Evaluation and management of rheumatologic emergencies in the ICU

DM Seminar:
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Dept. of Pulmonary medicine
Rheumatologic emergencies

• 19% of emergencies admitted to acute general medical wards have rheumatological problem as the cause for admission


• Complications of rheumatic diseases frequently present with protean manifestations

  Slobodin G. Emerg Med J. 2006;23(9):667-71
Neurological emergencies
Meningitis

Acute onset illness; Fever, head ache, signs of meningeal irritation

CSF Analysis

Neutrophilic pleocytosis
Elevated protein
Low glucose levels
Gram stain and CSF culture +

Lymphocytic pleocytosis
Normal/mild elevation of protein
Normal sugar levels
Gram stain and CSF culture -

Bacterial meningitis :
Empirical antibiotics
Ceftrixone + Vancomycin + Ampicillin

Aseptic meningitis

Active disease:
SLE
Behcet’s
Vasculitides

Treatment related:
• NSAIDS, IVIG induced
• Self limiting illness

Steroids
Stroke (CVA)

Focal neurological deficit / TIA
Neuroimaging S/O CVA → CT/MRI brain
ECHO, Carotid doppler
aPL, LAC

Vaso occlusion
Bland infarct

Secondary to APLA
Therapeutic anticoagulation
Oral anticoagulation
INR of 3

Libmansach’s endocarditis
• Steroids
• Anticoagulation

Vasculitic infarct:
immunosuppression

Carotid atherosclerosis
Athero-embolic infarct
• Aspirin
• Risk factor reduction
• Control disease activity
CNS vasculitis / diffuse vascular occlusion

- Headache
- Altered sensorium
- Seizures
- Multiple neurological deficits

Neuroimaging:
- Multiple infarcts (arterial + venous)
- LAC, aPL +

Vascular occlusion:
- APLA

Therapeutic anticoagulation

MRI brain:
- Infarctions and bleeds
- Grey (cortex and deep) and white matter

Angiography:
- Irregular narrowing, blunt ending and beading of small arterioles

Brain / leptomeningeal biopsy +

CNS vasculitis:
- SLE, PAN, Wegner’s
- Isolated CNS angitis

Methyl prednisolone +
- Cyclophosphamide

Neither ruled out

Immunosuppression +
- Anticoagulation
Seizures

- In SLE and primary CNS angitis seizures are reported in up to 10% patients and in APLA, Wegner’s granulomatosis to a lesser extent (2-3%)
- Multiple etiologies: post CVA, uremia, meningitis, disease activity

Treatment:
- Remove metabolic cause
- Seizures due to disease activity might respond to steroids alone
- Recurrent seizures respond to common antiepileptic drugs and long term treatment is not necessary

Other causes of altered sensorium include malignant hypertension, TTP, steroid induced psychosis, uremic encephalopathy
Hematologic emergencies
TTP in SLE

- Typical features of CNS, renal dysfunction, thrombocytopenia, anemia, schistocytes in PBS
- Mimics disease flare sometimes
- Plasma exchange is treatment of choice
  

- Plasma exchange daily sessions to be given till platelet count is >50,000/cc for 2 consecutive days
- Poor response to immunosuppressants when used alone
  steroids and CYC used as adjunctive therapy
- Relapses are common

Scleroderma crisis and catastrophic APLA mimic TTP
# Hematological emergencies

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Investigations</th>
<th>Treatment options</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Autoimmune hemolytic anemia (warm antibody)</strong> (SLE)</td>
<td>PBS</td>
<td>High dose steroids</td>
</tr>
<tr>
<td></td>
<td>Hemolytic W/U Coombs test</td>
<td>IVIG (less effective)</td>
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<td></td>
<td>Serum LDH</td>
<td>Splenectomy</td>
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<tr>
<td></td>
<td></td>
<td>Rituximab ?</td>
</tr>
<tr>
<td><strong>Autoimmune thrombocytopenia</strong> (SLE, APLA)</td>
<td>ANA</td>
<td>High dose steroids</td>
</tr>
<tr>
<td></td>
<td>aCL</td>
<td>IVIG (0.4 g/kg/d for 5 days)</td>
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<tr>
<td></td>
<td>LAC</td>
<td>Rituximab (refractory cases)</td>
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<td></td>
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<td>Splenectomy (refractory cases)</td>
</tr>
</tbody>
</table>
Autoimmune cytopenias - treatment

- IVIG administered alone or with steroids no benefit in terms of response rates or time to clinical response in thrombocytopenia


