IJBPAS, February, 2023, 12(2): 528-540

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

A RETROSPECTIVE CASE SERIES ON SLE {SYSTEMATIC LUPUS ERTHYMATOSUS} IN 2 SIBLINGS OF SAME FAMILY

M. MEGHANA GOUD[1*], V MOHAN GOUD[2] AND M. KRANTHI KUMAR[3]

1: Pharm. D. 4th Year Joginpally B R Pharmacy College, Hyderabad, Telangana

2: Department of Pharmaceutical Analysis, Joginpally B.R. Pharmacy College, Hyderabad, Telangana

3: Sr. Executive, Global Pharmacovigilance department, Aurobindo Pharma limited

***Corresponding Author: M. Meghana Goud: E Mail: mallammeghana1998@gmail.com**

Received 29th March 2022; Revised 29th April 2022; Accepted 19th July 2022; Available online 1st Feb. 2023

https://doi.org/10.31032/IJBPAS/2023/12.2.6811

ABSTRACT

Patient: female patient at age 23 ♀

Department / ward: rheumatology

Chief complaints: fever since 8 months, polyarthritis since 8 months, anemia

Final diagnosis: systematic lupus erythematosus with nephritis

Background:

Systemic lupus erythematosus is characterized by multiorgan involvement and the presence of autoantibodies (SLE). Because of the vast spectrum of symptoms and signs, SLE's development and organ involvement are unpredictable. SLE is characterized by cytopenia's such as anemia, leukopenia, and thrombocytopenia. Here the patient has lupus with nephritis and also thrombocytopenia.

CASE 1: We report a case of a young Indian female who presented with severe anaemia, fever since 8 months and polyarthritis since 8 months and also leukopenia, subsequently diagnosed as TB and several many other infections and then eventually SLE with nephritis.

CASE 2: The other case concerns the above case's sibling, a 47-year-old woman who presented with a plantar psorium on the palms and feet since 5yrs, as well as an uncontrollable fever. These indications were initially misdiagnosed as psoriasis, dengue fever, and other, but it was eventually determined to be SLE with thrombocytopenia.

Conclusion: SLE can cause nephritis, which is a complication. It's critical to understand this link because early detection and treatment are critical. Corticosteroids and immunosuppressants have been

demonstrated to be effective in treating SLE and the nephritis that comes with it. Finally, the patients are still receiving treatment and are alive, even though with some or the other complications.

Keywords: SLE, Thrombocytopenia, Nephritis, Discoid Rash, Fatty Liver, Covid 19

INTRODUCTION:

REASON FOR REPORTING CASE: our interest in reporting this case is systematic lupus erythematosus is a rare autoimmune disease, and especially it is been found in 2 siblings of same family. And the patient reported in this case is presented with many complications and still surviving, so this made us to report the uniqueness of the case and to show the novelty of the disease.

SYSTEMATIC LUPUS ERTHYMATOSUS:

SLE is an immunological disease which affects the skin, joints, brain system, and kidneys, among many other organs. Women with childbearing age and members of specific racial groups will more likely get the disease **[1]**.

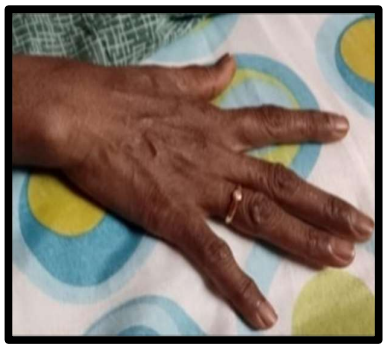
SLE is strongly linked to rare, inherited single-gene complement deficits, but the disease is inherited polygenically in the majority of patients. Immune dysregulation at the level of cytokines, T cells, B cells, and macrophages may result from genetic interactions with environmental factors, such as UV radiation exposure, Epstein–Barr virus infection, and hormonal influences. Because of the variability of SLE, diagnosis is mostly clinical and remains difficult. Clinical trials have benefited from classification criteria, although only one medicine (belimumab) has been authorized for treatment in SLE in the last 60 years.[1] Newer medications such as mycophenolate mofetil and glucocorticoid-sparing regimens have helped to mitigate the detrimental side effects of older drugs such as cyclophosphamide and glucocorticoids. Consequently, the detrimental effects of renal and neurological involvement, as well as late identification, have stifled future advancement. The increased risk of early cardiovascular disease associated with SLE, as well as the risk of infection aggravated by immunosuppressive medication, add to the burden. Treatment-resistant illness and symptoms like weariness continue to be obstacles. Newer medicines may give a better chance of success, and advances in stem cell and genetic procedures may one day lead to a cure **[1]**.

CASE 1: {YEAR 2004}

Diagnosis: SLE with musculoskeletal manifestations with polyserositis with nephritis with HTN with myocarditis with anemia and UTI

CLINICAL FINDINGS: 25 year old female presented with c/o fever on & off

for 7 months, polyarthritis involving both wrists, both elbows and PIP, MCP joints, malar rash, breathlessness on exertion for 4months and skin rash which are pruritic and oral ulceration which were bleeding and painful 20 days back.



ON EXAMINATION: pt. is conscious, cooperative, oriented, pulse-90/min, BP-166/100mm of hg RS/CVS/ABD/CNS are clinically normal

INVESTIGATIONS: Hb: 9.0gm/dl ESR: 88mm/hr. CUE: SG-1025, Alb: +, pus cells: 6-8 C3: 35mg/dl, C4: 6mg/dl, urinary protein : 5.8mg/day, ANA: 1.8+, anti ds DNA 258+ urine culture *E.coli* sensitivity to amikacin, cefotaxime, ceftazidime, cefoperazone, cefpirome

RADIOLOGY: CXR: moderate cardiomegaly, Xray PNS: Haziness noted in bilateral pantal sines, possibility of sinusitis.

CARDIOLOGY: 2Decho global hypokinesia, moderate LV dysfunction, mild MR pericardial effusion.

