PROLONGED FEVER IN AN ADOLESCENT BOY

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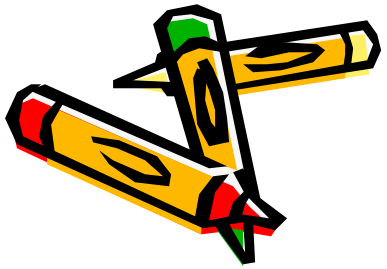
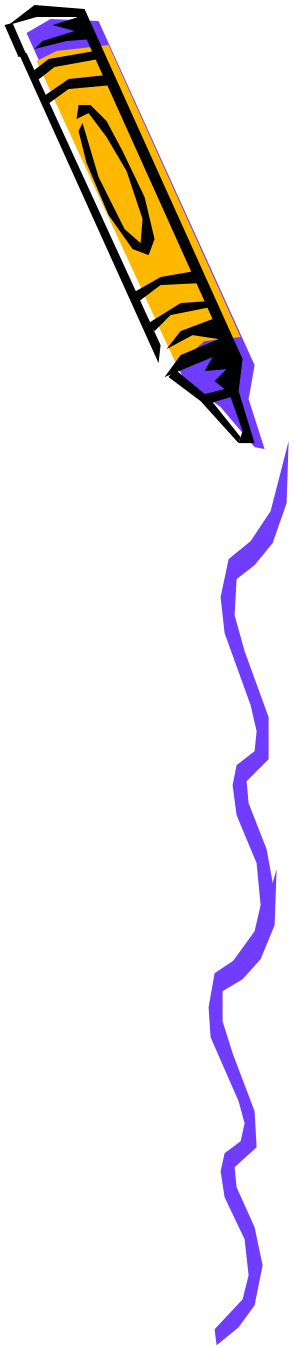
History

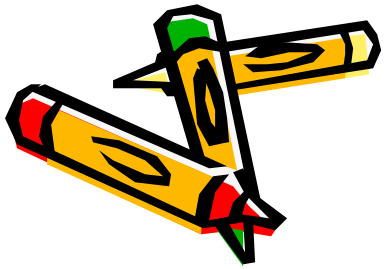
- 11 yrs old adolescent boy was admitted with c/o
- Fever -1 wk
- Myalgia -1 wk
- Arthralgia -1 wk
- Vomiting and loose stools -6 days.
- No history of prolonged drug intake.



History



- Past history – Nil significant
- Delivered by LSCS
- Birth wt-3.000 kg
- Neonatal period uneventful.
- Immunization History-Immunized as per NIS, Typhoid vaccine was given 6 months before
- Developmental History –studying in 7th std, average in studies.
- Family history nil significant.

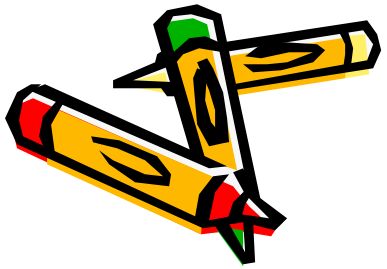


On Examination

- Child was febrile ;pale
- Toxic
- PR-110/mt
- RR-22/mt
- BP-100/70 mm Hg
- Wt-37.4 KG (50th to 75th centile)
- Ht-139.5 cm (25th to 50th centile)
- Upper eyelid puffiness(+)
- Tongue coated
- Petechial rash
- Joints- normal

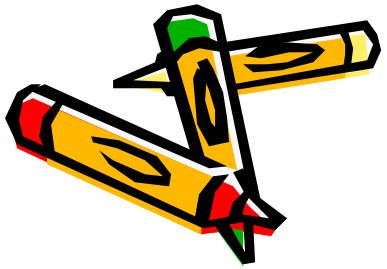
Systemic examination

- RS- Bilateral air entry equal ,no added sounds
- CVS- s1 and s2 heard normally
  soft systolic murmur along left sternal border
- P/A- Liver 3 cm below RCM
  Span 13 cm
  rounded margins
  soft in consistency
  Tenderness in the rt hypochondrium
  Spleen 1.5 cm below LCM
- No free fluid in the abdomen
- CNS – no signs of meningeal irritation
  no focal neurological deficit



