results in predominant C3 without significant Ig deposition on immunofluorescence microscopy and characteristic sausage-shaped, wavy deposits by electron microscopy within the glomerulus. Disease clinically presents as proteinuria, hematuria, acute nephritis or nephrotic syndrome and acute kidney failure.

We are presenting an 8 years old boy that was brought to the clinic because of macroscopic hematuria. At initial presentation he had high blood pressure and periorbital edema. Urinalysis revealed numerous erythrocytes, proteinuria and presence of casts in the urine. Creatinine and urea concentration was elevated with low serum protein and albumin. No pleural or pericardial effusion was found. Urine culture was sterile and urinary tract ultrasound did not show anatomical anomalies or urolithiasis. He had proteinuria of 1,69 g/24h (74,2 mg/m²/h) and albuminuria of 1096 mg/24h. Glomerular hematuria was confirmed with the finding of 100% dysmorphic erythrocytes in the urinary sediment. Serum C3 concentration was low with normal C4. In accordance with anamnestic data on febrile sore throat 2 weeks prior to hospitalization, the antistreptolysin titre was slightly elevated. Immunological screening excluded lupus nephritis (negative ANA and ANCA), and serological examinations excluded hepatitis C and B. The patient was treated with oral antimicrobial course, furosemide and amlodipine, regulated diet. Repeated serum creatinine and urea concentrations were being improved and lastly were normalized during outpatient monitoring. Constant recovery of proteinuria and albuminuria was being observed and microscopic hematuria resolved after 4 months. Low C3 concentration persisted for another 2 months. Renal biopsy revealed focal endocapillary proliferative glomerulonephritis (GN) and GN with the dominant C3 finding on the light and immunofluorescence microscopy that may suits post-infective GN as well as C3 glomerulopathy. Electron microscopy showed a finding which is in accordance with DDD. Additional testing revealed decreased classical (43 CH50/ml; ref. range 48–103 CH50/ml) and deficient alternative (3%; ref. range 70–125%) pathway activity as well as slightly positive C3NeF, supporting the diagnosis of DDD. Genetic analysis revealed a polymorphism of the complement factor H gene with an increased risk of developing DDD. The child was fully recovered after supportive therapy. After a follow-up period of 2 years patient remained asymptomatic with normal kidney function. Given the poor prognosis of the disease, the proper approach to such specific glomerulopathy is important to avoid or at least to slow the progression to end-stage renal disease.

495

TOXIC ALVEOLAR CELL INJURY IN YOUNG SMOKER

¹Anita Đula*, ²Roberta Šarkanji-Golub. ¹Dom zdravlja Zagreb – Zapad; ²Irma Golmajer – Vlahović, Ivan Pavić

10.1136/archdischild-2021-europaediatrics.495

Introduction Almost 2,500 children under the age of 18 burn their first cigarette every day, more than 400 of them will become active smokers (1), and half will die from the effects of smoking (2). Nicotine from cigarette smoke, as well as marijuana smoke (THC), consists of toxic mixtures of gases and particulates known to have adverse effects on the lungs (3,4). In this case report, we present a 15-year-old young man with hemoptysis caused by toxic alveolar cell injury resulting

from overexposure to tobacco smoke in combination with marijuana consumption.

Case Report A young man, aged 15, is admitted to our clinic due to occasional bloody cough during last couple of months. He was without known previous diseases, has been properly vaccinated and has not been in contact with tuberculosis (TB). He is an active athlete, did not lose weight, was not febrile lately.

Also, he is active smoker (up to 5 cig/day). At admission he was at good general condition with physical status without any abnormalities, including neat BCG scar. The laboratory work and chest radiography that was done didn't show any significant abnormalities. Additional exams (allergological, pulmonary, immunological, rheumatological, ENT) were uninformative too. Since the diagnostic work-up did not find the cause of hemoptysis, bronchoscopy was performed showing no specific macroscopic finding. Finally, a bronchoalveolar lavage (BAL) that was taken for cytologic analysis revealed toxic alveolar cell injury causing alveolar hemorrhage that was clinically manifested as hemoptysis. When confronted with the findings, the patient subsequently told us that he had been smoking excessively (>40 cig/day plus occasionally smoked marijuana) two weeks before the onset of symptoms, leading us to conclude that the problems were most likely caused by smoking a combination of tobacco and marijuana. The patient was advised to strictly avoid both active and passive smoking after which hemoptysis did not recur, which was an indirect confirmation of our assumption that smoking was a major cause of toxic alveolar cell injury.

Conclusion In addition to the most commonly known causes of hemoptysis in children with respiratory infections, aspirated foreign bodies and bronchiectasis, other rare causes should be kept in mind. To confirm toxic alveolar damage, the method of choice is bronchoscopy with cytological analysis of bronchoalveolar lavage (BAL). Also, young people need to be educated and informed about the dangers of smoking and to draw attention to the consequences of smoking.

496

WHEN THE RESULT OF ANCILLARY TEST DOES NOT SUPPORT NEUROLOGICAL CRITERIA OF BRAIN DEATH – A CASE OF A 3-YEAR OLD GIRL WITH HEAD TRAUMA

Nikolina Hamidović*, Anamarija Čondor, Igor Berecki. *University Hospital of Osijek*

10.1136/archdischild-2021-europaediatrics.496

Brain death occurs when a person has an irreversible brain injury causing total cessation of cerebral and brainstem function, different from coma or persistent vegetative state. The diagnosis of brain death is made clinically with different practices for need of confirmation with ancillary test in different countries.

Here we present a case of a 3-year old girl that was admitted to the emergency room after a fall with a Glasgow coma scale score 3 but still breathing spontaneously. She underwent head and cervical spine computed tomography (CT) that showed smaller bilateral frontotemporoparietal subdural hematoma and subarachnoid haemorrhage. After a neurosurgical consultation she was initially treated conservatively. About three hours later her condition profusely deteriorated with apnoea, hemodynamic instability and profound anaemia. Second CT scan showed oedema and decompression craniotomy was indicated. 72 hours after the trauma the patient was