- Rituximab (chimeric anti CD 20 antibody) has been used for refractory autoimmune hemolytic anemia* and thrombocytopenia^ with good response


Cardiac emergencies
## Cardiac ischemia

<table>
<thead>
<tr>
<th>Disease</th>
<th>Pathology</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>SLE</td>
<td>Accelerated atherosclerosis</td>
<td>•Clinical</td>
<td>•MX of ACS&lt;br&gt;•Risk factor control&lt;br&gt;•Disease activity control</td>
</tr>
<tr>
<td>RA</td>
<td></td>
<td>•Immunological markers</td>
<td></td>
</tr>
<tr>
<td>PAN</td>
<td>Arteritis</td>
<td>•Clinical</td>
<td>•MX of ACS&lt;br&gt;•Steroids + CYC</td>
</tr>
<tr>
<td>Churg-Strauss syndrome</td>
<td></td>
<td>•Angiogram- aneurysm&lt;br&gt;HBS Ag / P-ANCA</td>
<td></td>
</tr>
<tr>
<td>APLA</td>
<td>Thrombosis</td>
<td>•Clinical&lt;br&gt;•LAC/ aCL</td>
<td>•MX of ACS&lt;br&gt;•Lifelong anticoagulation</td>
</tr>
<tr>
<td>Takayasu arteritis</td>
<td>Arteritis</td>
<td>•Clinical features&lt;br&gt;•Angiogram-stenosis, occlusion&lt;br&gt;Post-stenotic dilatation, aneurysms</td>
<td>•MX of ACS&lt;br&gt;•Steroids&lt;br&gt;•Revascularization (After control of inflammation)</td>
</tr>
</tbody>
</table>
Arrhythmias

• Transient atrial fibrillation, flutter or PSVT in 20–30% of SSc patients
• NSVT was described in 7–13%
• SCD is reported in 5–21% SSc patients
• Management of AF, SVT, VT do not differ from patients without SSc

Seferovic PM. Rheumatology 2006;45:iv39–iv42
Myocarditis with CHF

- SLE presenting with CHF is rare with few case reports and case series
- Associated with nephritis (60%), and elevated anti-ds DNA antibodies (80%)
- Responsive to high dose steroids and supportive care
- Refractory cases require cyclophosphamide and IVIG

Chung JW. Rheumatol Int. 2008 28(3):275-80

- Case report of utility of immunoadsorption onto staphylococcal protein A

Griveas I. Ther Apher Dial. 2004;8:281-5
Pericardial tamponade

- Pericardial involvement is common in SLE (30-35%).

- Pericardial tamponade is uncommon and SLE contributes to 3.2% of all cases.

- Frequently associated with nephritis, Libman-Sacks endocarditis and myocarditis.

- Responds well to oral steroids and pericardial drainage

- Does not lead to constrictive pericarditis

*Weich HS. Lupus. 2005;14(6):450-7*
Pulmonary emergencies
Pulmonary embolism

- DVT/PE are seen in patients with APLA
  8% of patients with APLA develop thrombotic events during 5 yr follow up
  Strong association between LA and VTE (odds ratio, 9.4; 95% confidence interval [CI], 2.1 to 46.2)

- Initial evaluation and management does not differ from other cases of PE
  LAC cannot be tested in patients receiving heparin

- Patients with APLA, presenting with PE requires life long anticoagulation (INR - 3)
Ventilatory failure

- Acute or acute on chronic ventilatory failure can be seen in PM/DM

- High dose corticosteroids / pulse methyl prednisolone with methotrexate* and in refractory cases with IV Ig^ have been successfully used

  *Obón Azuara B. An Med Interna. 2005 Sep;22(9):434-6

- NIV can be tried (level C evidence)
  

- IVIG is less effective in PM

- Plasmapheresis ineffective in both the conditions
Diffuse parenchymal lung disease

- Severe pneumonia
- Acute pneumonitis with DAD. Eg: SLE, PM/DM
- Diffuse alveolar hemorrhage
- Eosinophilic pneumonia. Eg: Churg-Strauss syndrome
- Drug induced. Eg: Methotrexate induced DPLD
  Sulfasalizine induced DAH
- Cardiogenic pulmonary edema due to myocarditis
- Fluid over due to oliguric ARF
- Uremic lung
# Differentiation of DPLD