HOSPITAL COURSE: evaluated for chronic inflammatory polyarthritis, breathlessness, HTN h/o fever and dark pigmentation over nose. She was found to be having SLE with proteinuria of 5.8gm in 24 hrs. nephrologist was consulted. Kidney biopsy was deferred because of associated UTI. She was given three pulses of methylprednisolone after treating the UTI. She also received first pulse of endoxan on 9/3/4

DOA: 02/03/2004

DOD: 10/03/2004

NEXT ADMISSION OF PATIENT

DOA: 26/3/04 **DOD:** 29/3/04

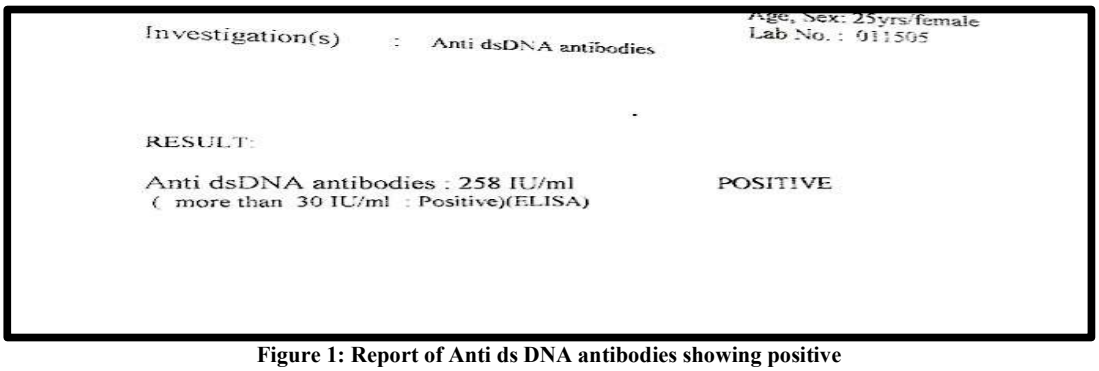
DIAGNOSIS: SLE with lupus nephritis on endoxan with myocarditis, endoxan toxicity {cutaneous GI}

CLINICAL FINFDINGS: 25years old female k/c/o SLE with nephritis with polyserositis with musculoskeletal manifestations. On cyclo pulse every 15 days received 2nd pulse patient started c/o pain in some difficulty in swallowing throat pain and angel of abdominal discomfort. She is having one episode of vomiting at 8.00am followed by two more. She is also having black stool since 20 days. She had 2-3 episode's of presymoge which improved on taking rest. No h/o

palpitations. She also reported that her teeth color is changing to black, even finger and toes.

HOSPITAL COURSE: admitted for management of cyclophosphamide toxicity in the form of hyperpigmentation of skin, blurring vision, vomiting and pain in throat. She responded to iv fluids, still having proteinuria 3+. And her renal parameters and electrolytes are normal.

DISCHARGE ADVICE: T. wysolone 10mg, T. pan 40mg, T. emset 4mg , T. enam 2.5mg.


Investigation(s) : Anti dsDNA antibodies

Age, Sex: 25yrs/female
Lab No. : 011505

RESULT:

Anti dsDNA antibodies : 258 IU/ml POSITIVE
(more than 30 IU/ml : Positive)(ELISA)

Figure 1: Report of Anti ds DNA antibodies showing positive

YEAR 2005: 28/9/05, 10/11/05, 17/11/05

c/o rt buttock pain hip movement normal and pain in mid thigh region

INVESTIGATIONS: C3: 32, C4: 8.6, ds DNA: 250IU/ml+ , Hb : 13.8 , TLC: 8500 , PLT: 2.0lkhs , ESR: 62mm/hr , plan for biopsy

Rx T. omnacortil 7.5mg, T. HCQS 200mg, T. azoran 50mg, T. supradyn, T. enam 2.5mg.

YEAR 2006: 23/1/06, 24/1/06, 7/03/06, 25/04/6, 29/6/06, 6/11/6, 7/11/6, 9/11/6

DIAGNOSIS: SLE with lupus nephritis, pyrexia, leucopenia{? Might be drug induced azoran

CLINICAL FEATURES: 26years old female SLE with lupus nephritis is on azoran since august 2004 admitted with c/o epigastric discomfort 1 day myalgias since 2 days fever 3 days back h/o dysuria increased frequency easy fatiguability, polyarthritis.

ON EXAMINATION: pt was c/c/c oriented pulse 80/min BP-110/70mmhg, RS/CVS/ABD/CNS clinically normal

INVESTIGATIONS: Hb: 12.60, PCV: 27.40, RBC: 4.40, MCV:83.60, MCH: 28.20, MCHC:33.70, TLC:2000 PLC:1,20Lkhs, ESR:20MM/HR, CUE: ALB:+, EPI CELLS:2-3, Puss cells: 2-3, SGPT:42.0

HOSPITAL COURSE: admitted for evaluation of fever 3 days back with increased frequency dysuria, epigastric discomfort, all her investigations reveal normal study except leucopenia{could be azathioprine induced} she was treated

symptomatically recovered hence discharged

DISCHARGE ADVICE: T. Omnacortil 7.5mg, T. HCQS 200mg, T. supradyn, T. osteocel, folinol, pan D 40mg

YEAR 2008: DOA: 5/2/08- 5/2/8

PRESENT ILLNESS; admitted in hsptl for MTP+laproscopic ligation. She had one previous delivery by caesarean section. The child is good she underwent medical termination of pregnancy by suction evaluation under GA. She also underwent laproscopic sterilization in same sitting. Right tubectomy was performed. Advised for tab. Cifran, tab dolo.

YEAR 2010: DOA: 30/11/10 – 08/12/10

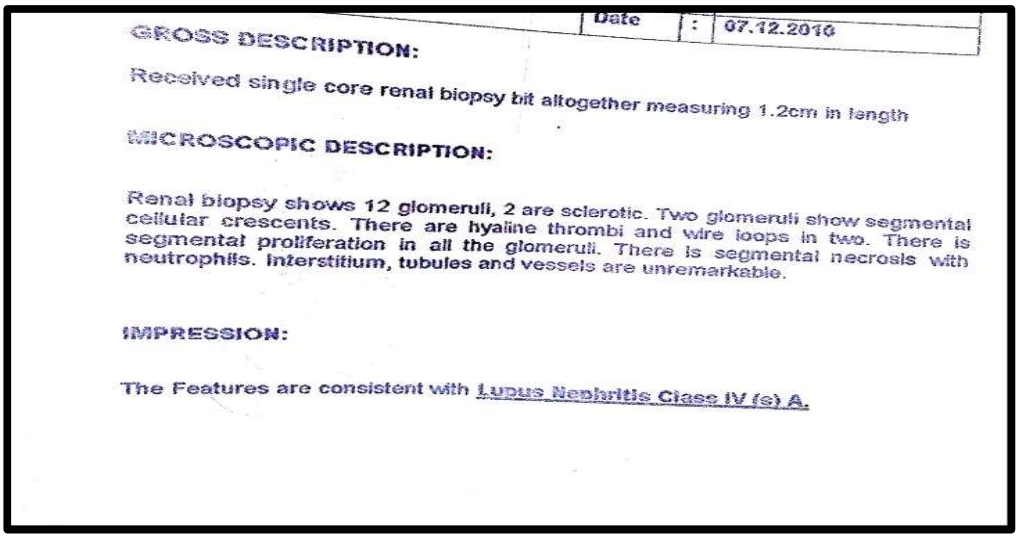
DIAGNOSIS: SLE LN post ELNT 2004 azoran, nephritis-flare

CLINICAL FEATURES: 24 year old female admitted known case of SLE since 2004 lymph node- post cyclophosphamide ELNT on azathioprine maintenance now admitted with h/o myalgia, arthralgias- 15 days, fatigue 15 days OP evaluation patient had active urinary sediment urinary protein 1gm drug h/o she was on azoran 100mg daily wysolone 2.5mg and losartan 50mg. past h/o diagnosed as SLE with nephritis in 2004, received cyclophosphamide ELNT last 6th pulse on 2/5/04 urinary protein was 5.8gm had myocarditis wysolone dose was increased to 40mg gradually tapered to 10mg.

