INTRODUCTION
Lupus enteritis (LEn), defined as vasculitis or inflammation of the small bowel, is an unprecedented manifestation of SLE that affects 0.2% to 5.8% of these sufferers. Diagnosis of this situation is hard, specifically with out different symptoms related to active SLE. To our understanding, just a few cases reviews point out lupus enteritis because of the simplest initial presentation of active SLE. Well-timed diagnoses and treatment of LEn are important to prevent the maximum serious effects: intestinal perforation and gastrointestinal bleeding.

SLE-mediated damage to the digestive system can occur as oral ulcers, pseudo intestinal obstruction protein-losing enteropathy, liver damage, autoimmune pancreatitis, lupus enteritis (LEn), and other headaches. LEn is also known as lupus mesenteric vasculitis, gastrointestinal vasculitis, or acute gastrointestinal syndrome. Pain is the primary abdomen symptom of LEn, which can be followed by diarrhea and vomiting. LEn must be suspected in SLE sufferers with intestinal symptoms after infection exclusion.

Gastrointestinal involvement is generally found in 40–60% of SLE sufferers. Clinically diagnosed gastrointestinal manifestations have been defined in 8–10% of sufferers. Post-mortem research records findings of gastrointestinal involvement in 60–70% of sufferers, suggesting that subclinical or unrecognized involvement is commonplace. Most gastrointestinal manifestations are typically slight. Vasculitis and thrombosis may be liable for life-threatening manifestations, mainly ischemia, perforation and infarction, if not identified early and adequately treated. This article will report a woman with SLE and lupus enteritis as the first manifestation of active systemic lupus erythematosus.

CASE PRESENTATION
Chief complaints
A 25-year-old female presented to the Emergency Department (ED) at Dr. Soetomo General Hospital complaining of diarrhea, abdomen pain, vomiting, and nausea.

History of present illness
The patient’s signs and symptoms began three months in the past and worsened 1 week before arrival on the emergency room. She reported the abdomen pain as sudden onset, diffuse, and started in the center upper region of the abdomen and radiated to the back and right lower a part of the abdomen. It became related to nausea, she additionally experienced non-bloody, non-bilious vomiting earlier than arrival. She said abdomen bloating together with abdomen pain. She additionally said one month of recurrence of diarrhea. The patient defined the diarrhea frequency as once in a while with blood. She experienced a loss of appetite and weight loss because of nausea and vomiting that she had been experiencing. The patient denied fever, chills, cough, shortness of breath, night sweats, and prior contact with tuberculosis patients.

CASE REPORT
A case report of a woman with SLE and lupus enteritis as the first manifestation of active systemic lupus erythematosus

Fitria Yulistiawati1*, Awalia2

ABSTRACT
Introduction: Lupus enteritis is a rare first manifestation of active systemic lupus erythematosus (SLE), diagnosis of this condition is hard, specially without other signs and symptoms associated with active SLE. this article will report a woman with SLE and lupus enteritis as the first manifestation of active systemic lupus erythematosus.

Case presentation: A 25-year-old woman presented to the Emergency department complaining of abdominal pain and vomiting. From her medical history, four years in the past, she reported the same signs and symptoms as now and turned into identified with appendicitis. Her abdomen examination found out mild abdominal distention without a abdominal tenderness. Wall thickening and irregularity of small and big bowel loops were observed on the CT abdomen. She turned into given a excessive dose glucocorticoid as initial treatment and turned into maintained with low dose glucocorticoid and cyclosporin. After 6 months of follow-up, the patient did not have a recurrence of signs and symptoms.

Conclusion: A high index of suspicions is essential to differentiate lupus enteritis from other differential diagnoses. For lupus enteritis, excessive-dose steroids were an efficient initial treatment.

Keywords: case report, lupus enteritis, systemic lupus erythematosus.
One year prior, the patient had developed arthralgias of her wrists, fingers, shoulders and ankles with morning stiffness which was not resolved in 1 hour and improved with activities. She also reported hair loss and fatigue. She also experienced skin rash on her arms and legs, especially after sunlight exposure. She was diagnosed with Systemic Lupus Erythematosus at Bangkalan General Hospital and regularly controlled every month. She has been treated with Hydroxychloroquine two hundred mg day by day, and she denied any flare from the time of SLE diagnosis until the onset of the signs and symptoms. There's no complaint about arthralgias at the time of examination. The rest of her overview of systems was non-contributory.

