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SLE AND ACUTE PANCREATITIS: A CASE REPORT

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Abstract

Introduction: Systemic lupus Erythematosus (SLE) is an auto-immune disease with multisystem involvement. Gastrointestinal involvement is fairly common in patients of SLE. Acute pancreatitis is a rare presenting feature among patients with SLE. SLE related pancreatitis has an annual incidence of 0.4-1.1 per thousand patients, occurring in 0.5-0.9% of patients with SLE. Case description: A 24-year-old male, presented to the emergency of Sharda Hospital, Greater Noida with complaints of vomiting and pain in abdomen for 2 days, dysphagia and fever since 1 month, oral ulcers since 2 months. At the time of admission patient was treated as a case of Acute pancreatitis with AKI. However, as more investigations became available and patient showed no significant clinical improvement an ANA (IFA) was sent. ANA was moderately positive and the ANA profile was strongly positive for Anti SM antibody, Ds DNA antibody and RNP-Sm antibody. Patient met the EULAR/ACR criteria for SLE and was treated with systemic steroids (methylprednisolone). Patients clinical condition improved and his liver functions and amylase/lipase normalized. Patient was discharged in satisfactory condition after a 2 week stay on HCQs and oral steroids. At 4 week follow up patient remained asymptomatic albeit a few occasional oral ulcers. Conclusion: SLE associated acute pancreatitis is a rare but known complication of SLE. In a patient of acute pancreatitis without any apparent cause SLE should be evaluated in presence of supporting clinical features like leukopenia, rash, ulcers etc. A strong index of suspicion should be maintained in such patients as they have a higher mortality and morbidity.

Keywords: SLE, Acute Pancreatitis, Gastrointestinal.

INTRODUCTION

Systemic lupus Erythematosus (SLE) is an auto-immune disease with multisystem involvement. The wide range of symptoms and manifestations associated with SLE are primarily due to development of auto antibodies. SLE is diagnosed based on the presence of clinical features and circulating auto antibodies. EULAR/ ACR published the latest criteria for diagnosis and classification of SLE in 2019. The EULAR/ACR criteria have a specificity of 93.4% and sensitivity of 96.1%, as opposed to 93.4% specificity and 82.8% sensitivity of the 1997 ACR criteria, and 83.7% specificity and 96.7% sensitivity of the 2012 Systemic Lupus International Collaborating Clinics (SLICC) classification criteria.1 Gastrointestinal involvement is fairly common in patients of SLE with studies estimating the incidence of GI involvement to be around 50%. Dyspepsia (72.7%), Heartburn (68.1%) and bloating (63.8%) are the three most common GI complaints found in patients with SLE. Abdominal pain had a prevalence of 19.2-23.5%.2,3 Acute pancreatitis is a rare presenting feature among patients with SLE with and prevalence of 0.7-4%. SLE related pancreatitis has an annual incidence of 0.4-1.1 per thousand patients, occurring in 0.5-0.9% of patients with SLE. 4,5,6,7,8

We report a case of a young male from western Uttar Pradesh who presented to the emergency with complaints of vomiting, abdominal pain and oral ulcers. He was ultimately diagnosed as a case of SLE.

Case Description

The patient gave his informed consent before writing of this article.

Patient a 24-year-old male from Jalpura, Greater Noida presented to the emergency of Sharda Hospital, Greater Noida with complaints of vomiting and pain in abdomen for 2 days, dysphagia and fever since 1 month, oral ulcers since 2 months. On presentations his vitals were BP- 114/72 mmhg, PR—104/min, Temp—101F, RBS—126 mg/dl, SpO2—98%, GCS was 15/15. On general examination patient had a faint hyperpigmented blackish rash present over the cheeks and bilateral ears. Oral cavity examination showed multiple areas of ulcerations and erosions were present over bilateral buccal mucosa and the base of the tongue. He also had mild pallor. Per abdomen examination revealed tenderness in the epigastric region and no other significant abnormality. Patient worked as a shopkeeper and had no history of alcohol or tobacco consumption. He did provide history of taking Unani medications from a local practitioner for his oral ulcers and dysphagia.

His investigations are as follows.

Haemoglobin	11.3	Bilirubin total	1.84	Urea	62.1
Total Leucocyte Count	2130	Bilirubin direct	1.52	Creatinine	1.5
DLC	P74, L19, M7, E0	SGOT	1080	Na/K/Ca	132/5/ 7.2
MCV	70	SGPT	667	Amylase	351
Platelets	46,000	ALP	142	Lipase	1477
ESR	09	Protein total	6.1	TSH	5.41
CRP	2.5	Albumin	2.9	Retic count	0.4%
Rheumatoid Factor	8	HIV	Non-Reactive	HBsAg	Non-reactive
Anti HCV	Non-reactive	ANA by IFA	1:320 +, Coarse speckled pattern	GBP	Pancytopenia
USG Abdomen	Miled hepatosplenomegaly, mild free fluid in abdomen	Dengue	Negative	Smear for Malaria	Negative
Urine albumin	+	Urine routine	NAD	PT/INR	14.7/1.08
Serum Ferritin	>1000	Serum Vit B12	827	Serum Iron	48
UACR	287	IgM Anti HAV	Negative	SSA-RO	Negative
TC	81	IgM Anti HEV	Negative	SSB-La	Negative
LDL	32	C3	26.4	Sm IgG	Positive (>200)
HDL	12	C4	3.3	RNP-Sm IgG	Positive (175)
TG	185	IgG		U1-SnRNP	Negative
Cortisol	25.3	SMA		Jo-1	Negative
ACTH	<5			Centromere	Negative
CECT abdomen	Bulky head of pancreas with fat stranding			Scl-70	Negative
				Ds-DNA	Positive (155)