ON EXAMINATION: pt. was c/c/c pulse 80/min, BP: 140/80mmhg RS/ CVS/ CNS/ ABD are clinically normal.

HOSPITAL COURSE: on evaluation pt. had active urine sediment 24 hr urinary protein 1.4gm renal biopsy was done.

DISCHARGE ADVICE: tab wysolone, tab goodvit, tab polybion, tab losartan, tab amlong, tab HCQS, tab mecalvit, tab septran, tab calsin.



Date : 07.12.2010

GROSS DESCRIPTION:

Received single core renal biopsy bit altogether measuring 1.2cm in length

MICROSCOPIC DESCRIPTION:

Renal biopsy shows 12 glomeruli, 2 are sclerotic. Two glomeruli show segmental cellular crescents. There are hyaline thrombi and wire loops in two. There is segmental proliferation in all the glomeruli. There is segmental necrosis with neutrophils. Interstitium, tubules and vessels are unremarkable.

IMPRESSION:

The Features are consistent with Lupus Nephritis Class IV (s) A.

Figure 2: Report of renal biopsy showing 12 glomeruli 2 are sclerotic

YEAR 2011 & 12: DOA: 29/12/11- 5/1/12

DIAGNOSIS: SLE, acute gastroenteritis now admitted in hospital with abdomen pain, loose stools, fever. Radiology: no sonological evidence of pancreatitis, small right adnexal cyst probably right ovarian cyst is seen in USG.

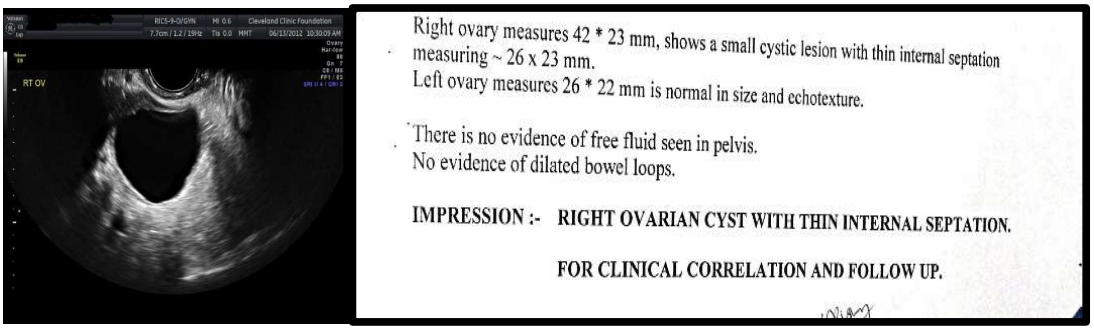


Figure 3: Report showing right ovarian cyst

YEAR 2016: c/o severe burning sensation in both eyes after started using SLE medication since 8-9 years c/o headache h/o ophthalmic consultation and they diagnosed as both eye RPE defect at macular area. Fundus examination within normal limits. Diagnosis both eyes POSTERIOR SUBCAPSULAR CATARACT -PSC steroid induced. OCT both eyes, AUTO FLUORESCENCE both eyes HVF both eyes advised for lubricant drops HVF 10-2pt

YEAR 2020 & 2021 & 2022: pt. 1st time visited to diabetology& endocrinology hypothyroid for 1st time denovo diabetes HTN -15 years irregular menstrual cycle h/o menorrhagia T3: 1.35, T4: 7.58, TSH: 10.130 advised for OGTT FBS; 111 PLBS: 147 HbA1C: 6.4 advised with the tab GLYCOMET 500mg BD.

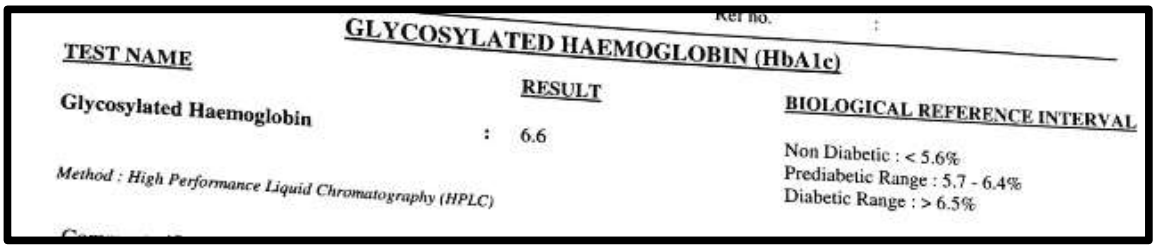


Figure 4: Report showing glycated haemoglobin HbAIc : 6.6

DIAGNOSIS: Bilateral atypical viral pneumonia COVID 19, SLE, TYPE 2 DM, HTN

A pt. 44 yrs old female came with complaints loose stools watery yellowish color since 4 yrs, fever, vomiting, since 1 day. On examination pt was c/c/c spo2 97% evaluated with all the investigations. Treatment given in hospital: inj lactoguard, inj metrogyl, inj nexpro, inj covifor, inj dexa, inj zofer, inj scorbix, inj clexane, inj actrapid, tab zincovit, tab HCQS, tab

azoran, cap lumia, cap doxylus, iv fluids. Review after 1 week FBS, PLBS, CBP, CRP, D dimer, Sr cr, LFT

