



# Steroid Induced Acute Pancreatitis in a case of Systemic Lupus Erythematosus: A Rare Challenging Entity

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**Keywords:** Pancreatitis; Steroids; Steroid induced; Acute pancreatitis.

## Abstract

**Background:** Steroid induced acute pancreatitis can be challenging to diagnose leading to fatal complications. There are very few reports about it in the literature. Careful review of history and exclusion of other common diagnosis on time is crucial to prevent further complications. Withdrawal of the used steroid and conservative management drastically improves the patient's clinical condition. Here we report a case of a young patient with steroid induced pancreatitis following the administration of intravenous steroid.

**Objective:** This report here shows a rare case of steroid induced acute pancreatitis which adds to the existing literature. We also aim to highlight the importance of its timely diagnosis and management.

**Method:** A thirteen-year female with Systemic Lupus Erythematosus Nephritis II, presented with multiple rashes and fever. On examination, there were multiple erythematous, and circular plaques over multiple areas of face and oral cavity. Laboratory findings showed raised ~~increased~~ ESR, low C3 and C4, positive Anti dsDNA, and ANA with hemoglobin 9.5 mg/dl and platelet counts of 100000/ml. She was diagnosed with SLE Nephritis Class II with mucocutaneous and hematological flare and started on intravenous methylprednisolone pulse therapy.

**Result:** During her second pulse dose, she experienced acute pain abdomen with multiple episodes of vomiting. Her lipase and amylase level were highly elevated. USG Abdomen and CT abdomen revealed radiological findings suggestive of acute pancreatitis. Other possible causes were excluded. Steroid - induced acute pancreatitis was considered and intravenous steroids were discontinued, and conservative management for acute pancreatitis was started. The patient improved symptomatically within 48 hours, with decreased biochemical markers. The patient was discharged with low dose oral steroid, with the plan to slowly taper **its** dose.

**Conclusion:** This case reports a rare case of steroid induced acute pancreatitis and create awareness in clinicians about importance of careful medical review and its timely management.



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## Introduction

Acute pancreatitis is defined as the inflammation of the pancreas; it is characterized by symptoms of abdominal pain radiating to the back, fever, nausea, and vomiting [1]. Drugs are a relatively rare cause of acute pancreatitis, with an estimated incidence of 0.1-2%. Only a few cases of steroid induced pancreatitis have been reported in the literature. However, due to its rarity, it can be challenging to make an early diagnosis. The majority of cases are mild, but severe and even fatal cases may occur, thus making its identification critical [2,3]. However, the diagnosis of steroid-induced pancreatitis is challenging requiring careful review of the medical history and exclusion of other possible etiologies [4].

Here, we report a case of acute pancreatitis induced by intravenous steroid in a young patient with Systemic Lupus Erythematosus (SLE) Nephritis class II, with a mucocutaneous and hematological flare.

## Case presentation

A thirteen-year female with biopsy proven SLE Nephritis Class II for 8 months presented, with rashes over facial region, oral ulcers, and fever for ten-days duration, with poor compliance to oral medications. She also complained of pain over right ankle joint for 5 days with history of hair loss.

At the time of presentation, she was febrile with other vital signs normal. On local examination, there were multiple erythematous, and circular plaques with area of erosion and scaling over malar area and forehead, and multiple erosions present over roof of oral cavity, upper mouth and corner of mouth. Other systemic examinations were normal.

Initial laboratory findings showed raised transaminases, decreased albumin, and increased ESR. Complete blood count showed hemoglobin 9.5 mg/dl with platelet counts of 100000/ml. C3, C4 were low and Anti dsDNA, ANA were positive. She was diagnosed with SLE Nephritis Class II with mucocutaneous and hematological flare.

In view of the flare, she was pulsed with methylprednisolone (20mg/kg/day). During the second pulse therapy, she experienced pain in epigastric and periumbilical region, associated with multiple episodes of vomiting. Her lipase level was found to be highly elevated to 4173.70 U/L and serum amylase was found to be 1915 U/L. USG Abdomen revealed bulky pancreas with heterogenous echotexture with peripancreatic collection within, likely to be acute pancreatitis with minimal to mild ascites.