Course in the Hospital

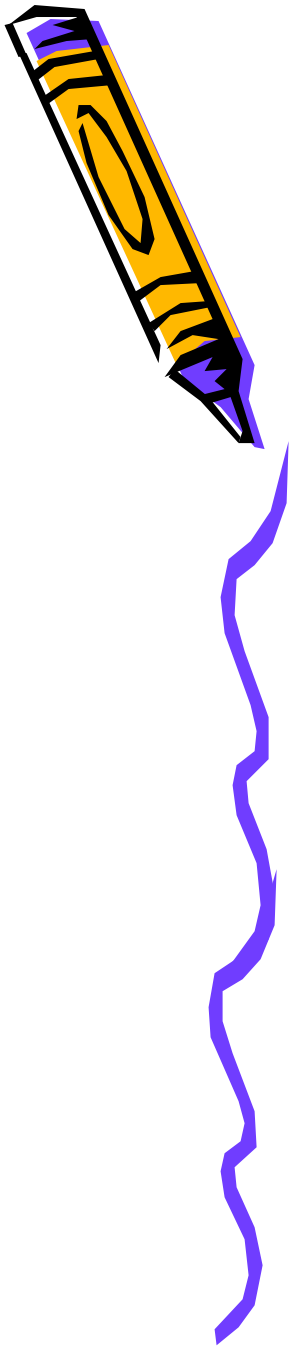
- Child was admitted for evaluation of fever.
- At admission the possibility of enteric fever was considered.
- He was started on iv ceftriaxone pending blood c&s.
- Child continued to have high spikes of fever .
- Infectious work up was negative.



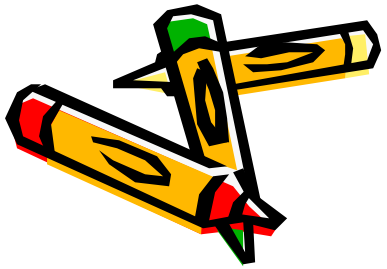
Investigations

Day of illness	D1	D4	D8
TC	4,570	5,100	5,700
DC	Neutrophil predominant	Neutrophil predominant	Neutrophil predominant
HB	7.7 gm%	7.8 gm%	7.5 gm%
Platelets	1,86,000	2,18,000	2,43,000

Investigations



- Widal - neg
- MP - neg
- Dengue Ag and Ab – neg
- Lepto IGM - neg
- Mantoux - neg
- Blood c&s - sterile
- Urea - 23 mg%
- Creatinine - 0.7 mg%
- Total protein - 5.1 mg %
- Albumin - 1.9 mg%
- Urine albumin - 2+
- Urine c & s - no growth



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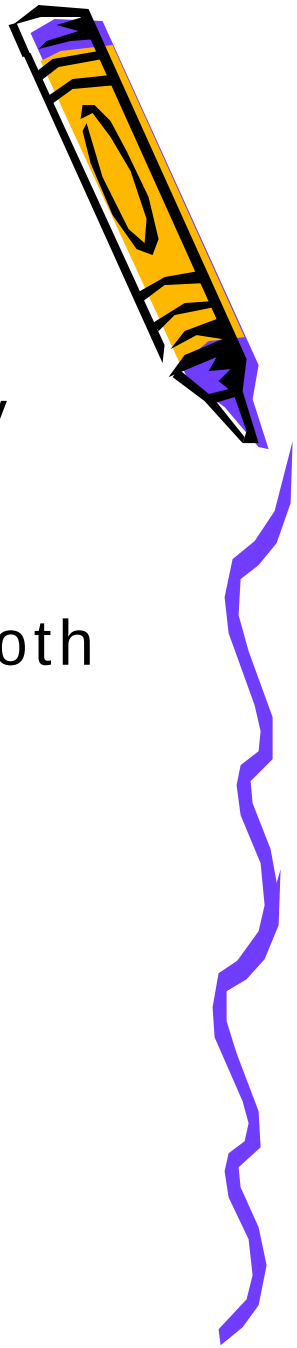
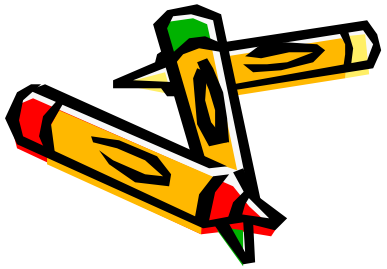