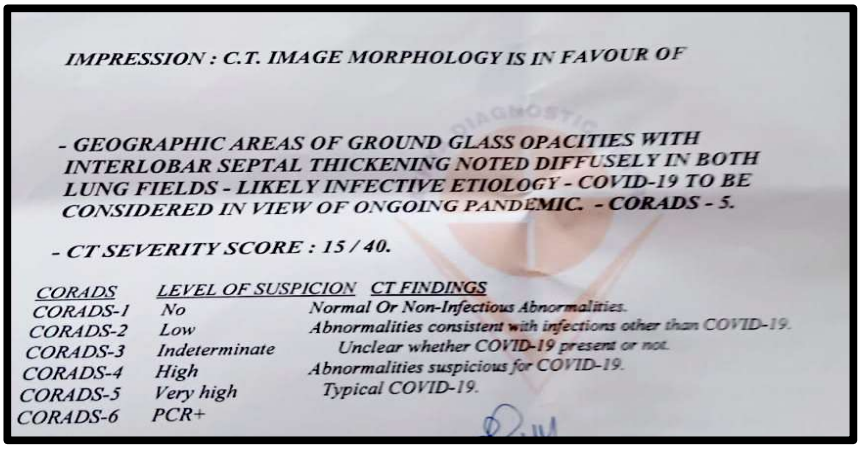


Figure 5: Report of COVID 19 showing CT score as 15/40

The patient in 2022 was still continuing the SLE medications recovered from covid 19 and good and healthy now.

CASE 2: 2016

DIAGNOSIS: psoriasis

CHIEF COMPLAINTS: A female patient at age of 42yrs has the chief complaints of rashes and silvery scales on the palms and feet and pigmentation over mouth with fever went to the dermatologist he then diagnosed as psoriasis and gave some lotions and creams to get rid of those scales or rashes, even pt had h/o ayurvedic medicine for 1 yr. skin biopsy is also done.

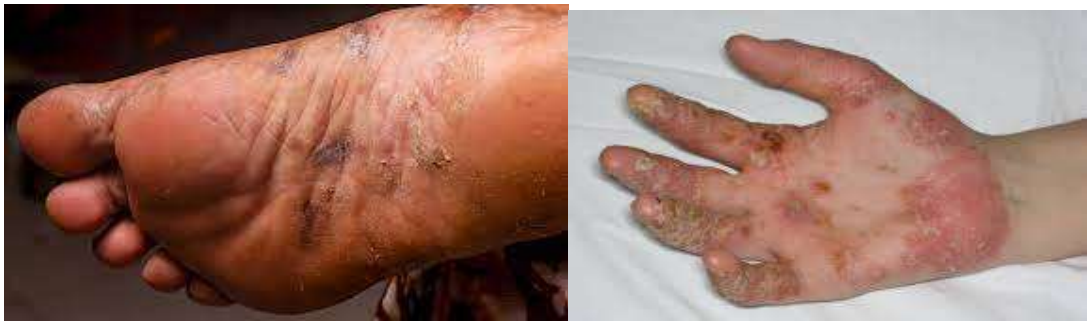


Figure 6: Plantar and palmar psorium of the patient

TREATMENT GIVEN FOR PSORIASIS: Mupirocin ointment, hydrocortisone cream, salicylic acid ointment, phototherapy,

However this became a misdiagnosis and patient had taken those psoriasis medicine at least for 3 yrs. Patient eventually had an unmanageable fever in 2020 which led to many lab examinations, where they discovered a drop in platelets to 90k, which was then diagnosed as dengue fever, as the dengue season was still ongoing. In this way many misdiagnosis happened.

YEAR 2021: A female patient of age 47 years is referred to a rheumatologist and she came with chief complaints of plantar and palmar psorium since 5 years, fever with chills no rash body aches, platelet count 1.6lakh due to dengue. Low grade fever, no fatigue, on & off joint pain, no oral ulcers, alopecia BP was found to be 120/90mmhg. On examination pt has rt ear focal areas, hyperpigmentation.

USG abdomen revealed GRADE 1 FATTY LIVER, chest Xray was found to be normal

PROVISIONAL DIAGNOSIS: SLE with thrombocytopenia, discoid rash, arthralgia, borderline nephritis

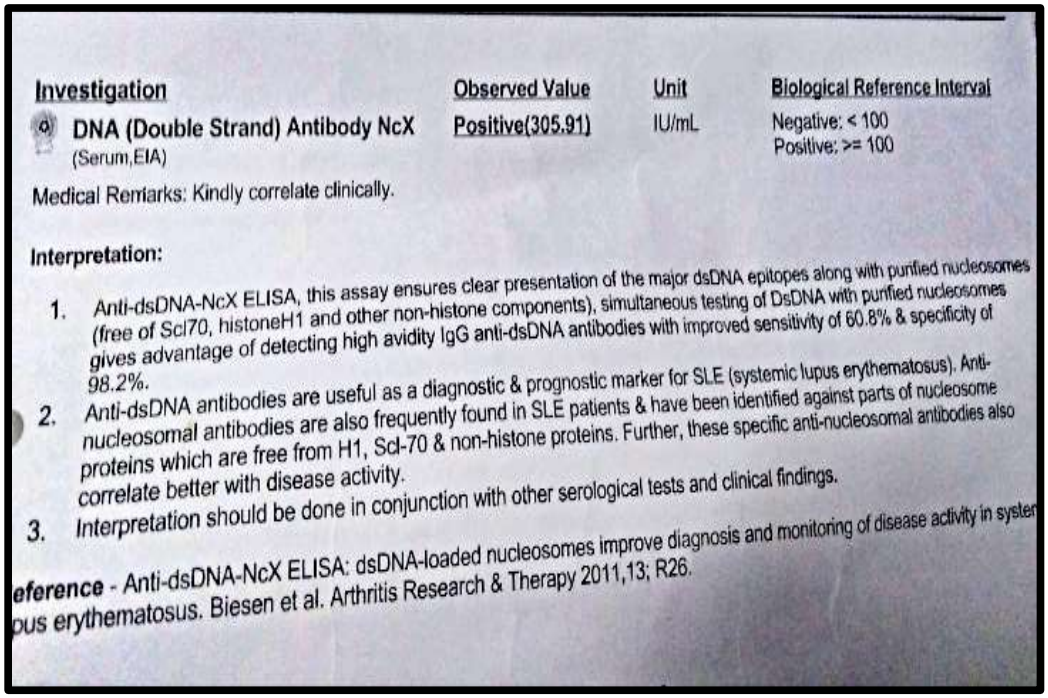
INVESTIGATIONS: Hb: 11.3, TLC: 5360, DLC: N55 L36, PLT: 98000, ESR: 45, S.cr: 1.2

RH factor: 2.31, CRP: 15.1, ANA +ve

ADVICE: dsDNA, C3, C4, spot urine protein, direct combs test, 2D echo, CBP, ESR

TREATMENT: T omnacortil – 20mg, T HCQS- 200mg, T caldikind plus, T MMF-36mg, review with reports after 4 weeks.