Initially at the time of admission patient was treated as a case of Acute pancreatitis with AKI. However, as more investigations became available and patient showed no significant clinical improvement an ANA (IFA) was sent. ANA was moderately positive and the ANA profile was strongly positive for Anti SM antibody, Ds DNA antibody and RNP-Sm antibody. Patient met the EULAR/ACR criteria for SLE and was treated with systemic steroids (methylprednisolone). Patients clinical condition improved and his liver functions and amylase/lipase normalized. Patient was discharged in satisfactory condition after a 2 week stay on HCQs and oral steroids. At 4 week follow up patient remained asymptomatic albeit a few occasional oral ulcers.

DISCUSSION

SLE in historical context was initially considered to be a disease which primarily affects the skin. In fact, the term Lupus is Latin for wolf and erythematosus is ancient Greek for “redness of the skin”.9 However, over the centuries as the understanding of the disease grew, it was Sir William Osler who added the term systemic to lupus erythematosus as he found that it not only involves the skin but other organs as well.10 In the decades of 1920-30, research carried out at Mayo Clinic USA demonstrated how SLE affected the kidney, heart, and lung tissue and this was attributed to the presence of LE cells. 10 Since these early days we have come a long way in the understanding of this disease and the latest EULAR/ACR criteria takes into account both the systemic and the dermatological findings along with presence of immunological abnormalities.1 However, Gastrointestinal abnormalities, except oral ulcers, are not part of the criteria to diagnose SLE.

SLE is usually seen in women of age bearing age. It predominantly affects females with a male : female ration of 1:9. The risk of developing SLE decreases post-menopause but its still twice of that in males. Although incidence of SLE is less in males when compared to females, its more severe and presents with more symptoms.11 GI involvement in SLE is well documented and is fairly common with an incidence of around 50%. Abdominal pain is a common complaint with a prevalence of 19.2% to 23.5%. 4,5 Acute pancreatitis is a rare presenting feature among patients with SLE with and prevalence of 0.7-4%. SLE related pancreatitis has an annual incidence of 0.4-1.1 per thousand patients, occurring in 0.5-0.9% of patients with SLE. 4,5,6,7,8 Abnormal liver enzymes, as seen in our patient, although not considered a significant finding in SLE is present in around 60% of the SLE patients.12 The common cause of elevated liver enzymes in patients with SLE are use of Hepatotoxic drugs, coincident viral hepatitis and NAFLD.13 Occurrence of autoimmune hepatitis (AIH) and SLE is rare and not usually reported in literature. 14,15

SLE related pancreatitis has not been widely reported in literature. This is likely due to underdiagnosis, where abdominal pain is often assumed to be because of acute gastritis. In such patients pancreatitis might usually resolve on its own with supportive treatment and corticosteroids.16 Studies have shown that patients with SLE related pancreatitis have a higher mortality and morbidity when compared to their non-lupus counterparts. This may be due to presence of other co-morbidities associated with SLE like lupus nephritis.17,18 In cases of SLE patients presenting with Acute Pancreatitis the usual causes of pancreatitis such as, trauma, alcohols abuse, hypertriglyceridemia, hypercalcaemia, gall stones, drugs, etc should be methodically ruled out.19,20 its after ruling out these causes that a diagnosis of lupus pancreatitis can be established. Various mechanisms have been postulated by which SLE may

lead to Pancreatitis such as organ ischemia secondary to hypovolemia or severe hypotension, atherosclerotic disease, vasculitis, pancreatic artery micro-thrombosis related to antiphospholipid antibody syndrome, intimal thickening/proliferation, presence of anti-pancreatic antibodies, organ inflammation due to T-cell infiltration, immunocomplex deposit, and complement activation, as well as viral infection due to the patient's chronic immunosuppressed state.16,18,19,20,21,22,23,24,25 In a study conducted in America, 63 patients in a sample size of 1811 patients has SLE related pancreatitis (3.5%). It was found that a triglyceride level of 200mg/dl or more had a positive correlation with the development of SLE associated pancreatitis.26

In our patient, we diagnosed SLE induced pancreatitis after ruling out he the common causes of Acute Pancreatitis such as gall stones, ethanol abuse, drug induced, hypercalcaemia, hypertriglyceridemia etc. After careful clinical examination and history was taken and the initial investigations showed both liver and pancreatic involvement a possibility of autoimmune condition was considered and an ANA was sent. Presence of rash over cheeks and ears, oral ulcers, leukopenia, thrombocytopenia were suggestive of SLE and once ANA was positive (1:320) we used the EULAR/ ACR criteria for SLE and the patient was Diagnosed as a case of SLE pancreatitis. Deranged liver function test was also an additional finding in our patient. We ruled out viral, autoimmune and ethanol as cause for this. However, a history of intake of Unani medication may be the likely cause of liver function derangement. Many studies have shown the hepatotoxic effects of Ayurvedic, unani or herbal medication on liver.27 In our case this seems to be the most likely cause of deranged liver profile.

CONCLUSION

SLE associated acute pancreatitis is a rare but known complication of SLE. In a patient of acute pancreatitis without any apparent cause SLE should be evaluated in presence of supporting clinical features like leukopenia, rash, ulcers etc. A strong index of suspicion should be maintained in such patients as they have a higher mortality and morbidity. Steroids are helpful in such cases and help in resolution.

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