ABSTRACT
A 27 year-old gentleman referred to us for second opinion for refractory Henoch-Schönlein Purpura (HSP) from outside hospital. Patient has a four months history of lower extremity rash, arthralgia, myalgia, abdominal pain with headaches, dark colored urine, and spiking fevers of 102F. Patient reports that symptoms worsened on two occasions when he switch from brand to generic losinopril. We report case of refractory HSP likely secondary to generic losinopril treated successfully with Rituximab.

INTRODUCTION
HSP is a small vessel vasculitis affecting the skin, joints, gut, and kidneys and predominantly affects children. It is defined by tissue deposition of Immunoglobulin A (IgA). HSP was described independently by Schönlein and Henoch in 1837 and 1874, respectively. The nephritis associated with HSP is characterized by IgA mesangial deposition. HSP nephritis histologic presentation is indistinguishable from IgA nephropathy (Berger’s Disease). They are differentiated by extra-renal manifestations. HSP has been associated with chronic liver disease, celiac disease, HIV, hepatitis C, sarcoid, and uveitis. There are sparse reports that generic losinopril can trigger HSP. Generic medication is usually covered in insurance plans because they are less expensive and have lower copay. Although they have equivalent active compound, the remaining excipients and filters may be the source of the immunogenic reactivity leading to HSP. Companies producing generic drugs have to show equivalency of bioavailability within a range of 80-125% of the brand name drug, but no strict standards control the inert materials and their impurities.

HISTORY OF PRESENT ILLNESS
Patient reports switching from a brand to generic losinopril two days before developing symptoms. On two occasions symptoms worsened when he was switched back to the generic losinopril. Patient denies travel history, exposure to chemicals or activities that may have resulted in environmental exposures to noxious compounds.

PAST MEDICAL HISTORY/ SOCIAL HISTORY/ FAMILY HISTORY
Patient reports chronic pain syndrome, lower extremity deep vein thrombosis, hypertension, and obesity. He denies smoking and drinks alcohol sporadically. Works in an office environment and is married with two kids. Patient denies use of recreational drugs. No past surgical history. Family history is non-contributory.

PHYSICAL EXAM
Patient vital signs were 129/80, 76, 18, 36.8C, Pox 96% in room air. Obese 27 year-old gentleman alert and oriented x 3 in no acute distress. Head was normocephalic atraumatic, conjuctiva was non-icteric, throat non-erythematous or sores noted; no thyromegaly and neck supple without prominent lymphadenopathy. Lungs were clear to auscultation bilaterally with no wheezing, rales or rubs. Heart was normal rate and rhythm with no murmurs, gallops or rubs. Abdomen soft, non-tender or distended (large body habitus) with normal bowel sounds. Bilateral lower extremity pitting edema 2+ and a bilateral pruritic erythematous rash (Figure 1)

REFERENCES
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DISCUSSION
Patient was discharged home with improving renal and extra-renal manifestations and satisfied with his recovery of his refractory adult onset HSP. He will be followed in clinic for complete resolution of his acute kidney injury. His medication regimen was modified to avoid losinopril during his recovery period. Current limited evidence shows that adding Rituximab as the only immunomodulator in refractory HSP. Although data is limited, Rituximab appears promising for refractory HSP and possibly other vasculitides.

CONCLUSION:
The HSP nephritis is a rare kidney disease leading to ESRD in up to 30% of adult patients during long-term follow-up. HSP is characterized by systemic vasculitis of unknown etiology. It is frequently associated with skin, joint, and gastrointestinal manifestations, as well as renal involvement in approximately 50% of patients. Renal involvement is characterized by vascular and/or mesangial IgA deposition and associated with a negative long-term prognosis. The etiology of HSP remains obscure, but a variety of antigenic stimuli, including drugs such as cyclophosphamide, ampicillin, erythromycin, clarithromycin, quinine, enalapril and losinopril have been considered triggers. Most of all, reports of a case of adult HSP after treatment with enalapril. Desler et al., reported on a similar case with losinopril, which like enalapril, does not contain the thiol moiety. Both cases had improved significantly 1-3 months after withdrawal of ACE-I. Drug-associated HSP typically resolves rapidly after discontinuation of the offending drug except in refractory cases where further more aggressive treatments may be necessary. For refractory cases of HSP, Rituximab treatment is showing some reported success inclusive of this case.