The abnormal reports of the patient are been placed below:



Investigation	Observed Value	Unit	Biological Reference Interval
DNA (Double Strand) Antibody NcX (Serum,EIA)	Positive(305.91)	IU/mL	Negative: < 100 Positive: >= 100

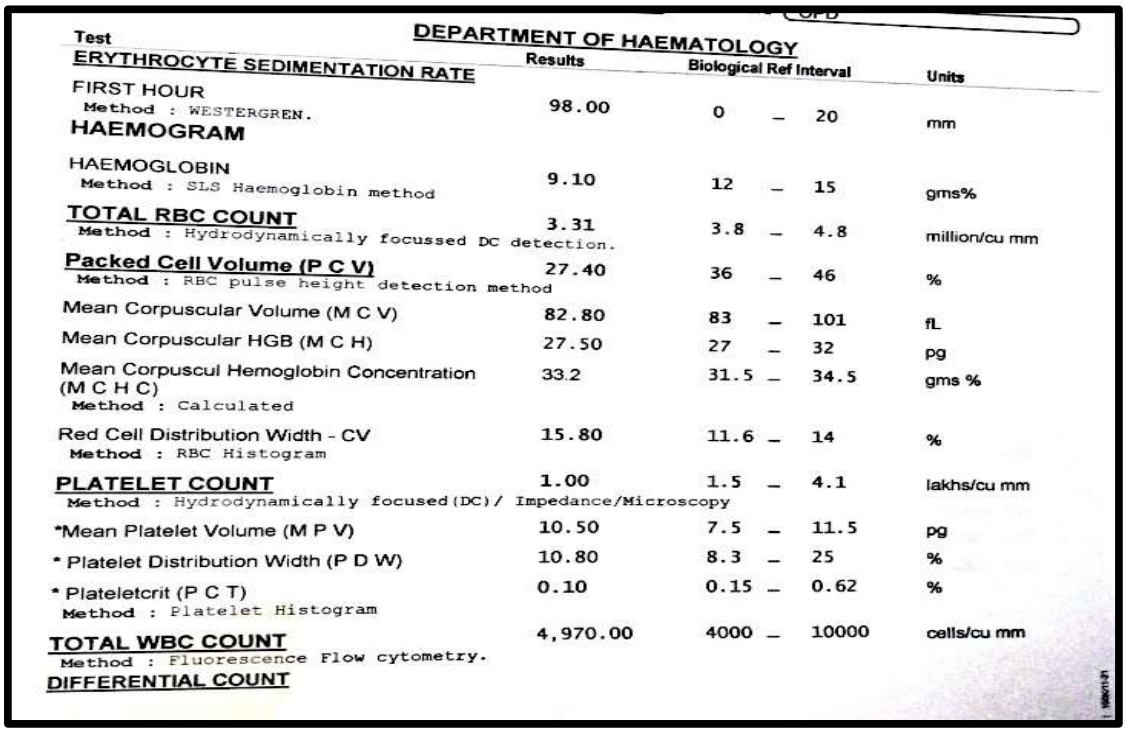
Medical Remarks: Kindly correlate clinically.

Interpretation:

1. Anti-dsDNA-NcX ELISA, this assay ensures clear presentation of the major dsDNA epitopes along with purified nucleosomes (free of Scl70, histoneH1 and other non-histone components), simultaneous testing of DsDNA with purified nucleosomes gives advantage of detecting high avidity IgG anti-dsDNA antibodies with improved sensitivity of 60.8% & specificity of 98.2%.
2. Anti-dsDNA antibodies are useful as a diagnostic & prognostic marker for SLE (systemic lupus erythematosus). Anti-nucleosomal antibodies are also frequently found in SLE patients & have been identified against parts of nucleosome proteins which are free from H1, Scl-70 & non-histone proteins. Further, these specific anti-nucleosomal antibodies also correlate better with disease activity.
3. Interpretation should be done in conjunction with other serological tests and clinical findings.

eference - Anti-dsDNA-NcX ELISA: dsDNA-loaded nucleosomes improve diagnosis and monitoring of disease activity in syste
ous erythematosus. Biesen et al. Arthritis Research & Therapy 2011,13; R26.

