Histopathology and Forensic Diagnostics Difficulties in a Patient with Multiple Affections and Double Cranio-Cerebral Injuries

Cris Precup, Mircea Ifrim, Ovidiu Bulzan, Csongor Toth, Gyori Zsolt, Hategan Ovidiu
Anatomy Department, Faculty of Medicine Pharmacy and Dental Medicine
“Vasile Goldis” Western University of Arad, Romania

REZUMAT

Dificultăți de diagnostic histopatologic și medico-legal la un pacient cu multiple comorbidități și traumatisme cranio-cerebrale multiple - prezentare de caz

Această lucrare este o prezentare de caz. Cazul a fost ales deoarece a prezentat mai multe probleme referitoare la diagnosticul de certitudine al cauzei decesului și, prin urmare, stabilirea mecanismului tanato-generator. Pacienta este de sex feminin dintr-un mediu rural care a decedat după internare (03 martie 2013), în Spitalul Clinic de Urgență din Arad, Secția de Neurologie. Pacienta este internată prin Unitatea de Primire Urgențe în urma unui traumatism cranio-cerebral în regiunea temporo-parietală stânga, cu insuficientă respiratorie ventilată mecanic și comă gradul II. Dificultățile privind diagnosticul și stabilirea legăturilor de cauzalitate apar atunci când coroborând datele clinice mai sus menționate cu antecedentele personale patologice: hipertensiune arterială, diabet zaharat de tip II, cardiopatie ischemică cronică, fibrilație atrială cronică. După analiza informațiilor din arhiva Serviciului Județean de Medicină Legală Arad devine clar că persoana a mai fost victimă unei agresiuni, în urma căreia, de asemenea, a rezultat un traumatism cranio-cerebral în regiunea temporoparietală stângă (februarie 2009), în urma unui traumatism produs prin lovitură de topor la nivelul extremității cefalice. În urma traumatismului care a avut loc în februarie 2009 pacienta a suferit un traumatism cranio-cerebral pierderea temporară a conștienței și a fost internată pentru investigații și tratament de specialitate. După deces, s-a dispus efectuarea necropsiei medico-legale pentru lămurirea tuturor detaliilor macroscopice și microscopice legate de caz. Aspectele dificile în stabilirea cauzei de deces constau în existența a două traumatisme cranio-cerebrale care au avut loc la un interval de timp de patru ani și comorbiditățile asociate.

Cuvinte cheie: traumatism, autopsie, leziuni cerebrale

Corresponding author: Cris Precup – Senior Lecturer, PhD MD
Department of Anatomy and Embryology, Faculty of Medicine, Pharmacy and Dental Medicine
“Vasile Goldis” Western University, Arad, Romania
E-mail: precupcris@yahoo.com
ABSTRACT
This paper is a case presentation. This case was chosen because it presented several problems with indicating, with certainty, the cause of death and therefore establishing the tanato-generating mechanism. The patient is a female from the rural environment who dies after a hospitalization (03 March 2013) in the Clinical Emergency Hospital in Arad, Department of Neurology. The patient is admitted through the emergency unit due to a brain injury in the left parietal-temporal region, assisted respiratory failure and second degree coma. Diagnostic and causation difficulties occur when clinical data mentioned above is corroborated with previous medical history: type II diabetes hypertension, chronic heart disease, chronic atrial fibrillation. After analyzing the information in the archive of the Forensic medicine service in Arad it becomes clear that the person was the victim of an assault that also resulted in a head trauma in the left parietal-temporal region (February 2009), after being hit with an axe in the cephalic extremity. After the incident in 2009 the patient suffered head trauma with temporary loss of consciousness and was admitted for investigation and specialist treatment. Following the death, a necropsy was performed in order to clarify all the macroscopic and microscopic details. The difficulty of the case rests is the existence of two head traumas that have occurred 4 years apart and the associated pathology.