- X-RAY chest - apparent cardiomegaly
  lung fields normal

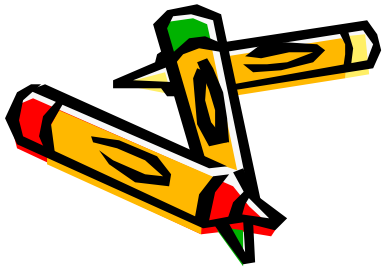
- Ultrasound abdomen – Hepatosplenomegaly, both kidneys upper limit of normal, bilateral basal pleural reaction.

- ECHO - good LV function, mild circumferential pericardial effusion.



Reconsidering the diagnosis

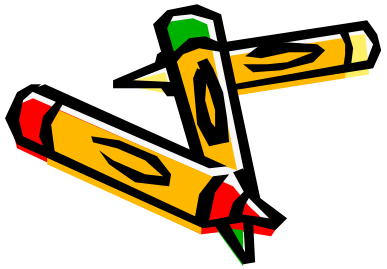
- He continued to have high spikes of fever inspite of adequate antibiotic coverage.
- Child developed oral ulcers on the 5th day of hospitalization which gradually increased and become confluent on 7th day.
- He also developed erythematous rash over the ear on the 7th day of hospitalization.



- In view of multisystem involvement the possibility of

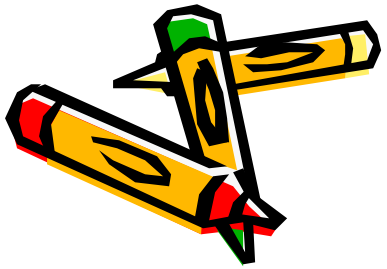
  connective tissue disorder

  was considered even though it is considered as an uncommon presentation in male.



Further work up

- ESR - 130 mm/hr
- ANA - Positive
- Anti ds DNA – Positive
- CRP -1.2 mg/dl



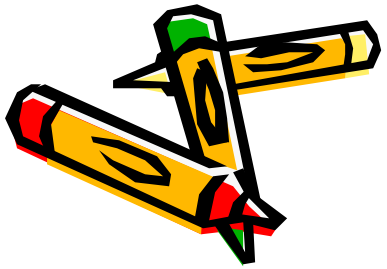
DIAGNOSIS

1. Oral ulcers
2. Serosal involvement – pleural and pericardial
3. Skin –erythematous rash
4. Immunologic –Anti dsDNA +ve
5. Antinuclear antibody +ve