**History of past illness**

Her medical history is hypertension in the past 4 years, and she has been treated with candesartan 8 mg daily. She also had a history of appendicitis operation 4 years ago. The patient presented with the same symptoms as now but without diarrhea.

**History of Obstetric**

When she was in her first pregnancy 3 years ago, she had a miscarriage. The pregnancy was 6-month-old.

**Physical examination**

Physical exam on presentation, the patient's vital signs had been: 36.7 °C, heart rate of 85 bpm, blood pressure of 158/90 mmHg, respiratory rate of 20, and oxygen saturation of 98% on room air. Her abdomen examination found out normal bowel sounds. There was mild abdomen distention without a abdomen tenderness. Shifting dullness was not found in physical examination for ascites. Post appendectomy operation scar lesions are in the lower right quadrant of the abdomen.

**Imaging examinations**

A contrast computed tomography of the abdomen revealed wall thickening and irregularity of small and large bowel loops. That finding was in accordance with the appearance of enterocolitis. It also revealed hepatomegaly and minimal ascites in the pelvic cavity. On ultrasound examination of the abdomen, an intraluminal, solid mass with an unclear margin in the pelvic cavity was found, giving the appearance of a colon rectosigmoid mass. The liver, kidney, gallbladder, spleen, and pancreas showed no abnormality on the abdomen ultrasound examination. The patient's plain abdomen radiographs were normal. The patient also underwent a lumbosacral spine x-ray showing no sacroiliac joint sacroiliitis.

**Laboratory examinations**

The patient had leukopenia with a WBC count of 2460/uL and lymphopenia with an absolute lymphocyte count of 440/uL. There is a positive antinuclear antibody titer of 1:1000. Erythrocyte sedimentation rate (ESR) was 8 mm/h (normal: 0–8 mg/l). C reactive protein (CRP) was 0 mg/L (normal: 0–15 mm/h). Complement components C3 and C4 were 27 mg/dL and 3.7 mg/dL, respectively (normal: 86-160 mg/dL and 17-45 mg/dL, respectively). There is no sign of neutrophile activity, and a small number of eosinophile. Of lymphocytes, histocyte, plasma cells, and a small number of eosinophile. There is no sign of neutrophile activity, specific inflammation process, or sign of malignancy. The patient was diagnosed with lupus enteritis.

**Management**

The patient was given systemic steroids with methylprednisolone 62.5 mg once daily intravenous, and within 7 days, the abdomen pain and diarrhea began to resolve. The patient has been discharged on a tapering dose of steroids methylprednisolone 16 mg three times daily per oral.

**Outcome and follow-up**

She had significant improvement in abdominal pain, diarrhea, and vomiting after the tenth day of care. She turned into discharged on methylprednisolone 16 mg by mouth three times every day and...
**CASE REPORT**

Table 1.  **Laboratory examination.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Parameter</th>
<th>Value</th>
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<tr>
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<td>MCV</td>
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<tr>
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<tr>
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<td></td>
<td>BGA</td>
<td>Room Air</td>
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</tbody>
</table>

planned for a follow-up visit in 1st week. before the patient could do her follow-up visit, she had a recurrence of signs and symptoms three days after discharge and became delivered to the emergency unit four days later for worsening signs and symptoms.

The patient complained of bloating, abdomen pain, and vomiting. during her second hospitalization, she received a pulse dose of 500 mg of Metil Prednisolone intravenous for three days. Methylprednisolone become tapered off to 16 mg three times every day while discharged from the hospital. From her follow-up visit, methylprednisolone became tapered off once more until a maintenance dose of 4 mg once every day. The patient was additionally given cyclosporin (Sandimun®) 25 mg two times daily. After 6 months of treatment with cyclosporin and 4 mg of methylprednisolone, the patient did not have a recurrence of signs and symptoms.

