Authors: **Tabian Daniel[1], Diac Madalina[1], Damian Simona Irina[1,2], Riscanu Laura Adriana[1,2], Knieling A.[1,2], Enea M.[2]**
Scientific adviser: Bulgaru Iliescu Diana, MD, PhD, Associate professor
[1]Iasi Forensic Institute, Romania
[2]*Grigore T. Popa* University of Medicine and Pharmacy, Iasi, Romania

Background. Cases when unexpected death occurs, when the patient is in apparent health or the event comes during presumably normal activity, especially when it is unwitnessed or when the victim is found without apparent signs of foul play, may pose a significant challenge to the coroner in reaching a proper determination of the cause and the manner of death. Spontaneous intracerebral hemorrhage (ICH) is a bleeding into the parenchyma of the brain and accounts for approximately 10 % to 20 % of all strokes. ICH is a multi-factorial disease caused by several interacting and overlapping risk factors and etiologies. When massive ICH, not connected with head trauma, has occurred, and it is multifocal or not located in one of the typical sites for hypertensive hemorrhage, one of a multitude of other causes must be suspected. High alcohol intake increases the risk of all stroke subtypes and of the development of liver diseases and may induce hypertension, by affecting brain function and producing a series of alcohol-related or alcohol caused diseases and is associated with changes in the coagulation system. Liver cirrhosis is a well-known risk factor for ICH, due to impaired coagulation, despite the relatively rare occurrence of ICH in cirrhotic patients.
Case report. The authors report a case of a 48 years old man, who was admitted in the Neurosurgery Department, being found in the street, with a present state of consciousness, presenting aphasia, right hemiplegia. The first computer tomography revealed left side temporal-parietal-occipital intracerebral hematoma of 52/20/45 mm, postcentral intergyral subarachnoid hemorrhage, and cerebral atrophy. On the second computer tomography, the lesions underwent moderate resorption, and a conservative treatment for ICH was chosen. After 28 days from the admission in the hospital, the patient died, due to a cardio-respiratory arrest. The release diagnosis was: Left Side parietal-occipital itracerebral haematoma. Right hemiparesis. Hepatic encephalopaty. Mixed decompensated alcoholic liver cirrhosis. Scleral and tegumentary jaundice. Ascites. Hypersplenism. Severe thrombocytopenia. Hypoalbuminemia. Bronchopneumonia. Schizophrenia. The body was brought for autopsy at the Iasi Forensic Institute. The necropsic examinatiation revealed: right side occipital epicranial hemorrhagic infiltration, a left side parietal-occipital lobe blood collection, cerebral oedema, pachypleuritis, bronchopneumonia, ascites (5 liters), cirrhosis, and splenomegaly.
Conclusions. Proper documentation of injuries, along with history of the case, has a huge importance in reaching a conclusion on both the cause and the manner of death. In this case, the absence of external head injuries, the absence of underlying brain lesions that would be suggestive for a head trauma, the presence of risk factors for primary non-traumatic ICH, make a context in which the case may be properly interpreted.
Key words: Head injuries, intracerebral hemorrhage, case report.

21. FOSTER KENNEDY SYNDROME AS AN INITIAL PRESENTATION OF NEUROFIBROMATOSIS TYPE 2: A CASE REPORT

Authors: **Alexandru Sumleanschi, Serghei Borodin**
Scientific adviser: Aureliu Bodiu, MD, PhD, Professor, Department of Neurosurgery, Republican Clinical Hospital
Nicolae Testemitanu State University of Medicine and Pharmacy of the Republic of Moldova

Background. Foster Kennedy syndrome (FKS) is described as ipsilateral optic atrophy and contralateral papilledema from an intracranial mass. FKS is uncommon manifestation of Neurofibromatosis type 2 (NF2), which is generally presented with hearing loss and tinnitus.
Case report. In this report we present a 26-year-old female with the atypical presentation of NF2. First symptoms were progressive vision loss and cognitive dysfunction. Ophthalmological examination revealed right-sided papilledema and left-sided optic atrophy. Magnetic resonance imaging (MRI) of the brain revealed bilateral vestibulocochlear schwannoma and three intracranial meningiomas, involving the parafalcine region and the olfactory groove. Whole spine MRI showed one intramedullary tumor at C1-C2 level, multiple spinal canal nodules in cervico-dorsal regions and one Th12-L2 extramedullary tumor. Based on clinical and imaging findings the diagnosis of neurofibromatosis type 2 was established. The patient underwent surgical resection of giant parasagittal meningioma, subtotal resection of the olfactory groove meningioma and total resection of Th12-L2 meningioma. Six months after brain surgery, she underwent Gamma knife radiosurgery for remnant frontobasal meningioma and for both vestibulocochlear schwannomas. Despite the combined treatment of intracranial lesions, only an insignificant vision improvement was achieved.
Conclusions. FKS can be the presenting symptom of NF2. Early detection and treatment of ophthalmologic manifestations of NF2 may prevent amblyopia development.
Key words: Foster Kennedy syndrome, neurofibromatosis type 2, intracranial meningioma, intramedullary tumor, extramedullary tumor

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22. INTRACEREBRAL HEMORRHAGE IN A PATIENT WITH MOYAMOYA SYNDROME: CASE REPORT

Authors: **Pavel Gavriliuc[1, 2], Elena Costru-Tașnic[1], Tatiana Pleșcan[1], Dacin Ianuș[2]**
Scientific adviser: Gavriliuc Mihail[1], MD, PhD, Professor, Department of Neurology and Neurosurgery
[1]*Nicolae Testemitanu* State University of Medicine and Pharmacy of the Republic of Moldova
[2]Institute of Neurology and Neurosurgery of the Republic of Moldova

Background. Moyamoya disease is a cerebrovascular disease that is characterized by bilateral chronic and progressive stenosis or occlusion of the arteries around the circle of Willis with development of collateral circulation, of unknown etiology. It has a high incidence in Japan and Asian population, with fewer cases described in Europe. Similar angiographic findings can be seen in patients with other medical conditions that are described as Moyamoya syndrome. Main clinical features include transient ischemic attacks, ischemic strokes, and hemorrhagic strokes.
Case report. We describe a 38-year-old female patient that presented with an intracerebral hemorrhage with a typical location for hypertensive bleeds. She had no vascular risk factors, but a high normal blood pressure (140/90 mmHg), and elevated ESR. A magnetic resonance angiography showed occlusion of internal carotid artery with development of collateral cerebral circulation on the side of the bleeding. Unilateral affection and elevated ESR were more characteristic for a moyamoya syndrome within a systemic disease.
Conclusions. Despite a typical hypertensive location of the bleeding, vascular imaging is warranted in all patients with intracerebral bleedings to evaluate for atypical etiologies. Our case represents a patient that might benefit from revascularization surgery in the context of multifactorial risk factor control.
Key words: Moyamoya syndrome, stroke, hemorrhage, intracerebral, collateral flow.

23. POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME MIMICKING