5 out of 11 criteria based on 1997 revised classification criteria for SLE

Final diagnosis

SYSTEMIC LUPUS ERYTHEMATOSUS

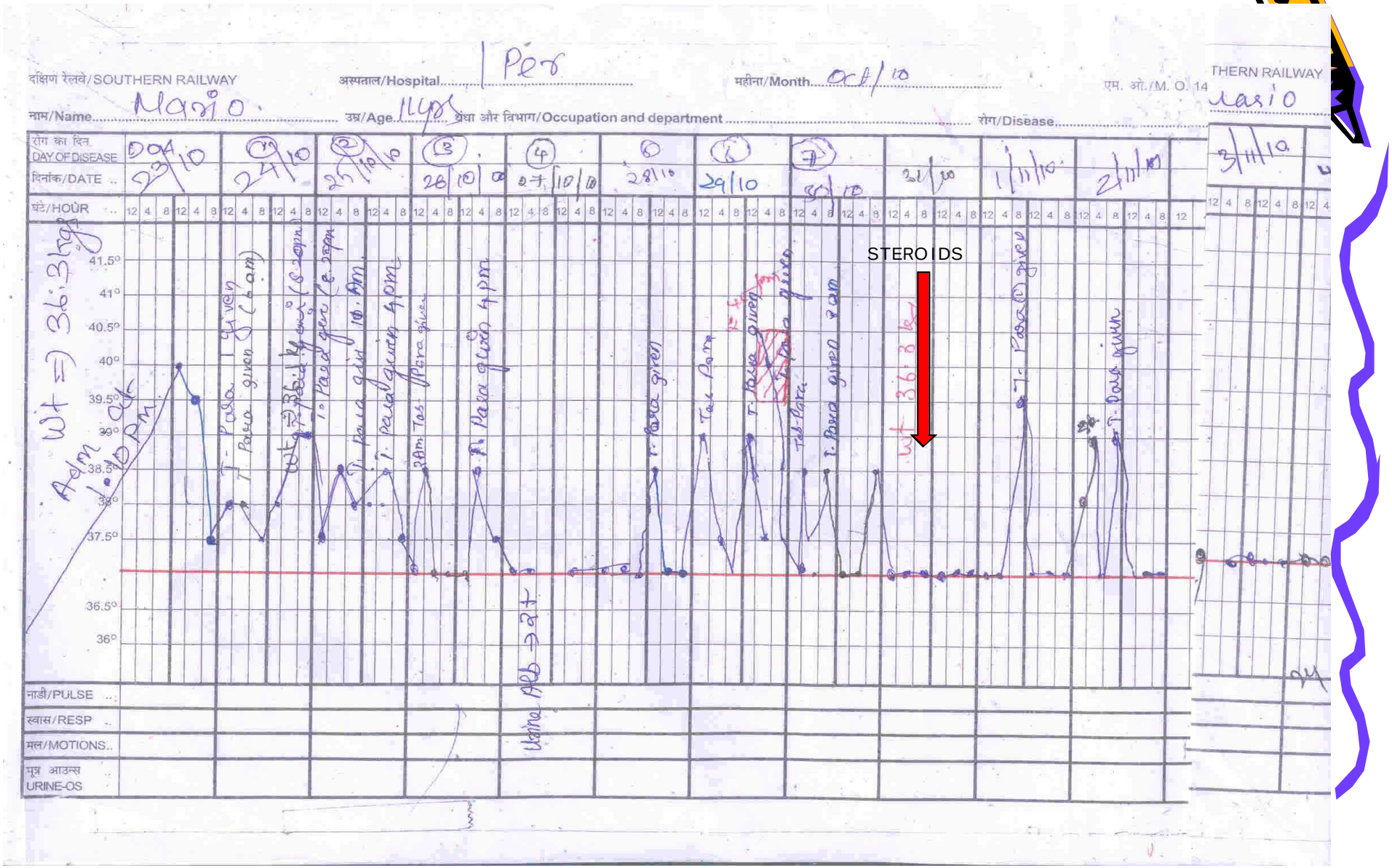


Management

. He was started on steroids- prednisolone 1 mg/kg /day .

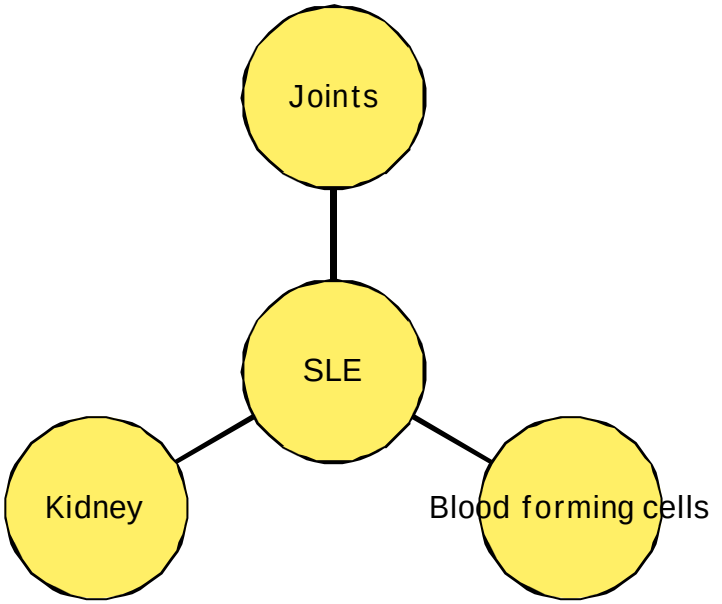
- Child clinically improved.
- He was referred to nephrologist for renal biopsy and further management.
- Parents not willing at present inspite of counselling