**DISCUSSION**

This case has reported a case of lupus enteritis as the initial manifestation of active SLE. Lupus enteritis is visible in only 13% of sufferers without a previous diagnosis of SLE. To our understanding, there are only 10 formerly stated cases in which lupus enteritis turned into the initial presentation of active SLE. Damage to the digestive system is common in those sufferers because the SLE can result in recurrent oral mucosal ulcers, lupus hepatitis, autoimmune pancreatitis, protein-losing enteropathy, and LE. Furthermore, gastrointestinal reactions and liver dysfunction may result from the use of non-steroidal anti-inflammatory agents, glucocorticoids, immunosuppressants, and other drugs. Some scholars agree with that lupus enteritis is the premise of a sequence of processes consisting of intestinal vasculitis, mesenteric arteritis, abdomen serositis and lupus peritonitis. The majority assume that lupus enteritis is vasculitis or inflammation of the intestinal tract. They believed that immune complex deposits and complement activation cause lupus enteritis. Supplement activation can then reason microvascular damage and elevated permeability leading to submucosal edema of the intestine. BILAG defines lupus enteritis as small bowel vasculitis or inflammation identified with imaging and/or biopsy. It could be fatal in 11% of active cases and requires expeditious diagnosis and treatment. If left untreated, this could lead to bowel infarction and perforation. A literature review through Ju et al. stated that the worldwide prevalence of LE in SLE sufferers became about 0.2 to 9.7%. A few terms were used to explain gastrointestinal tract vasculitis, including lupus mesenteric vasculitis, mesenteric arteritis, lupus enteritis, lupus arteritis, lupus vasculitis, lupus vasculitis, lupus vasculitis, gastrointestinal vasculitis, intra-abdomen vasculitis, and acute gastrointestinal syndrome. Even though the underlying lesion in most cases of gastrointestinal (GI) vasculitis in SLE is small vessel arteritis or venulitis, vasculitis in biopsy studies is not found in all cases. Gastrointestinal activity is not among the 17 SLICC (Systemic Lupus international collaborating Clinics) criteria for SLE. For a patient to be identified with SLE, they have to fulfill at least four criteria, with at least 1 clinical criterion and one immunologic criterion or Lupus nephritis as the only clinical criterion inside the presence of ANA or anti-dsDNA antibodies. Our patient meets five clinical criteria, which might be (1) nonscarring alopecia, (2) Joint disease, (3) serositis with ascites, (4) renal with urine protein to creatinine ratio becoming >=0.50 g/gCr, (five) leukopenia and lymphocytopenia; and meets 2 immunologic criteria which can be (1) positive ANA and (2) low complement.

The American College of Rheumatology (ACR) established eleven criteria in 1982, revised in 1997 as a category tool for implementing SLE definitions in clinical trials. If a person has any four out of eleven signs, she is with SLE. In our case, the patient meets five criteria: (1) Photosensitivity; (2) polyserositis (ascites); (3) Arthritis; (4) Hematologic disease (Leukopenia, Lymphocytopenia); (5) positive antinuclear antibody titer of one; one thousand. From the above, the affected person with SLE is obvious. In most similar case reports, lupus
enteritis was not the initial diagnosis, with initial impressions ranging from infectious gastroenteritis to acute appendicitis. Further, diagnostic uncertainty can cause unnecessary invasive and expensive procedures, including appendectomy, exploratory laparoscopy, laparotomy, and SBE. From anamnestic, our patient also has a history of operation for acute appendicitis. The patient got here with comparable signs at that point because the signs and symptoms she experienced while lupus enteritis turned into identified.

SLE patients with gastrointestinal manifestations generally have a high SLE disease activity. But preceding studies stated inconsistent findings concerning the affiliation among LEn and active SLE. Buck et al. stated that lupus mesenteric vasculitis happened only in sufferers with active disease (SLE disorder activity index [SLEDAI] score > eight). However, Lee et al. showed that the SLEDAI score did not differ significantly among SLE patients with abdomen pain and LEn and SLE patients without LEn. Their findings advised that the SLEDAI score can be improper for disease evaluation and treatment selections in patients with LEn.3

Our patient scored five in the SLEDAI score, which is from alopecia (2+), low complement (2+), and WBC <3 x 10^9/L (1+). The above SLEDAI score classified the patient's sickness activity as mild (SLEDAI=1 to 5). Our patient denied signs and symptoms of other organ involvement. This created diagnostic difficulty in differentiating whether or not abdomen signs and symptoms result from the patient's SLE activity or other reasons. Primarily based on the patient's activity index, which became mild, the affected person became treated with 62.5 mg methylprednisolone once each day, intravenously. However then the patient returned with the same abdomen signs in less than 1 week. within the second hospitalization, methylprednisolone became increased to 500 mg each day intravenously for three days. The patient showed improvement in her stomach signs and symptoms and said no signs in 6 months follow-up.