Figure 7: Double stranded DNA antibody was found to be positive 305.91

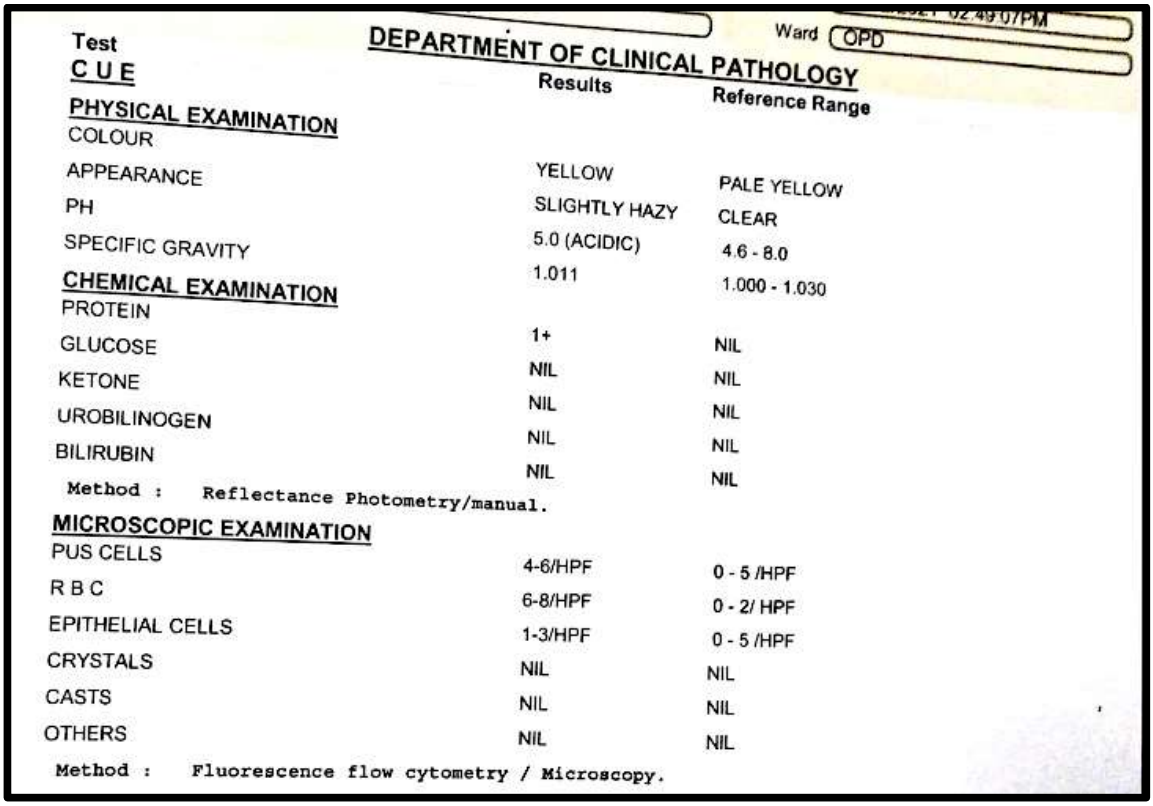


DEPARTMENT OF HAEMATOLOGY

Test	Results	Biological Ref Interval	Units
ERYTHROCYTE SEDIMENTATION RATE			
FIRST HOUR Method : WESTERGREN.	98.00	0 – 20	mm
HAEMOGRAM			
HAEMOGLOBIN Method : SLS Haemoglobin method	9.10	12 – 15	gms%
TOTAL RBC COUNT Method : Hydrodynamically focussed DC detection.	3.31	3.8 – 4.8	million/cu mm
Packed Cell Volume (P C V) Method : RBC pulse height detection method	27.40	36 – 46	%
Mean Corpuscular Volume (M C V)	82.80	83 – 101	fL
Mean Corpuscular HGB (M C H)	27.50	27 – 32	pg
Mean Corpuscul Hemoglobin Concentration (M C H C) Method : Calculated	33.2	31.5 – 34.5	gms %
Red Cell Distribution Width - CV Method : RBC Histogram	15.80	11.6 – 14	%
PLATELET COUNT Method : Hydrodynamically focused(DC)/ Impedance/Microscopy	1.00	1.5 – 4.1	lakhs/cu mm
*Mean Platelet Volume (M P V)	10.50	7.5 – 11.5	pg
* Platelet Distribution Width (P D W)	10.80	8.3 – 25	%
* Plateletcrit (P C T) Method : Platelet Histogram	0.10	0.15 – 0.62	%
TOTAL WBC COUNT Method : Fluorescence Flow cytometry.	4,970.00	4000 – 10000	cells/cu mm
DIFFERENTIAL COUNT			

Figure 8: Report of hematology showing abnormal ESR

Here in the CBP it was found that ESR was increased due the disease infection, and even the hemoblogin was decreased than normal, RBC count was decreased.



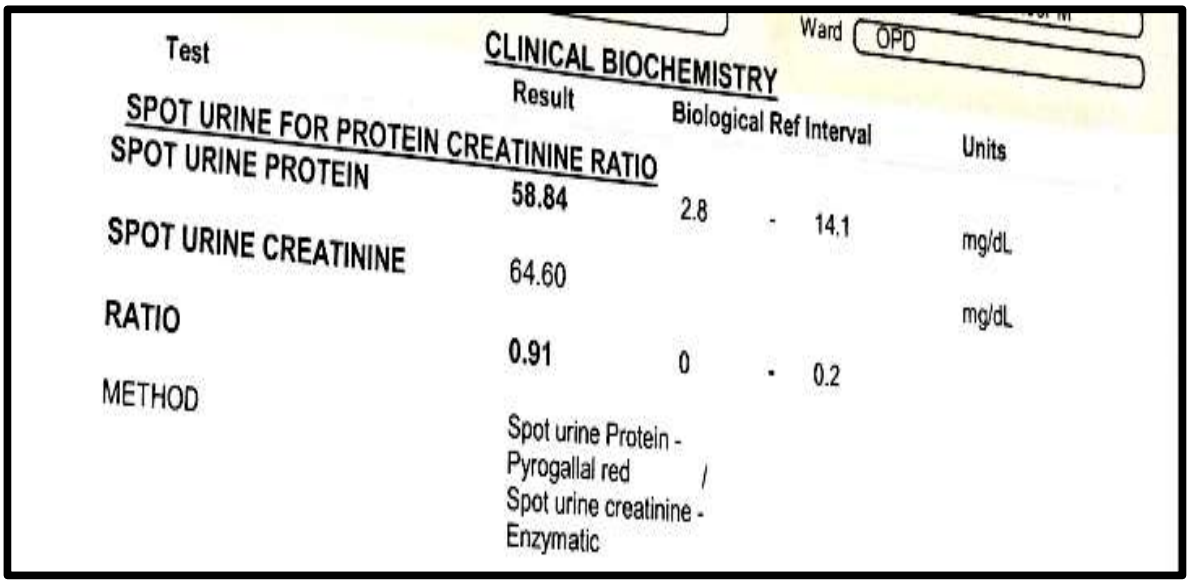
DEPARTMENT OF CLINICAL PATHOLOGY

Ward OPD

Test	Results	Reference Range
C U E		
PHYSICAL EXAMINATION		
COLOUR	YELLOW	PALE YELLOW
APPEARANCE	SLIGHTLY HAZY	CLEAR
PH	5.0 (ACIDIC)	4.6 - 8.0
SPECIFIC GRAVITY	1.011	1.000 - 1.030
CHEMICAL EXAMINATION		
PROTEIN	1+	NIL
GLUCOSE	NIL	NIL
KETONE	NIL	NIL
UROBILINOGEN	NIL	NIL
BILIRUBIN	NIL	NIL
Method : Reflectance Photometry/manual.		
MICROSCOPIC EXAMINATION		
PUS CELLS	4-6/HPF	0 - 5 /HPF
R B C	6-8/HPF	0 - 2/ HPF
EPITHELIAL CELLS	1-3/HPF	0 - 5 /HPF
CRYSTALS	NIL	NIL
CASTS	NIL	NIL
OTHERS	NIL	NIL
Method : Fluorescence flow cytometry / Microscopy.		

Figure 9: Report of pathology

Here in the complete urine examination the colour of urine was found to be yellow and the appearance was slightly hazy, and the protein showed 1+ and pus cells were 4-6, RBC were 6-8, wheras epithelial cells were found to be 1-3 all the changes says that the patient is having some sort of infection.