Temperature pattern



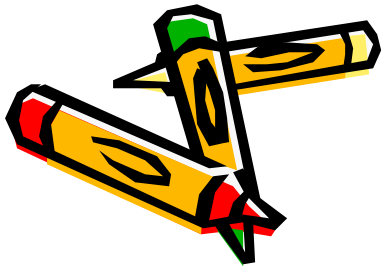
Discussion
SLE

- Multisystem autoimune disease.
- Autoantibodies are directed against self antigens .
- Inflammatory damage to many target organs



Incidence

- 6-18/100000 in white and 20-30/100000 in black population.
- Girls:Boys varies from 4:1to 8:1.
- 20% of SLE present in childhood.
- Rare below 8 yrs.



Potential causes of the female predilection

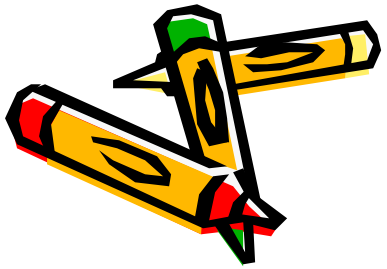
Hormones

- estrogen and its hydroxylation

  estrone is preferentially hydroxylated at the C-16 position, resulting in the accumulation of 16-hydroxylated metabolites.

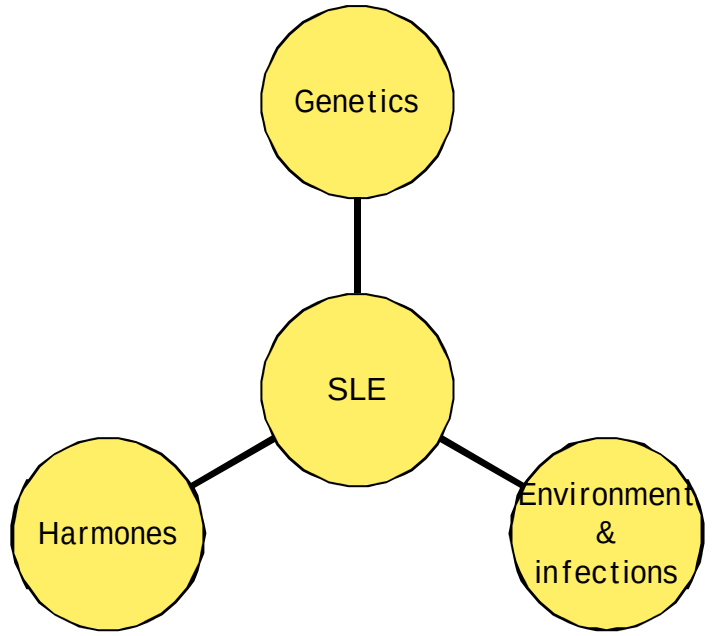
- Little androgenic hormone.
- Increased Prolactin levels may increase [illegible]ase activity .

- Genetics
  - Random X chromosome inactivation
  - Maternal microchimerism
  - Determinant SLE associated genes are located on the X chromosome
  - Demethylation of these genes inactivate X chromosomes.