Lupus enteritis offers with very non-specific signs and symptoms, which include abdomen pain (97%), ascites (78%), nausea (49%), vomiting (42%), diarrhea (32%), and fever (20%).2,15,16 Similar to the findings of previous research, Li et al. stated that the clinical signs and symptoms in LEn sufferers were not specific and abdomen pain became the most not unusual symptom.17,18 The most common signs and symptoms were abdomen pain and diarrhea.19 About 90% of our LEn sufferers had abdomen pain of various severity, and a few sufferers additionally had nausea, abdomen distension, diarrhea, and vomiting. Clinicians can also suspect LEn when abdomen signs and symptoms arise in SLE sufferers. This highlights the want for timely abdomen CT, specially enhanced abdomen CT that is extra sensitive in detecting intestinal abnormalities. In our case, the affected person complained about recurrent abdomen pain and diarrhea as the main symptoms leading to diagnosis. She additionally experienced nausea and vomiting. Even though physical examination did not reveal ascites, on CT abdomen examination, minimal ascites were located inside the pelvic cavity.

CT and pathology have come to be the gold standard for diagnosis. Common features consist of bowel dilation, circumferential bowel-wall thickening, unusual bowel wall enhancement (target sign), and engorgement of mesenteric vessels with a multiplied quantity of visible vessels (comb’s sign). Those are nonspecific because the above-defined abnormalities also can be seen in sufferers with pancreatitis, peritonitis, or Crohn’s disease.1,5,10,15 Most authors had a problem making the correct analysis even after obtaining the CT, given its lack of specificity.19,20 Lupus enteritis is the main reason for submucosal edema of the jejunum and ileum, leading to the appearance of the “target sign” and “comb sign”).21 In our case, the CT abdomen revealed a bowel wall thickening and irregularity of small and huge bowel loops. This gave the arrival of enterocolitis which also became nonspecific.

Further to CT manifestations, ultrasonography and magnetic resonance enterography (MRE) can also diagnose LEn. For example, Demiselle et al. defined a patient with LEn who had characteristic intestinal wall edema and ascites based on ultrasonography.21 Cicero et al. used MRE to examine a formation with the appearance of a thumbprint because of bowel ischemia and bowel wall edema in a patient with LEn.22 Our patient’s ultrasonography confirmed no edema on the intestinal wall or ascites. Moreover, it gave the appearance of an intraluminal, solid mass with an uncertain margin within the pelvic cavity which yielded malignancy of the rectosigmoid as a differential analysis. Endoscopy is usually neither helpful nor necessary in diagnosing lupus enteritis since only superficial tissue is analyzed.11,23 The yield of biopsy is only about 6%.24 Endoscopy with biopsy should be reserved to confirm or rule out alternative etiologies in cases of diagnostic uncertainty. Digestive endoscopy has low sensitivity in detecting LEn, and more than half of these patients have normal endoscopy findings. Because there was suspicion of rectosigmoid malignancy as a differential diagnosis based on the bloody stool and change in bowel habits symptoms, and based on ultrasound examination, we decided to perform a colonoscopy for our patient.