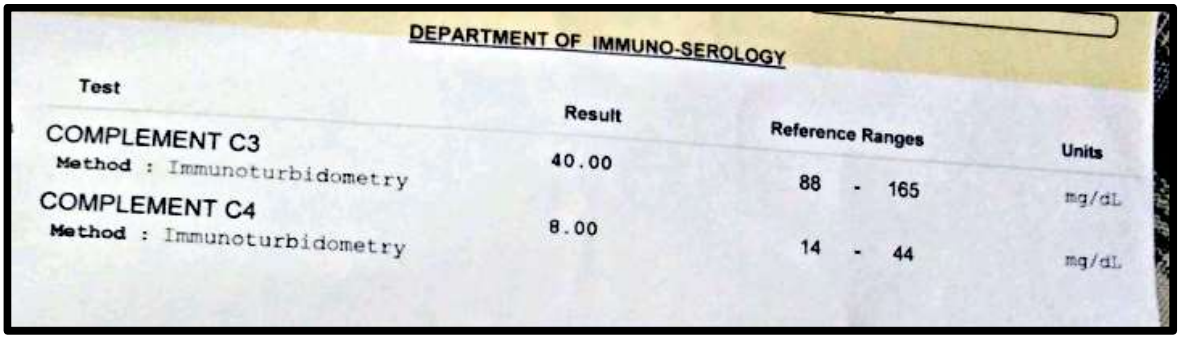
Ward OPD

CLINICAL BIOCHEMISTRY

Test	Result	Biological Ref Interval	Units
SPOT URINE FOR PROTEIN CREATININE RATIO			
SPOT URINE PROTEIN	58.84	2.8 - 14.1	mg/dL
SPOT URINE CREATININE	64.60		mg/dL
RATIO	0.91	0 - 0.2	
METHOD	Spot urine Protein - Pyrogallal red / Spot urine creatinine - Enzymatic		

Figure 10: Report of clinical biochemistry

Here the spot urine protein was increased than the normal because Proteinuria is a clinical measure of disease severity and kidney damage that is included in the activity and damage indices for lupus. Ie: protein in urine causes renal tubule and interstitial fibrosis.

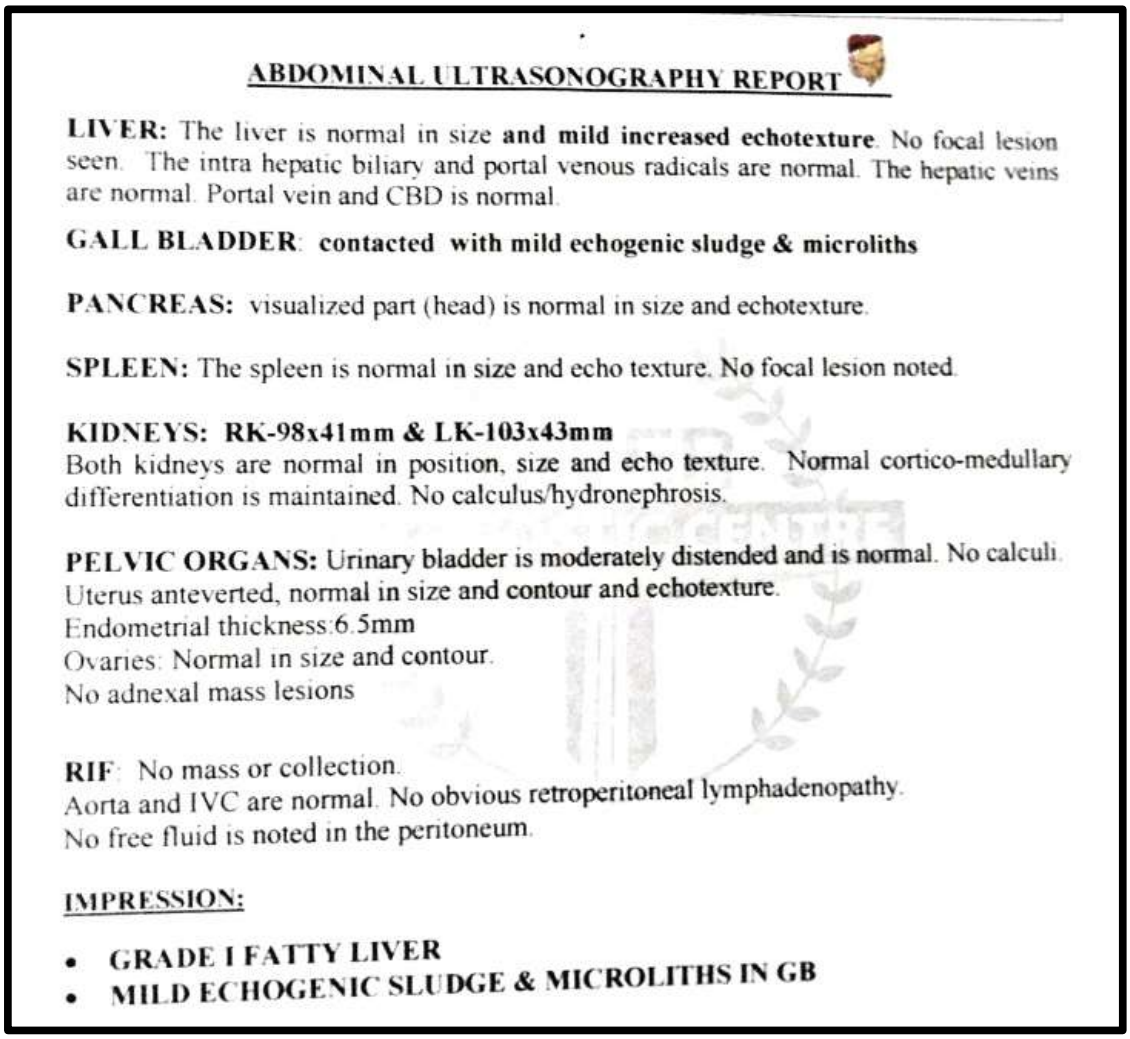


DEPARTMENT OF IMMUNO-SEROLOGY

Test	Result	Reference Ranges	Units
COMPLEMENT C3	40.00	88 - 165	mg/dL
Method : Immunoturbidometry			
COMPLEMENT C4	8.00	14 - 44	mg/dL
Method : Immunoturbidometry			

Figure 11: Report showing Immuno serology

C3 and C4 are indeed acute inflammatory reactants, implying that inflammatory events boost the rate of production. Likewise, the increased synthesis rate in SLE may compensate for the increased breakdown caused by upregulation.



ABDOMINAL ULTRASONOGRAPHY REPORT

LIVER: The liver is normal in size **and mild increased echotexture**. No focal lesion seen. The intra hepatic biliary and portal venous radicals are normal. The hepatic veins are normal. Portal vein and CBD is normal.

GALL BLADDER: **contacted with mild echogenic sludge & microliths**

PANCREAS: visualized part (head) is normal in size and echotexture.

SPLEEN: The spleen is normal in size and echo texture. No focal lesion noted.