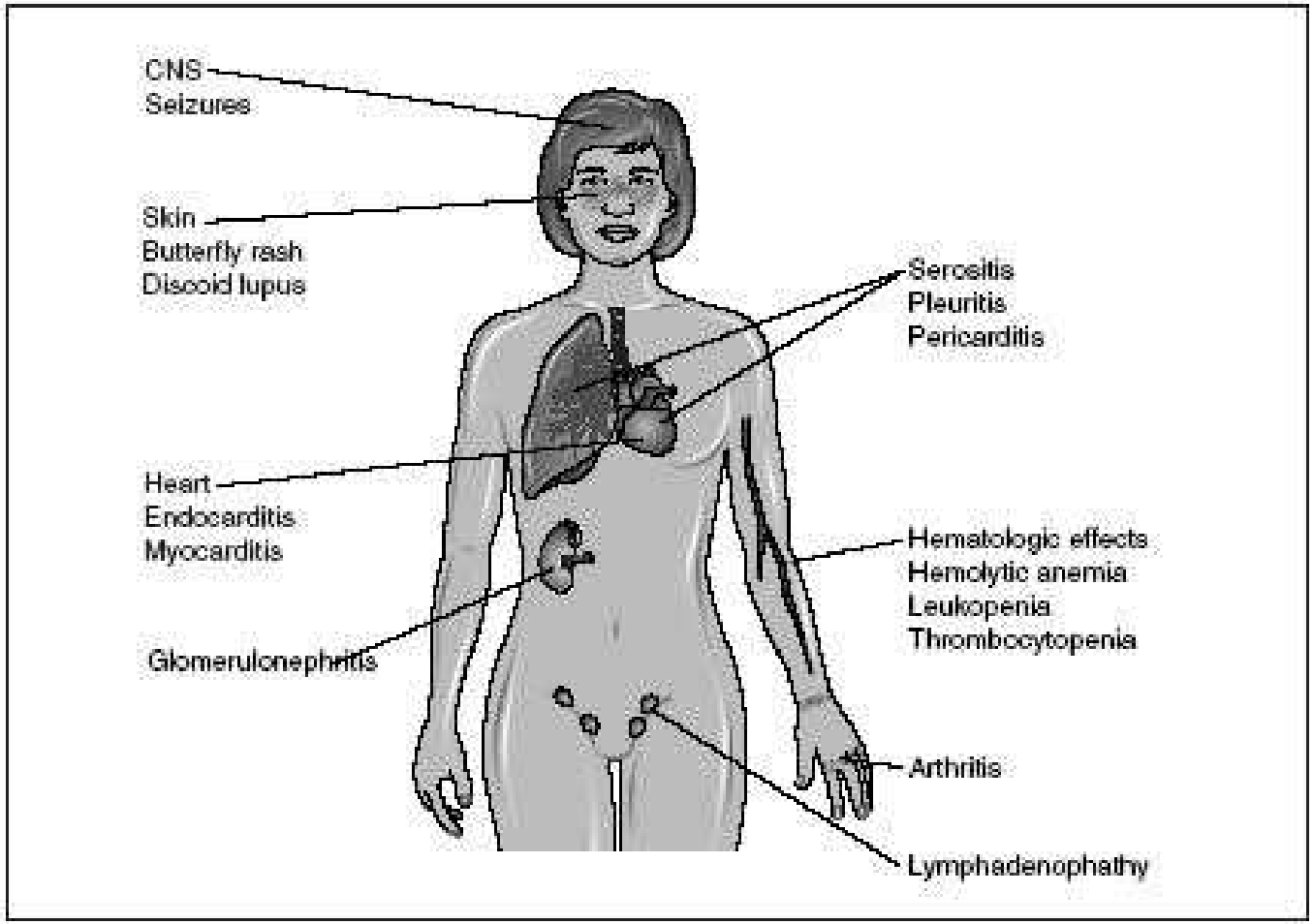


Etiology

- Immune dysregulation
- Autoantibody formation
- Circulating and tissue bound immune complexes.
- Complement fixation
- Recruitment of inflammatory cells
- Tissue injury.

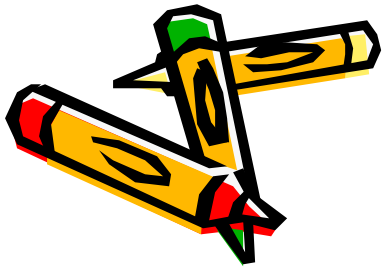


Presenting manifestations



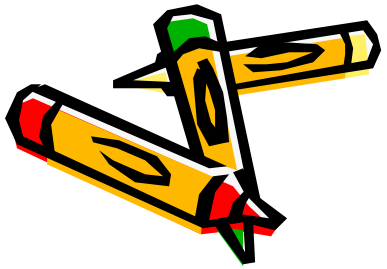
1997 Revised classification criteria for SLE

