KH, Kim MC, Kang JK, Choi CR. Spontaneous evolution of posttraumatic subdural hygroma into chronic subdural haematoma. Acta neurochirurgica. 1994; 127: 41-7
7. Ricciardelli EJ, Richardson MA. Cervicofacial cystic hygroma. Patterns of recurrence and management of the difficult case. Archives of otolaryngology--head & neck surgery. 1991; 117: 546.
8. Isenberg SF. Cystic hygroma: recurrence in an adult 34 years later. American journal of otolaryngology. 1995; 16: 347
9. Charabi B. Cystic hygroma of the head and neck-a long-term follow-up of 44 cases. Acta Oto-Laryngologica. 2000; 120: 248-50
10. Arseni C, Oprescu I. Neurotraumatologie, Editura Didactică si Pedagogică, Bucuresti 1983, p. 163.
11. Hymel KP, Jenny C and Block RW. Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. Child maltreatment. 2002; 7: 329-48.
12. Kenny MC. Child abuse reporting: Teachers' perceived deterrents. Child abuse & neglect. 2001; 25: 81-92
13. Van Haeringen AR, Dadds M, Armstrong KL. The child abuse lottery--will the doctor suspect and report? Physician attitudes towards and reporting of suspected child abuse and neglect. Child abuse & neglect. 1998; 22: 159.