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AN INSIDIOUS CASE OF ASPIRIN INDUCED LEUKOCYTOCLASTIC VASCULITIS MIMICKING CARDIORENAL SYNDROME.

Suela Mumajesi[1],Leart Berdica[2] ,Matilda Imeraj[1], Erjola Bolleku[1], Myftar Barbullushi[1]

[1]Department of Nephrology, UHC" Mother Theresa "Tirana, Albania

[2] Anatomo-Pathology and Forensic Department of Medicine Faculty of Tirana Medical University.

Abstract: Male 57 years , was presented with fatigue, dark urine, limbs edema and itching. Light rash was observed during inspection. The signs were firstly observed after taking baby aspirin. Initially, we thought of acute glomerulonephritis, but laboratory examinations and skin biopsy showed that it was leukocytoclastic vasculitis..

_ *Key words: CVD, LVC, NSAIDs, aspirin, cardiorenal syndrome*

Introduction: Aspirin is a nonsteroidal anti-inflammatory drug (NSAIDs), widely used. Often patients undergoing percutaneous coronary stent with stable coronary disease, take aspirin either to reduce the risk of other thrombotic events, or to prevent the risk of myocardial infarction. Along with its benefits: anti-inflammation and pain reducer, aspirin is also associated with several Adverse Drug Reactions (ADRs) including leukocytoclastic vasculitis (LCV). Leukocytclastic vasculitis is one of the main sources of vasculitis. In most of cases, leukocytoclastic vasculitis are self-limited and usually result in complete recovery without sequel1. We will present a case of a male patient, 57 years of age, who has been taking daily aspirin 100mg, after coronary revascularization. Although, we all think it's a small dose, the patient presented many side effects: systemic vasculitis and cardiorenal syndrome type 2, which are rare presentation, but we should not ignored.

Case report: Male, 57 years, was presented to the ambulatory of renal care, because of fatigue, dark urine, limbs edema and itching. The patient was immediately hospitalized at our department for further investigations. Three days before admission the patients had subfebrile temperature. The patient history shows, cerebrovascular accident ten years ago and coronary revascularizations four months ago. During this period, he was taking clopidogrel and a month ago, he switched into baby aspirin. Several days after starting the use of aspirin, the symptoms appeared and got aggravating. During inspection it was noticed light rash with macula popular elements mainly on abdomen (Figure 1), and in the lower extremities (Figure2) , which were palpable non-blanching purplish in color, and resembled cutaneous vasculitis. The use of aspirin was immediately stopped. The patient has no history neither of diabetes nor smoking. Physical finding: he was afebrile with blood pressure of 140/85 mm Hg,pulse rate of 86/minute and body mass index (BMI) 25.2 kg/m2, tonsils normal. EKG shows sinusal rhythm, and abdomen was free of pain.

Laboratory examination have shown normal complete blood count,lipid profile was normal, liver functional test normal, fasting glycaemia normal, serum creatinine 2.8 mg/dl (NR 0.3-1.2mg/dl), urea 114 mg/dl (NR 0-50 mg/dl).Inflammatory tests including C-reactive protein, erythrocyte sedimentation and gamma interferon were within normal range. Urine examination revealed the presence of urinary red blood cells and proteinuria 6.6 gr/day. Microscopic urinalysis for red blood cell (RBC) morphology showed 72% dismorfic RBCs and 28% izomorfic RBCs. Echocardiography was normal. Computed tomography of the abdomen and thorax was normal (no evidence of vasculitis). Additional workup including ANA, RF, ANCA antibodies, complement levels, HIV antibodies and hepatitis B and C serology was within normal limits. The skin biopsy was firstly performed. Our first thoughts where: cardiorenal syndrome type 2 due to nephrotic syndrome, a probable membranous nephropathy or any type of vasculitis. He started methylprednisolone pulse 1 g intravenously daily for 3 days followed by 60 mg daily. He was rapidly improved. The vascular elements disappeared and he was discharged after 10 days. The next follow up was within a months. When he came back, neither vascular elements were present, nor red blood cells in urine examination, urea and creatinine levels were all normal, but mild proteinuria was present. Skin biopsy showed leukocytoclastic vasculitis (Figure 3). In this case, methylprednisolone was tapered fast. The next follow-up was three months afterwards and he was reported in good condition with normal urea and creatinine levels, no vascular elements, no proteinuria.