<table>
<thead>
<tr>
<th></th>
<th>Clinical features</th>
<th>Radiological features</th>
<th>BAL</th>
<th>Lung biopsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>AHRF</td>
<td>Multilobar consolidation</td>
<td>Microbiological stains and Culture +</td>
<td></td>
</tr>
<tr>
<td>Lupus pneumonitis</td>
<td>AHRF</td>
<td>Multilobar consolidation (lower lobes)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DAH</td>
<td>• AHRF</td>
<td>Multilobar consolidation MRI:↑ signal in T2W image</td>
<td>• Hemorrhagic</td>
<td>• Capillaritis +/-</td>
</tr>
<tr>
<td></td>
<td>• Hemoptysis</td>
<td></td>
<td>• Hemosiderin laden macroph.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Fall in Hb</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>MTX induced DPLD</td>
<td>• h/o MTX</td>
<td>Diffuse interstitial pattern</td>
<td>Lymphocytosis High CD4:CD8</td>
<td>Desquamative lesions</td>
</tr>
</tbody>
</table>

- AHRF: Acute Respiratory Distress Syndrome
- BAL: Bronchoalveolar Lavage
- MTX: Methotrexate
- MTX induced DPLD: Methotrexate-induced Desquamative Pulmonary Lesions
- DAD: Diffuse Alveolar Damage
- NSIP: Non-Specific Interstitial Pneumonitis
- Org. pneum.: Organized Pneumonia
- Capillaritis: Capillary inflammation
- DAH: Diffuse Alveolar Hemorrhage
Lupus pneumonitis

- 2-4% of patients with SLE, but it is presenting manifestation in 50% of them
- Histological picture shows predominant diffuse alveolar damage
- High dose steroids + broad spectrum ABx
- Steroid resistant cases might respond to CYC, IVIg, plasmapheresis, Rituximab*


- Mortality rates 50% once respiratory failure sets in
Methorexate induced DPLD

- Subacute to fulminant hypoxic respiratory failure
- Most cases (50%) develop during 1st 4 months of treatment with MTX
- Responds to withdrawal of MTX and early institution of steroids
- Mortality rate of 15-20%, up to 50% on re-challenge

## Diffuse alveolar hemorrhage and Pulmonary renal syndromes

<table>
<thead>
<tr>
<th>Disease</th>
<th>Renal</th>
<th>Arthritis</th>
<th>Skin vasculitis</th>
<th>ANA</th>
<th>C_L</th>
<th>Immun. markers</th>
<th>Tissue Ab. Staining</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wegner’s granulomatosis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>N</td>
<td>C-ANCA</td>
<td>_</td>
</tr>
<tr>
<td>Microscopic polyangitis</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>N</td>
<td>P-ANCA</td>
<td>_</td>
</tr>
<tr>
<td>Goodpasture’s syndrome</td>
<td>+</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>N</td>
<td>Anti - GBM</td>
<td>Linear</td>
</tr>
<tr>
<td>SLE</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>N</td>
<td>Anti ds-DNA</td>
<td>Granular</td>
</tr>
<tr>
<td>Isolated pulmonary capillitis</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>N</td>
<td>_</td>
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</table>

Isolated DAH reported in RA, PM/DM and MCTD

Pulmonary renal syndrome described in RA, SSc and MCTD
## DAH - Treatment

<table>
<thead>
<tr>
<th>Condition</th>
<th>Treatment</th>
<th>Notes</th>
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<tbody>
<tr>
<td>SLE</td>
<td>Steroids + CYC</td>
<td>Plasmapheresis: used as salvage, no survival benefit (retrospective analysis)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rituximab: anecdotal use</td>
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<tr>
<td></td>
<td></td>
<td>Nephrol Dial Transplant.2008;23(1):385-6</td>
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<tr>
<td></td>
<td></td>
<td>IVIg: anecdotal use</td>
</tr>
<tr>
<td>Wegner’s granulomatosis &amp; Microscopic polyangitis</td>
<td>Steroids + CYC</td>
<td>Plasmapheresis: useful (uncontroled study n=20)</td>
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<tr>
<td></td>
<td></td>
<td>Rituximab: case reports</td>
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<tr>
<td></td>
<td></td>
<td>Intern Med. 2007;46(7):409-14</td>
</tr>
<tr>
<td>Goodpasture’s</td>
<td>Oral steroids + oral CYC + Plasmapheresis</td>
<td></td>
</tr>
<tr>
<td>Unknown etiology</td>
<td>Steroids + CYC + plasmapheresis</td>
<td></td>
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Renal emergencies
<table>
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<tr>
<th>Disease</th>
<th>Lungs</th>
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<td>Linear</td>
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<td>Anti GBM disease</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>N</td>
<td>Anti - GBM</td>
<td>Linear</td>
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<tr>
<td>SLE</td>
<td>+</td>
<td>+</td>
<td>±</td>
<td>+</td>
<td></td>
<td>Anti ds-DNA</td>
<td>Granular</td>
</tr>
<tr>
<td>Renal limited crescentic GN</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>_</td>
<td>N</td>
<td>P-ANCA</td>
<td>_</td>
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<tr>
<td>Cryoglobulinemia</td>
<td>_</td>
<td>+</td>
<td>+</td>
<td>_</td>
<td></td>
<td>Cryocrit Anti HCV</td>
<td>Granular</td>
</tr>
</tbody>
</table>