1. Malar rash
2. Discoid rash
3. Photosensitivity
4. Oralulcers
5. Arthritis
6. Serositis
7. Neurologic disorder
8. Hematology disorder



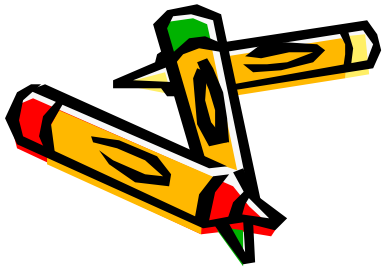
Criteria con…

9. Renal involvement
10. Immunologic
    - Anti DNA Ab
    - Anti smith Ab
    - Antiphospholipid Ab
11. Antinuclear Ab



Management

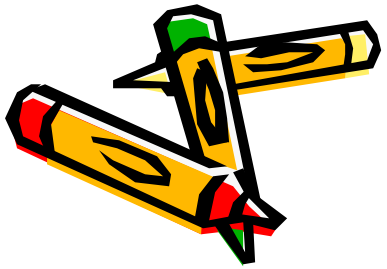
- General
  - Counselling
  - Education
  - Avoidance of exposure to sun
  - Sunscreen cream
  - Reduction of activity
  - Immunization
  - Vitamin D
  - Calcium supplentation



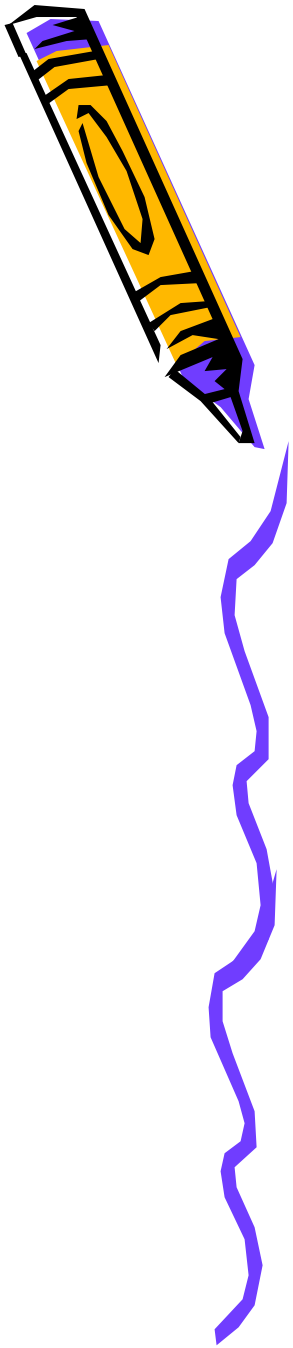
Management

- Specific

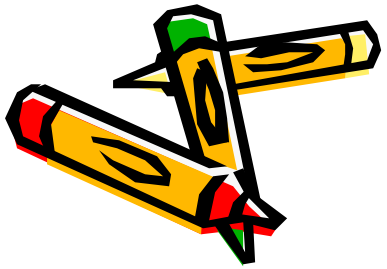
  NSAIDs-Naproxen
  Hydroxychloroquine
  Glucocorticoids
  Immunosuppressive therapy
    cyclophosphamide
    Azathioprine
    cyclosporin A
    Mycophenolate mofetil
  Biologicals
    Rituximab-antiCD20 Monoclonal Ab