Fifty-six percent (56%) of sufferers stated within the literature underwent an endoscopic procedure with biopsy: 1 patient had a colonoscopy, 2 patients had an upper endoscopy, 1 patient had each endoscopy and colonoscopy, and 1 patient had a small balloon enteroscopy (SBE). Only the small balloon enteroscopy by means of Chowichian et al. yielded a definitive diagnosis of vasculitis, reiterating that endoscopy is of low yield in lupus enteritis. There is no unified view approximately the endoscopic appearance of lupus enteritis. Only one chinese professor defined a “deep ulcer” in a case report of lupus enteritis.20 Pathological findings in lupus enteritis consist of cellular infiltration of the submucosal and muscular layers, without or with edema or vasculitis. A few research mentioned biopsy outcomes with overt inflammation. Lupus enteritis is hardly ever diagnosed on biopsy, with rates as low as 6%.11 Even though the underlying lesion in most cases of gastrointestinal (GI) vasculitis in SLE is small vessel arteritis or venulitis, vasculitis in biopsy studies
isn’t always located in all cases. In our case, a colonoscopy was performed. The colonoscopy found out patchy hyperemia with prominent vascularization on the rectum and hyperemia and edematous mucosa of ascending colon, descending colon, transverse colon, and sigmoid colon colonoscopy. The ulcer was not founded on a colonoscopy examination. The basis of diagnosis in patients with SLE, the colonoscopy appearance was more inclined to intestinal vasculitis and bowel wall edema, often determined on lupus enteritis. Laboratory testing may aid in the diagnosis of lupus enteritis. Our patient had numerous laboratory markers consistent with SLE, which includes numerous hematologic (leukopenia and lymphopenia) and autoimmune (positive ANA and decreased complement levels). Leukopenia, hypoalbuminemia, hypocoomplementemia (C3 and C4), increased IgA level, and positivity for anti-nucleosome-ANA antibodies have been extensively more not unusual in sufferers with lupus enteritis. Lee et al. analyzed one hundred seventy five SLE patients and assigned them to three groups: (i) SLE + LEn with abdomen pain, (ii) SLE alone with + abdomen pain, and (iii) SLE alone without abdomen pain. They confirmed that leukopenia was more common in the first group than in the other two groups, in agreement with our findings.

A reduced complement C3 is usually a sensitive indicator of active SLE. A reduced C3 level was more common in SLE sufferers with LEn than those without LEn. Laboratory research from comparable case reports yielded a positive ANA in 100%, a positive ds-DNA in 80%, low complement levels in 70%, and positive anti-Smith antibodies in 20% of cases.

The diagnosis of SLE intestinal pseudo-obstruction was made based on concurrent urinary tract abnormalities consisting of ureterohydronephrosis and/or cystitis, typically absent in lupus mesenteric vasculitis. In our case, to exclude infectious causes of enteritis, fecal routine examination and gene Xpert for feces were done. Both examinations confirmed no sign of infection had occurred. There’s no concurrent urinary tract involvement from imaging examination to exclude the diagnosis of SLE intestinal pseudo-obstruction.

It’s also essential to rule out concomitant lupus nephritis, that is found in 65% of all lupus enteritis cases. It seems to co-exist in most SLE cases presenting initially with lupus enteritis. In our case, we did a urine evaluation for lupus nephritis screening. From it, we determined showed hematuria blood 2 (+) and protein 1 (+), urine protein to creatinine ratio was >=0.50 g/gCr and urine albumin to creatinine ratio was >=300 mg/gCr. With normal serum albumin and normal renal function test, more investigations and observation are had to exclude no renal involvement. The patient can be planned for a 24-Hour Urine Protein test and ds-DNA antibody.

There are presently no available recommendations or guidelines for the treatment of LEn. There are also no prospective controlled studies on treating lupus enteritis, however steroids seem to be the consensus first-line treatment. Consistent with a 2013 review by Janssens et al., the route and dose depend on the severity of abdomen pain and the response to signs and symptoms. Sufferers with slight abdomen pain tolerating oral intake need to receive oral prednisone at 1 mg/kg per day, sufferers with intense abdomen pain or not tolerating oral intake may additionally receive as high as methylprednisolone 250 mg to 1 g IV daily.

Depending at the clinical state or other organ involvement, steroid management can be intravenous or oral, preferably in the case of a severe lupus burst, as tissue edema resulting from enteritis may also reduce drug absorption.

Previous research stated that most LEn sufferers achieved remission following excessive-dose glucocorticoids, without or with the addition of immunosuppressive therapy. The major immunosuppressants used in these patients are cyclophosphamide, azathioprine, mycophenolate mofetil, and rituximab. Early case reviews determined that most sufferers with LEn obtained glucocorticoids and cyclophosphamide. Lian et al. retrospectively analyzed patients with SLE and acute gastrointestinal syndrome and confirmed that combined cyclophosphamide and glucocorticoids...
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significantly improved patient prognosis. Patients who do not respond to pulse dose steroids or have other severe SLE features, which includes lupus nephritis, need to be treated with IV cyclophosphamide or mycophenolate. In our case, the patient was given extra immunosuppressive therapy with ciclosporin after receiving excessive-dose glucocorticoids. She reported much better improvement in her signs and symptoms on 6 month-to-month to follow up.