CT of the abdomen was performed which revealed bulky pancreas with antero-posterior diameter at the level of body of pancreas ~ 35mm with peripancreatic fluid collection and minimal bilateral fluid collection (Figure 1,2,3).

Other usual causes of acute pancreatitis were excluded. No gallstones could be visualized, both serum calcium and triglyceride levels were within the normal range. Steroid-induced acute pancreatitis was considered based on history, imaging study results, and biochemical markers. Intravenous steroids were discontinued, and management for acute pancreatitis was started as per standard protocol. She was kept at a stress dose steroid of intra venous hydrocortisone at 50 mg/m2/day, she received iv. Antibiotics piperacillin and tazobactam along with



**Figure 1:** Computed Tomography of abdomen and pelvis without contrast showing bulky pancreas with black arrow.



**Figure 2:** Computed tomography of abdomen and pelvis without contrast showing peripancreatic fluid collection with white arrow.



**Figure 3:** Computed tomography of chest showing minimal bilateral pleural effusion with basal atelectasis.

metronidazole, iv. Fluids and kept nil per oral with nasogastric drainage. The patient improved symptomatically within 48 hours, and gradually oral feeding was initiated. The lipase level decreased to 690.4 U/L and amylase to 420 U/L after the third day of stopping high dose steroid. The patient was discharged with low dose oral Prednisolone 30 mg/day on 10th day of admission, to control the ongoing disease activity, with the plan to slowly taper its dose.

## Discussion

Our case presents a young female with SLE Nephritis II with mucocutaneous and hematological flare for which intravenous steroid was started. She developed the symptoms of acute pancreatitis during the second dosing based on the clinical and lab parameters. The patient showed immediate clinical improvement following cessation of the steroid and the management of acute pancreatitis. Our case demonstrates the relation between steroids and acute pancreatitis and highlights the value of timely diagnosis and intervention.

Acute Pancreatitis is a serious condition with significant potential morbidity and mortality [5]. While mild acute pancreatitis carries a mortality of <1%, mortality rates for severe pancreatitis can reach as high as 30% [6]. Gallstone disease and alcohol abuse predominate in western countries and together are responsible for 70-80% of all cases. Other causes include hypercalcemia, hypertriglyceridemia, scorpion bite, tumor, drugs etc [7].

The mechanism by which steroid might induce acute pancreatitis is unknown; however, might be related to the alteration of lipid and calcium metabolism, the known systemic effect of corticosteroid; another mechanism found after injecting rabbits with steroids hypothesized that corticosteroids might obstruct small pancreatic ductules by leading to increased viscosity of pancreatic secretions, resulting in pancreatic changes. These changes included reduced basophilia, vacuolization of acini, peripancreatic fat necrosis, and hyperplasia of the islets of Langerhans [8]. The alteration of pancreatitis follows dose dependent pattern after corticosteroid regimen. Laboratory pancreatic alterations appear to be induced within days after pulse corticosteroid administration whereas acute pancreatitis develops within 4-14 days of the initial exposure to the oral agent [9,10].

A detailed evaluation of the patient's medical history and exclusion of other probable etiologies confirmed the diagnosis of steroid-induced pancreatitis. Discontinuation of the steroids and supportive care usually resolve the underlying acute pancreatitis and improve the patient's condition [11]. Complications include local and systemic ones, which can present as pancreatic pseudocyst, pancreatic necrosis and infection, chronic pancreatitis, and multiorgan failure [12].

## Conclusions

Steroids are commonly used in hospital settings for various condition, and often overlooked while making a diagnosis of pancreatitis in clinical setting of acute abdomen. Careful medical history is imperative to consider the diagnosis of steroid induced pancreatitis following exclusion of other common diagnosis. A timely discontinuation of the offending steroid along with other supportive care can drastically improve the patient's condition and also prevent complications.

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