Key words: trauma, autopsy, brain injury

INTRODUCTION
This paper is a case presentation. This case was chosen because it presented several problems with indicating, with certainty, the cause of death and therefore establishing the tanato-generating mechanism. The patient is a female from the rural environment who dies after a hospitalization (03 March 2013) in the Clinical Emergency Hospital in Arad, Department of Neurology. The patient is admitted through the emergency unit due to a cranio-cerebral trauma in the left parietal-temporal region, assisted respiratory failure and second degree coma.

CASE REPORT
Under the Ordinance issued by Arad City Police, a necropsy was conducted on G. V, age 67, a resident of the village of Fiscut (rural), Arad County. The forensic objectives established in the ordinance were: whether the death was violent, if the body shows signs of violence and what were the causes of death.

The team has asked about the history and circumstances of death. Resolution of making the autopsy shows that on March 2013, the patient died in the Arad County Hospital, Department of Neurology. The general clinical observation sheet from the Arad Emergency County Hospital, gives information on the reasons for hospitalization as well as information regarding the period of hospitalization, between 03/08/2013 – 03/12/2013.

The patient was brought to the hospital by ambulance and from the observation sheet which was brought from the receptions unit - Emergency Hospital Arad County, we see why the patient was brought to hospital, II degree coma, with endotracheal intubation. From the medical history provided by her caregivers, the patient suffered from chronic ischemic heart disease, hypertension, chronic atrial fibrillation and type II diabetes.

Corroborating the information provided by the medical team and the forensic investigation team we realize that the patient was found in the yard, at home, in a coma, and was sent by SMURD (emergency rescue service based in Romania) to Timisoara County Hospital for diagnosis and specialized treatment due to the deterioration of neuro-logical status and the outcome of C.T. brain scan that shows a hypodense lesion in the left parietal region. SMURD reaches Timisoara with the patient with a referral diagnosis of mixed cerebral coma grade III. The diagnosis does not change for 72 hours. Due to the socio-economic conditions of the patient’s tutors, the patient and the medical team are transferred from Timisoara County Hospital to Arad County Hospital. The main diagnosis at the time of discharge is cardiac arrest, mixed cerebral coma, repeated ischemic stroke with sequelae in the basal nuclei, sequelae of head trauma in the left temporoparietal region, acute respiratory failure requiring ventilatory support, type II diabetes, exacerbated chronic renal failure, chronic atrial fibrillation, ischemic heart disease and chronic hypertension.
it can be seen the patient had a body with multiple conditions.

After analyzing the information from the archive of Arad County Department of Forensic Medicine it was clear that our patient was the victim of an assault that also resulted in a head trauma in the left parietal-temporal region (February 2009) after being hit with an axe in the cephalic extremity. The patient had, once more, suffered a head trauma with temporary loss of consciousness, and was admitted for investigation and specialist treatment, and she survived. This type of injury was studied by Cychowska M in 2013 [1,2]

The next stage is the external examination of the corpse by the rigorous autopsy protocol. [3,4,5,6,8]

The corpse submitted for autopsy belongs to a female, aged 66, of medium height and with the muscular and adipose tissues proportionally represented. The indices of the actual death are present through the cadaveric lividity of a shade of purple placed on the dorsal declive of the trunk and limbs, which do not turn pale when applying pressure (imbibitions stage) and a cadaveric rigidity in the final stage. There are no traumatic external injuries detected. The body shows signs of medical treatment: wound points in areas of choice for injection treatment (Right subclavicular region - three plagues point and on the elbow fold). A pearly white xifo-subombilical scar measuring 25 cm and a leg oedema are noticed.

The internal examination of the body, at the cephalic extremity, reveals an area of 8/8 cm located in the left temporo-parietal region gray-white coloration (scar). The left temporo-parietal region of the neurocranium shows a lack of bone in an area of 4,5 / 4,5 cm (slightly irregular white edges). Left parietal region of the duramater presents additional to the underlying lack of bone described above a whitish gray scar of about 4.4 cm (the connective tissue is thickened and there is no bone covering).