KIDNEYS: RK-98x41mm & LK-103x43mm
Both kidneys are normal in position, size and echo texture. Normal cortico-medullary differentiation is maintained. No calculus/hydronephrosis.

PELVIC ORGANS: Urinary bladder is moderately distended and is normal. No calculi.
Uterus anteverted, normal in size and contour and echotexture.
Endometrial thickness:6.5mm
Ovaries: Normal in size and contour.
No adnexal mass lesions

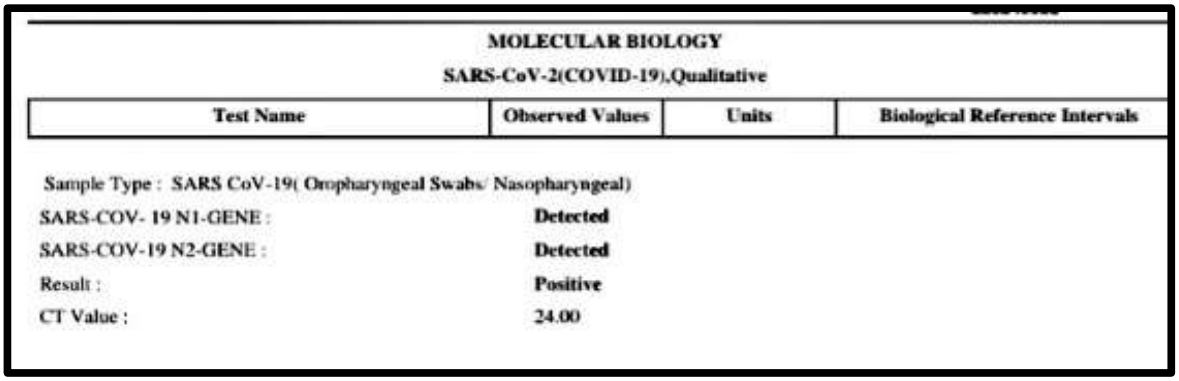
RIF: No mass or collection.
Aorta and IVC are normal. No obvious retroperitoneal lymphadenopathy.
No free fluid is noted in the peritoneum.

IMPRESSION:

- **GRADE I FATTY LIVER**
- **MILD ECHOGENIC SLUDGE & MICROLITHS IN GB**

Figure 12: Abdomen ultrasonography report

USG abdomen shows GRADE 1 FATTY LIVER because Patients having systemic lupus erythematosus (SLE) have quite a 25% to 50% probability of having abnormal liver tests at some point in their lives. Fatty liver is a prominent histopathologic feature in SLE.



MOLECULAR BIOLOGY

SARS-CoV-2(COVID-19),Qualitative

Test Name	Observed Values	Units	Biological Reference Intervals
Sample Type : SARS CoV-19(Oropharyngeal Swabs/ Nasopharyngeal)			
SARS-COV- 19 N1-GENE :	Detected		
SARS-COV-19 N2-GENE :	Detected		
Result :	Positive		
CT Value :	24.00		

Figure 13: Covid 19 report of patient

YEAR 2022: Pt. suffered with COVID 19 in this year and was tested positive, had symptoms like cough and a CT value of 24, she has been treated with HCQS and AZITHROMYCIN as she is already on corticosteroidal therapy she was suggested to take azithromycin as covid treatment. The patient is still on immunosuppressives and doing well she is being cured with the rash and psorium for now due to corticosteroid therapy.

CASE DISCUSSION

Patient education is crucial while treating a patient with Systemic Lupus Erythematosus. It's important to remind people that quitting smoking and protecting their skin from the sun can help them live longer and live healthier lives. Photosensitivity affects people with SLE. Smoking will cause hypertension, which is frequent in SLE, and reduces the effectiveness of the antimalarial medicine hydroxychloroquine (Plaquenil), which is used to treat SLE **[2]**. It is helpful to educate the patient on the importance of ongoing their exercise regimen after they have completed physical therapy treatment. Exercise on a regular basis can help avoid weariness and joint stiffness regular, moderate physical activity can also help to minimize stress's neuroendocrine and immunological consequences. This is particularly crucial for someone who grieves from an autoimmune disease **[2]**. Here in this above case the patient is undergoing treatment with steroids and immunosuppressives so patient is educated well. Before joining any resistive training, muscle-energy techniques, or self-mobilization techniques, be conscious of any changes in bone density and screen for the existence of increasing osteoporosis. This could be an adverse effect of taking steroidal anti-inflammatory drugs for a long time **[3]**. Diabetes, hyperlipidemia, and obesity have all been associated to long-term steroid therapy. Keep an eye out for any signs of renal involvement. Weight gain, edema, and hypertension are all possible symptoms. If there are signs of cognitive deterioration, a referral is necessary. It's possible that Raynaud's syndrome will develop. If this is the case, emphasize the significance of keeping the hands and feet warm and protected **[3]**.

ADRS DETECTED IN 2 CASES:

Prednisolone with metformin: minor interaction is shown as prednisolone decreases effects of metformin by pharmacodynamic antagonism **[4]**

HCQS with azathioprine: serious interaction is shown. Azathioprine and HCQS both increase immunosuppressive effects. But here in this case it can be suggested **[4]**.

HCQS with azithromycin: the patient is suggested azithromycin in covid treatment even she is already on HCQS. HCQS and

azithromycin both increase QTc interval **[4]**.

CONCLUSION

Yes SLE is a rare and autoimmune disease which will lead to several more complications while treating the disease, so no need to worry as corticosteroid and immunosuppressive therapy is effective in treating disease activity. And nephritis is the common complication seen in patients affected with SLE. Doing regular exercise for curing polyarthritis and having good diet rich in antioxidants and vit C will improve the disease activity. Here the patients mentioned undergone many complications even suffered with COVID 19 and still they were having their great lifestyle since they are properly educated about the disease and medication that they are using, so educating the pt is also very important.

CONSENT form is obtained from the patients.

No other conflicts of interests.

REFERENCES:

[1] Kaul, A., Gordon, C., Crow, M. *et al.* Systemic lupus erythematosus. *Nat Rev Dis Primers* **2,** 16039 (2016). https://doi.org/10.1038/nrdp.2016.39

[2] Goodman CC, Fuller KS. Pathology: Implications for the Physical Therapist. 3rd edition. St. Louis, Missouri: Saunders Elsevier, 2009.

[3] Goodman CC and Synder Tk. Differential Diagnosis for Physical Therapists: Screening for Referral. 5th edition. St. Louis, Missouri: Saunders Elsevier, 2013

[4] https://medscape.com