There aren’t any recommendations concerning the use of immunosuppressive agents combined with glucocorticoids for lupus enteritis sufferers. In clinical exercise, a comprehensive evaluation of the patient’s complications, which includes lupus nephritis, neuropsychiatric lupus, and other important organ damage, is important to guide treatment decisions, including combined immunosuppressive remedy. About half of LEn sufferers also had active lupus nephritis, some others had neuropsychiatric lupus, autoimmune hemolytic anemia, diffuse alveolar hemorrhage, and other complications. therefore, more than half of patients with LEn obtained glucocorticoids in combination with cyclophosphamide. About 20.9% of these patients received mycophenolate with glucocorticoids, and 16.3% received glucocorticoids alone. death from LEn is uncommon. Our patient had recurrent abdomen signs after glucocorticoids by myself. She was rapidly relieved by high-dose glucocorticoids and continued long term treatment with low-dose steroids and cyclosporin.

Even though it is a potentially severe manifestation, most patients had resolution of signs and symptoms with adequate treatment. Intestinal perforation and demise have been rarely reported. If perforation happens because of lupus enteritis, surgical intervention is needed emergently. Early surgical intervention instead of steroid therapy as a treatment for lupus enteritis has been advocated but does no longer seem legitimate in view of high steroid responsiveness, reversibility and limited morbidity and mortality. Immunosuppressive agents can resolve lupus enteritis, but recurrence is common. Maruyama et al. confirmed that LEn was likely to recur and reported that 29% of their LEn sufferers experienced recurrence. Those authors advised that bowel wall thickness exceeding 9.0 mm can be a predictor of recurrence, additionally they stated that among the five patients with recurrence, 2 patients initially obtained glucocorticoids alone. Hydroxychloroquine, azathioprine, and mycophenolate mofetil might be taken into consideration for long-term maintenance treatment, although it is unclear whether recurrence can be avoided.

The prognosis is commonly excellent for sufferers with lupus enteritis, given its good response to steroids. despite the fact that, it is nevertheless imperative to identify and adequately treat this disease manifestation in a timely manner, as it may have a mortality of 2.7%. Lupus enteritis is estimated to recur in up to 23% of cases, which correlates with a decrease cumulative dosage of prednisone and a shorter duration of treatment. Patient has been given cyclosporin (Sandimmun”) 25 mg two times every day after the first follow-up meeting from the second one hospitalization. After 6 months of therapy with cyclosporin and 4 mg of methylprednisolone, the patient did not have a recurrence of signs and symptoms. The patient feels satisfied with the treatment she has been receiving because there has been no recurrence of the signs and symptoms during the 6-month observation.

CONCLUSION

Lupus enteritis as the only providing manifestation of active SLE is very uncommon. they have the potential for severe complications or even death. A high index of suspicions is important to differentiate disease activity from infection or other secondary reasons. Diagnosis of lupus enteritis requires a combination of high clinical suspicion from signs and symptoms, laboratory examination, and imaging. Abdomen CT showed the diagnosis of lupus enteritis in our patient. The result from the colonoscopy and biopsy examination additionally supported the diagnosis.

Timely administration of excessive-dose glucocorticoid therapy is effective and may improve the prognosis of these sufferers. whether or not to combine a glucocorticoid with an immunosuppressive agent requires comprehensive attention of the comorbidities of individual LEn sufferers. More high-quality registration research are needed to recognition on this uncommon complication of SLE. Excessive-dose steroids had been an efficient initial treatment for this moderately severe lupus enteritis patient. Our patient has remained in remission on methylprednisolone 4 mg every day and cyclosporine 25 mg twice every day.

INFORMED CONSENT STATEMENT

Informed written consent was obtained from the patient to publish this report and any accompanying images.

AUTHOR CONTRIBUTIONS

Fitria Yulistiarwati was the patient’s physician and contributed to manuscript drafting and writing. Awalia, the patient’s rheumatologist, reviewed the literature and is responsible for revising the manuscript.

CONFLICT-OF-INTEREST STATEMENT

The authors declare that they have no conflict of interest.

FUNDING STATEMENT

All authors have declared that no financial support was received from any organization for the submitted work.

REFERENCES