In the temporo-parietal region on the left side, on a surface measuring approximately 8sqcm, the leptomeningeal is thickened with a yellowish brown color, without sheen, without substance (with pearly white edges) measuring 4/4 cm in the central area. (Fig. 1) The blood vessel at the base of the brain have moderately thickened walls with a permeable lumen. In the left cerebral hemisphere [affecting cortex (with lack of substance on a surface area of 4.4 cm) and underlying white matter], the temporo-parietal region, the circumvolutions and the grooves are missing and the nerve tissue has a yellowish brown color with a low, brittle consistency. We analyzed the possible causes of this type of injuries by surgical point of view. [9,10]

On the cross-section, the normal architectural design is missing, the nerve tissue looks like a yellowish grey semi-solid mass. (Fig. 2 and 3) Right cerebral hemisphere- with the convolutions widened and the grooves between them erased, with a low, slightly brittle consistency and with the normal architectural design faded. On the cross-section the grey matter is faintly delimited from the white matter which has a wet appearance with a matted gloss. With multiple reddish dots which disappear when are being scraped with a knife. In the grey basal nuclei and the brainstem the nerve tissue has a low and brittle consistency with the erasure of the normal architectural design. The diencephalon and cerebellum have a clear architectural design.

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**Figure 1.** Dissection of the head (external view)

**Figure 2.** Examination of the parietal and temporal bone (internal view)
subarachnoid space and cerebral ventricles contain a reddish fluid in a very small amount and the choroid plexus are reddish.

When internally examining the oropharyngeal region we found the lips with a reddish brown lining and the tongue with a gray velvet like lining on seriate sections of muscle mass without any traumatic injuries can be observed. The ovoid tonsils are covered by reddish lining. The subcutaneous tissue in the cervical region does not have any traumatic injuries. The muscles of the neck do not have any traumatic injuries. Both on the surface and in the cross-section, the thyroid gland have a reddish purple color. The hyoid bone and the laryngeal cartilages are intact. The larynx exhibits a whitish gray lining. The pharynx exhibits a gray, wet and shiny lining. The cervical spine has no traumatic injuries. The soft parts of the right hemithorax of the upper third part of the anterior region underlying the point wounds of the injection treatment described in the external examination under “sings of medical treatment” exhibit a moderate dark hemorrhagic infiltration of 4 sq cm surface. The pleuras are thickened, fibrosed, matte with the gloss weakened. The pleural cavities contain a minimum amount of sero-citrine liquid. The trachea and the large bronchi have unreached walls, exhibit a reddish gray lining and an open lumen. The lungs occupy 2/3 of the thoracic cavity, are of a brownish gray color, with the antracotic design clear, they have a soft consistency, a diminished elasticity, with small, difurred crepitation. On the cross-section they have an inhomogeneous brown-gray color and a spontaneous large blood drain. The pericardium in slim with the visceral foil wet, glossy and transparent. The pericardial cavity exhibits a minimum quantity of serocitrine liquid. The heart is firm in consistency and measures 12/11/6 cm. The heart cavities contain liquid blood and reddish-black, nonadherent clots. The valves are slightly thickened and transparent. The endocardium exhibits a reddish brown color with a low consistency and diminished elasticity. When observing seriate sections that were made along its thickness, we are presented with an inhomogeneous surface and small white areas on a reddish brown background; the thickness of the myocardium at the VS level is 1,7 cm. the coronary arteries present slightly thickened walls, with diminished elasticity and a permeable lumen. The aorta has walls that have not been breached, with diminished elasticity and the heart is yellowish with rare hard, white plaques. The pulmonary arteries are free, the heart is smooth, of a yellowish white.

The abdominal wall does not exhibit any traumatic injuries. The peritoneum is thickened, exhibiting multiple fibrous adhesions through the supramesocolic floor; the peritoneal cavity contains a minimum amount of serocitrine fluid. The stomach had walls that have not been breached, a gray with blackish spots lining (easier to spot in the area of the lesser curvature and the pyloric channel) with pronounced folds and contains gray-green liquid. The intestines exhibit walls that have not been breached, a white and gray lining and content appropriate to the segment.

The liver weighs 2200 grams, the visceral peri-
toneum and the capsule are smooth and transparent with a dull gloss, the surface is smooth, yellowish brown, with a hard consistency and slightly brittle. The cross-section is yellow, with a slightly grainy aspect and a small quantity of reddish brown blood draining.