Malignant hypertension, scleroderma renal crisis and TTP are mimics
# RPGN- Treatment

<table>
<thead>
<tr>
<th>Disease</th>
<th>Therapy</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>SLE</td>
<td>Steroids + CYC</td>
<td>Rituximab: case reports</td>
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<td>* Lupus. 2003;12(10):798-800</td>
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<td></td>
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<td>Arthritis Rheum. 2007;56(4):1263-72</td>
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<td>IVIg: useful as salvage (uncontroled study ,n=7)</td>
</tr>
<tr>
<td>Wegner’s granulomatosis &amp;</td>
<td>Steroids + CYC</td>
<td>Plasmapheresis: useful in cases requiring RRT</td>
</tr>
<tr>
<td>Microscopic polyangitis</td>
<td></td>
<td>* Ther Apher. 2001;5(3):176-81</td>
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<tr>
<td></td>
<td></td>
<td>Rituximab: useful in refractory cases</td>
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<tr>
<td></td>
<td></td>
<td>(uncontroled study, n= 6*, 9^)</td>
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<tr>
<td></td>
<td></td>
<td>*Arthritis Rheum 2005;52:262– 8</td>
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<tr>
<td></td>
<td></td>
<td>IVIg: beneficial as single agent in wegner’s</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n=12)</td>
</tr>
<tr>
<td>Goodpasture’s</td>
<td>Oral steroids + oral CYC +</td>
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<tr>
<td></td>
<td>Plasmapheresis</td>
<td></td>
</tr>
<tr>
<td>Cryoglobulinemia</td>
<td>•IFNα+ ribavirin</td>
<td>Rituximab: useful in refractory cases</td>
</tr>
<tr>
<td></td>
<td>•Steroids+ CYC</td>
<td>retrospective analysis of 25 cases</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* J Nephrol. 2007 ;20(3):350-6</td>
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</tbody>
</table>
Scleroderma renal crisis (SRC)

• **Accelerated hypertension and RPRF** occurring in a patient with systemic sclerosis
• CHF, arrhythmias, pericardial effusion (2° to HTn, renal failure)
• Upto 10% of patients with SSC develop SRC
• 10% of patients are apparently normotensive (eg: 95/60 to 145/80)

**Risk factors:**
- Early disease (75% of cases develop in first 4 yrs of disease)
- Diffuse SSC (25% patients develop SRC)
- Renal hypoperfusion (sepsis, dehydration, drugs decreasing renal perfusion)
- Steroid use (>40mg/day): Case control studies suggest increased risk

*Steen VD. Arthritis Rheum 1998;41:1613– 9*
# Scleroderma renal crisis (SRC)

Factors that occur before onset of SRC that may be helpful in predicting SRC

<table>
<thead>
<tr>
<th>Predictive of SRC</th>
<th>Not predictive of SRC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse skin involvement</td>
<td>Previous blood pressure elevation</td>
</tr>
<tr>
<td>Rapid progression of skin involvement</td>
<td>Abnormal urinalysis</td>
</tr>
<tr>
<td>Disease duration &lt;4 years</td>
<td>Previous increase in serum creatinine</td>
</tr>
<tr>
<td>Anti-RNA polymerase III antibody</td>
<td>Anti-topoisomerase I (Scl-70) or anti-centromere antibodies</td>
</tr>
<tr>
<td>New anemia</td>
<td>Pathologic abnormalities in renal blood vessels</td>
</tr>
<tr>
<td>New cardiac events</td>
<td></td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td></td>
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<tr>
<td>Antecedent high-dose corticosteroid</td>
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</tr>
</tbody>
</table>

SRC – lab findings

Mild azotemia
• Might progress despite control of BP (peak level upto 3.8 mg/dl)
• Not attributed to ACE inhibitors
• ACEI should not be discontinued for this reason

Microscopic hematuria

Proteinuria

Granular casts

Hemolytic anemia

Thrombocytopenia (rarely below 50,000/cc)
SRC- Treatment

Adequate control of blood pressure with ACE inhibitors (around 120/70)

Captopril is preferred (shorter half life and greater flexibility)

Addition of other agents (preferably ARBs) might be required for adequate control
SRC – treatment and prognosis

• Pre- captopril era <10% of patients survived the 1\textsuperscript{st} year
  Current 5 yr survival rate is 65%.