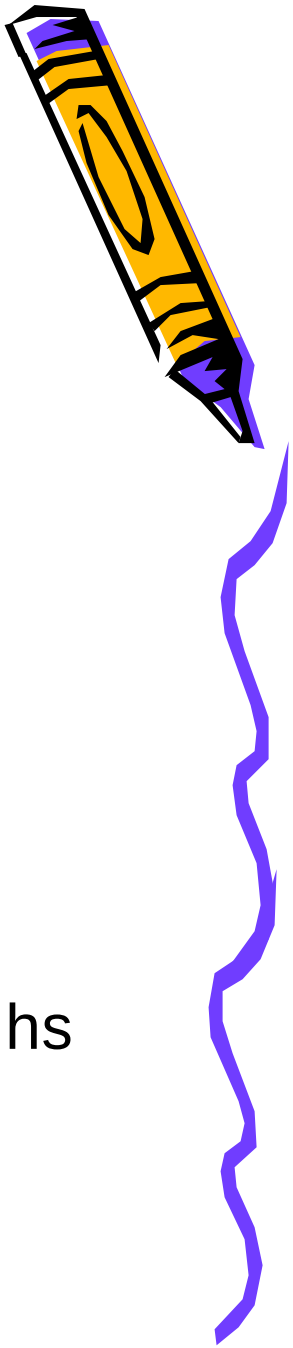
Complications



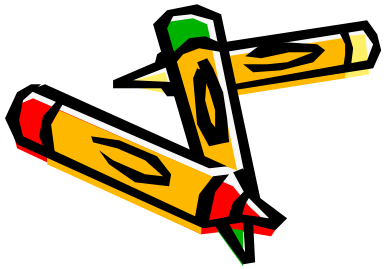
- Major cause of death is
  - Infection
  - Nephritis
  - CNS disease
  - Pulmonary Haemorrhage
- Prognosis
  - 5 yr survival rate-95-100%
  - 10 yr survival rate 86%



Follow up



- Haematology
- ESR
- C3, C4
- anti-DNA (quantitative)
- AST, ALT
- albumin
- Creatinine
- urinalysis.
- every 2–4 weeks at diagnosis, to every 2 months or less when the disease is under control.



Review of Literature

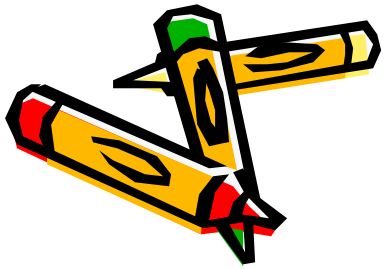
1. Kuwait Medical Journal 2007, 39 (2):181-183

Al-Adan Hospital, Kuwait

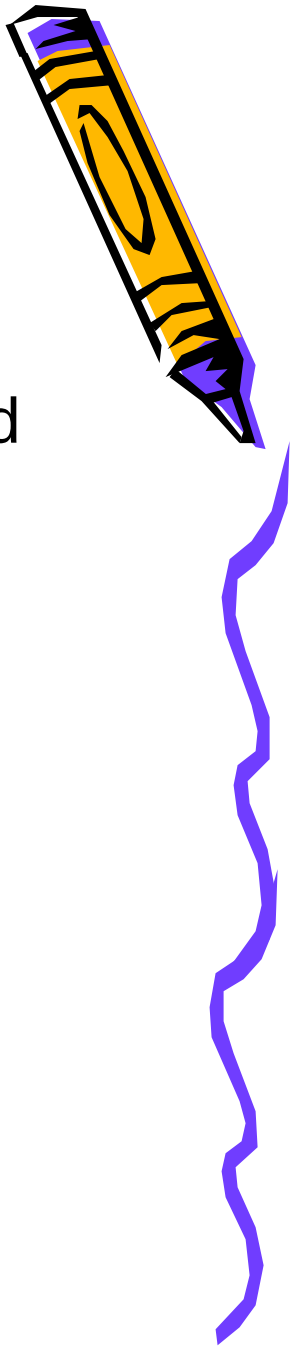
2. Indian Pediatrics 2009;46: 711-715

Clinical features and outcome of SLE

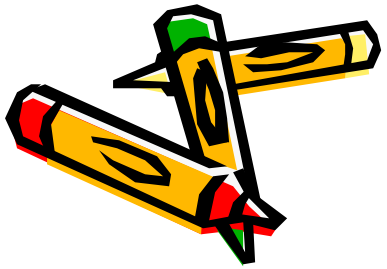
3. Rjumachi 1998 June;38(3):496-503



Carry home message



- Adolescent boys should also be investigated for SLE, when they present with generalized weakness and fever for which no focus of infection
- Evolution of clinical signs should be evaluated carefully to arrive at correct diagnosis
- Awareness and active suspicion can work towards early diagnosis so that appropriate treatment can be initiated and several serious complications can be prevented.




THANK YOU
ANNE GEDDES