The cystic fossa of the lower face of the liver is free (gall-bladder – absent), pearly white material is present (postcolecistectomy scar); the left and right hepatic ducts and the hepato-biliary duct are permeable.

The spleen has a low, brittle consistency, weighs 220 grams with a transparent, smooth and highly glossy capsule. On the cross-section it is purple with white dots and streaks. The pancreas is yellowish gray with a high consistency. On the cross-section we are presented with a glandular lobular design with whitish glandular lobular and interlobular reddish spaces.

The kidneys exhibit diminished elasticity, a transparent capsule, with diminished shine, which are hard to peel off and with the surface that has been decapsulated exhibiting irregularities. On the cross-section, the cortical region is grayish brown with red striations and the medular region is reddish brown and the cortical-medular contrast is diminished. The abdominal aorta and the inferior vena cava have walls that have not been breached and a permeable lumen. The bladder exhibited gray-white lining is devoid of content. The internal genitalia have no traumatic injuries. The pelvis bone exhibits no traumatic injuries.

The external examination of the perineum, the external genital organs and the anal region show that there are no traumatic injuries present. On a careful examination of the upper and lower limbs no traumatic injuries are present except for those caused during the treatment.

After macroscopic examination the following diagnoses are established: Advanced autolysis with decay of variable intensity; scarring on the soft sides of the left parieto-temporal region; lack of bone substance in the left parieto-temporal region; scarring on the duramater in the left parieto-temporal region; lack (old) of substance in the left left parieto-temporal region in the leptomeninges; minimal subarachnoid hemorrhage; left temporo-parietal cerebral softening; old lack of substance in the cerebral cortex in the left temporo-parietal region; moderate cerebral and coronary atherosclerosis; miocardofibrosis; bilateral pahipleuritis; emphysema, liver dystrophy; renal dystrophy, old cholecystectomy, chronic pancreatitis and reactive spleen.

For diagnostic certainty tissue sampling is performed for the histopathological examination. The material taken for histopathological examination included fragments of: cerebral hemisphere (left parieto-occipital), brainstem, thalamus and basal nuclei, lung, heart, liver and kidneys.[8] The fragments were prepared as per standard protocol in a solution containing 10% formaldehyde, paraffin inclusion and application of hematoxylin-eosin staining.[5]

The result of microscopic examination revealed the following:

In the fragment of the cerebral hemisphere extravasated red blood cells in the subarachnoid space were identified as well as the widening pericellular and perivascular spaces with dissecting the brain substance, in the lumen of cerebral vessels more red blood cells and a few leukocytes are seen as well as small extravased perivascular blood cells in the cerebral nerve substance, areas with irregular edges (made out of a fibrillar material produced by an eosinophil together with fibroblasts, fibroblasts, macrophages, and glial cells) where the normal architectural design is missing, the nerve substance looks irregular with an areolar appearance, the neural silhouettes can no longer be identified; a small amount of amorphic material, with a basophilic tint, some red blood cells many glial cells and some macrophages and lymphocytes in those areas and moderate diffuse glial hyperplasia.

In the brainstem fragment, pericellular and perivascular visually empty spaces were identified, with the dissection of nerve substance; in the lumen of the vessels red blood cells and some white blood cells can be observed, small scattered haematic extravasations perivascular in the cerebral nerve substance, neurons with finely granulated cytoplasm and with difficult to see nuclei, in certain areas, and clusters of glial hyperplasia.

In the thalamus and basal nucleus fragments pericellular and perivascular visually empty spaces were identified, with the dissection of nerve substance; in the lumen vessels a large number of red blood cells and a few white blood cells can be seen, small scattered haematic extravasations perivascular in the cerebral nerve substance, neurons with finely granulated cytoplasm and with difficult to see nuclei, in certain areas, and clusters of glial hyperplasia.

