Results: 51 patients were diagnosed for acute HAV (N = 32) or HEV (N = 19). There was a higher proportion of men in HEV cases than HAV cases (84 % vs. 40% p = 0.002). In comparison to hepatitis E cases, patients with acute hepatitis A were more symptomatic (87% vs. 63 % p = 0.04) and presented more prurit (28% vs. 5% p = 0.05). Ferritin level (mean 3706 ± 5170 µg/L vs. 982 ± 1739 µg/L p = 0.01) were higher in patients with HEV compared to patients with HAV.
Conclusion: Although HAV infections seemed more symptomatic, HEV infections are associated with higher levels of ferritin. Furthermore, HEV infections affected a higher proportion of men than HAV infections. However, there are no clinical parameters or biomarkers that differentiating clearly HAV from HEV.

0015 Polyarteritis nodosa associated with chronic lymphocytic leukemia

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ABSTRACT

We reported the case of a 63 years old female who presented to the ER with fever, shivering, nausea and lower back pain that appeared four days earlier.

She was treated since 2011 for chronic lymphocytic leukemia (with deletion 17p) and had undergone several therapeutic lines (alemtuzumab, ibrutinib, rituximab-idelalisib). The latter was stopped 3 months earlier and had been complicated by grade III lymphocytic colitis. She started Venetoclax 2 months before the admission.

Blood test revealed elevated CRP up to 32 (n.v. <0,5 mg/dl), a grade III thrombocytopenia (32 000/mm^3) but no neutropenia nor lymphopenia. Urine test showed leukocyturia (207/µl) and slight hematuria (11/µl). She was hospitalized with presumptuous diagnosis of urinary tract infection and received IV cefuroxime.

A few days later, high fever without any cyclic pattern remained and she developed a purpuric non-palpable rash, beginning on the lower limbs then extending to the trunk. All bacteriological tests were negative and antibiotic was discontinued as toxidermia was a plausible cause, but the rash continued to spread. Cutaneous biopsy showed no immune complexes or other specific signs.

Infectious agents such as EBV, CMV, VZV, HHV8, Coxsackie, Parvovirus B19, Measles, Rickettsia, Coxiella, Hantaan, Syphilis, Brucella, Leptospira, Puumala, Leishmania, Anaplasma, and tropical diseases like malaria and dengue (she recently came back from Morocco) were ruled out. Auto-immune serologies (ANCA among others) were negative and complement fractions were normal.

Bone marrow biopsy did not show hemophagocytic syndrome nor agressive lymphomatous transformation.

Abdominal CT angiogram did not reveal microaneurysm but a radiologic aspect of inflammatory cholecystitis without any biological or clinical feature.

Some days later, she developed lower limbs pain and paresthesia. Electromyography was consistent with multiple mononeuropathy. A neuromuscular sural biopsy showed segmental necrotizing arteritis consistent with periarteritis nodosa. Significant proteinuria appeared and leukocyturia and hematuria persisted.

A diagnosis of dysimmunitary, **chronic lymphocytic leukemia-linked polyarteritis nodosa** was concluded.

Hepatitis B serology was negative.

She received corticosteroids (Methylprednisolone 1g pulse therapy then slowly tapered), with positive response of the fever and rash. Cyclophosphamide was not considered due to the haematologic disease and a low Five Factor Score (0/5). Proteinuria was treated with an ACE inhibitor.

A few months later, she developed bilateral necrotizing retinal arteritis, treated with laser and topical corticosteroids.

Venetoclax was rapidly discontinued after admission as suspected to be the main cause of the vasculitis, but was later rechallenged without any side effect. A triggering role of the previous idelalisib is not excluded.

The patient died 1 year later from hemorrhage in a context of thrombocytopenia.