• Risk factors for bad outcome:
  Initial creatinine level >3 mg/dl
  Uncontrolled BP within 3 days
  Male gender, older age, presence of CHF

• 40% patients require long term RRT

  Steen VD. Ann Intern Med 2000;133:600–3
Abdominal emergencies
Mesenteric ischemia

- Acute abdomen
- Bloody diarrhoea
- Peritonitis

CT angiogram:
- Bowel wall thickening
- Ascites
- Clot in mesentric vein

Mesentric vein thrombosis
- Seen in APLA

Visceral angiogram
- Aneurysmal dilatation of visceral arteries
- Poly Arteritis Nodosa
- Churg Strauss Synd.

Normal
- Wegner’s
- HSP
Mesenteric vein thrombosis

Peritonism -
- Heparin + Thrombolysis
  - Non Viable
    - Resection
    - Anti-coagulation
  - Viable
    - Thrombectomy
    - Anti-coagulation
    - Re-look surgery

Peritonism +
- Large segment
- Small segment
- Re-look surgery

Laparotomy
Visceral vasculitis

- PAN
- HSP
- Churg – Strauss Synd.
- RA
- Wegners granulomatosis
- Rarely: SLE, SSc, PM/DM, giant cell arteritis
- Acute abdomen with bloody diarrhoea
- Bowel infarction
- GI bleed
- Bowel perforation
- Cholecystitis
- Pancreatitis
- Appendicitis
## Visceral vasculitis - management

<table>
<thead>
<tr>
<th>Disease</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAN</td>
<td>• Clinical features</td>
<td>Steroids +/- CYC</td>
</tr>
<tr>
<td></td>
<td>• Visceral angiography: anurysmal dilatation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• P- ANCA + rarely</td>
<td>Lamivudine/IFN γ</td>
</tr>
<tr>
<td></td>
<td>• HBS Ag + (50%)</td>
<td></td>
</tr>
<tr>
<td>HSP</td>
<td>• Clinical features</td>
<td>Steroids</td>
</tr>
<tr>
<td>Wegner’s granulomatosis</td>
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<tr>
<td></td>
<td>• C-ANCA +</td>
<td></td>
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<tr>
<td>RA</td>
<td>• Clinical features</td>
<td>Steroids</td>
</tr>
<tr>
<td></td>
<td>• SC nodules, RF+</td>
<td></td>
</tr>
</tbody>
</table>
Catastrophic APLA

• Seen in 1% of cases of APLA

• Multiple thromboses of medium and small arteries developing over a period of days

• Stroke, cardiac, hepatic, renal, adrenal, intestinal infarction and peripheral gangrene

• May present with adrenal insufficiency, but renal insufficiency uncommon (unlike TTP)

• aPL, LAC + (not seen in other mimics)
  Thrombocytopenia is usually present
  RBCs not fragmented (r/o TTP) and FDP not elevated (r/o DIC)
Catastrophic APLA

Diagnostic criteria:
1. 3 organ/system involvement
2. Simultaneous/within a week
3. HPE: small vessel occlusion
4. LAC/aCL + twice 6 wks apart

Definitive: all 4 criteria
Probable:
criteria 1,2,4
criteria 1,3,4
All criteria but 2 organ involvement
All criteria but LAC/aCL tested once

Treatment:
Anticoagulation +
Steroids +
PE or IVIG

Cause of death: CNS, cardiac involvement and infection

70% success

Buciarelii S. Arthritis Rheum. 2006;54(8):2568-76
Summary

• Rheumatic diseases can present with protean manifestation involving any organ system

• High index of suspicion in a given clinical setting and appropriate investigations help in early diagnosis

• Current therapeutic modalities (immunosuppression, plasmapheresis, IVIg) give good results when initiated in time

• Newer therapeutic modalities like Rituximab is being explored as salvage therapy in various clinical settings with good results
Thank you