In the lung fragment, a thickened visceral pleura
can be seen, presenting parenchymal fibrosis with intensely eosinophilic interalveolar septa, thinned, some being fragmented, with visibly large void spaces, with polygonal outline and enlarged interalveolar septa; in the enlarged lumen of the blood capillaries a large number of red blood cells and a few white blood cells can be seen; we can also see areas where we can observe the presence of a eosinophilic fibrillar material accompanied by fibrocytes, fibroblasts, and some macrophages (intracytoplasmic dark brown pigment) and lymphocytes, areas where intraalveolar eosinophilic amorphous material contains macrophages, lymphocytes, epithelial cells and some granulocytes, there are also bronchi and bronchioles with leukocyte infiltration in the walls; the lumen of the bronchiolar is an amorphous eosinophilic material with lymphocytes, macrophages, granulocytes and some epithelial cells present. (Fig. 4)

In the heart muscle fragment was observed small haematic extravased at the submesotelial connective tissue level of the visceral serous pericardium. At the level of myocardium it was found a large intermyocytes space, in the lumenum of the heart blood vessels it was found lots of hemata and a few leucocytes. At the subendocardial region it was found an eosinofil fibrilar tissue (with fibrocytes, fibroblasts and a few monocytes) and myocytes with small eosinofil variation of the sarcoplasma, faded striations and in some areas with hardly observable nucleus.

In the liver fragment it was observed a widening of the sinusoid capillary lumenum at the level of the Porta vein branches and at the centro-lobular veins. The lumenum contains lots of hemata and a few leucocytes small haematic extravased at the Porta branches and hepatic lobules level, hepatocytes with vacuolar cytoplasm, poorly outlined cell membrane and well structured nucleus

In the kidney fragment was identified a space enlargement of renal filtration region, small hematic extravased in the renal filtration spaces with an enlargement of the interstitial spaces, blood vessels with a lot of hemata and a few leucocytes and granulocytes, areas with renal glomeruls which have an intensely eosinophilic fibrillar appearance, diffuse urinary tubular epithelium cells with intensely eosinophilic cytoplasm finely granular appetite - few hemata and some epithelial cells. (Fig. 5)


DISCUSSIONS

In this case, from the patient’s medical history we
keep in mind that she was the victim of an aggression from a known individual, which happened in the evening of 02.02.2009, in Fiscut, when she was hit with an ax in the cephalic extremity. She suffers a cranio- cerebral injury with a temporary loss of consciousness and is admitted in the emergency room for medical investigations and specialized treatment.

After a rigorous anamnesis we succeeded to unlock this forensic problem. There was a criminal trial in the first aggression case; the tutors wanted the actual hospital costs to be paid by the aggressor. For this purpose they asked if there is or not a causal link between the first case and the second.

We concluded, according to the identified information that four years later, (2013) she suffers a decompensation episode due to type II diabetes, after which she is admitted through the ambulance services. The 67 year old patient, who was admitted to the Diabetes and Nutrition ward, is showing a deterioration of her neurological condition, which is why a CT scan is performed which shows a hypodense lesion left parietal region.

The patient, having acute TCC sequelae due to aggression, insulin-dependent type II diabetes, chronic renal failure and atrial fibrillation is immediately admitted to the Diabetes ward in 04.03.2013, in a I degree comatose state and metabolic decompensation. Taking into consideration the CT findings with the disabling ischemic changes and suspected brain tumor she is sent to Arad- ATI – Neurosurgery where she is sent to ATI – diabetes ward, Arad, she is admitted through the Department of Neurology although sent to ATI – diabetes ward.

Murty OP in 2009 concluded also that type II diabetes raise the diagnostic difficulties to give a clear forensic diagnosis. [7,8]

CONCLUSIONS

Death was due to bronchopneumonia and acute interstitial nephritis, subsequent to prolonged metabolic diabetic coma (with diabetic hyperosmolar syndrome and diabetic keton-acidosis). There is a high degree of difficulty in terms of causal link, between the injury and the death causing mechanism in patients, with multiple affections.

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Figure 5. Microscopic view of the kidney fragment, HE stain, X20, amorphous material contains macrophages, and few lymphocytes glomerulosclerosis and granular distrophy of urinary tubes, a

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