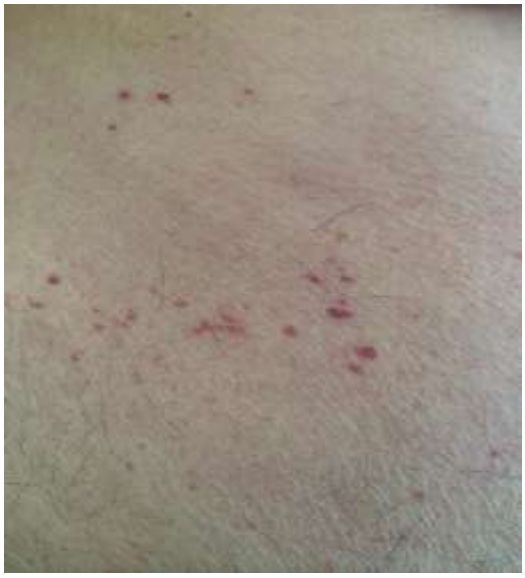

Figure 1: Maculo papulous elemnts in abdomen

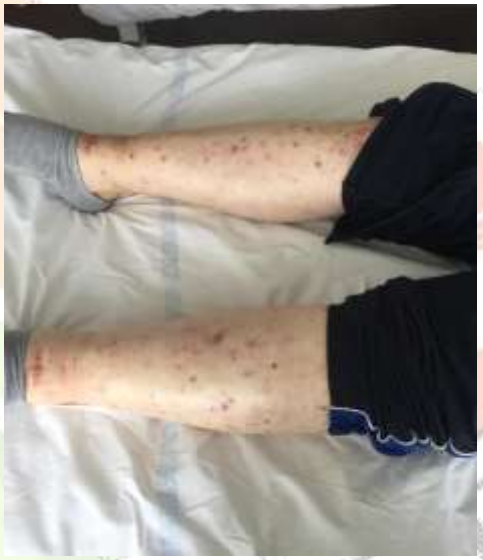

Figure 2: Maculo papulous elemnts in the legs

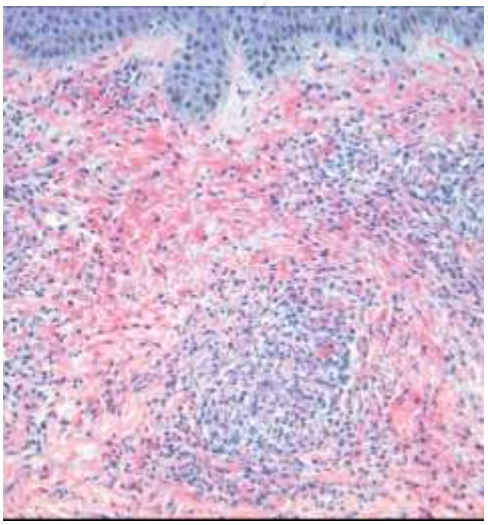

Figure 3 :Leukocytoclastic inflammatory infiltrate around the small vessels.
Dr.Sc. Leart Berdica
Anatomo-Pathology and Forensic Department of Medicine Faculty of Tirana Medical University.

Discussion: Cardiorenal syndrome is largely observed among patients with Cardiovascular Disease. Cardiorenal syndrome type two, is often recognized in patients treated with ACE-inhibitors, ARBs, diabetic patients with uncontrolled glycaemia, sepsis and in cases of frequent used of pain relievers such as non-steroidalanti-inflammatory drugs (NSAIDs). The renal involvement secondary to NSAIDs use, is mediated via inhibition of prostaglandin synthesis from arachidonic acid by non-specific blocking of the enzyme cyclooxygenase, leading to vasoconstriction and reversible mild renal impairment in volume contracted states2,3. Aspirin induced leucostoklastic vasculitis is very rare, a few cases are reported. The study reported by eHealthMe5 was based on 93 reports from the Federal Drug Association (FDA) on (roficoxib) and Celebrex (celecoxib) and was thought to be due to the sulfa component in celecoxib and roficoxib. Presentation of symptoms to those agents was in the form cutaneous vasculitis, and renal involvement in the form of proteinuria and hematuria. Garcia et al8.reported that 15% of patients who presented with drug induced vasculitis had evidence of renal impairment and acute renal failure4. LCV can occur at any age group and is thought to effect men and women in equal numbers but few studies suggest male predominance5. In conclusion, in our case the diagnostic dilemma was due to the unusual occurrence of vasculitis. The patient was presenting acute glomerulonephritis.However, laboratory findings did not support our theory, meanwhile regarding vasculitis, we could not detect any systemic changes. Multiple NSAIDs including aspirin can act as a potential trigger for LCV6. In conclusion, the best treatment of such cases of LVC is the identification and removal of potential etiological triggers7.

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