Case report
ESIM Summer School
July, 6th, 2010

- male patient, 52 years

- reduction in general condition for 2 days
- fever 40 °C
- dyspnea on exertion “crushing chest pain”
- progressive paresis of legs

- medication at admission: 50 mg azathioprine/d
  30 mg prednisolone/d

- regular visits to Namibia (returning 6 weeks prior to admission)

Medical history:

- 1/2 year ago: fever up to 40 ºC for 8 weeks:
- diagnosis: ANCA-negative large-vessel vasculitis of pelvis and legs (ANA, ANCA, RF, Anti-CCP negativ)
- hypoimmunogammaglobulinemia (IgG 467 mg/dl [664-1495])

high-dose corticosteroids and MTX

change to Azathioprine due to MTX-drug toxicity

(2 weeks before current admission)

Clinical examination:

- Reduced general aspect and obesity (100 kg, 181 cm)
- 39,6 ºC, P 75/min, RR 97/55 mmHg, RespR 16/min
- maculopapular exanthema on trunk
- paraparesis of legs, especially hip and knee flexors, muscle strength 4/5

Results I

- General:

- CRP 276 mg/l (0,5-5)
- PCT >2 ng/ml (<0,5)
- ESR 57/88 mm/h
- Crea 1,92 mg/dl (0,5-1,1)
- Total bilirubin 1,4 mg/dl (< 1)
- WBC 15,5/nl (4,3-10)
- IgG 259 mg/dl (700-1600)

- **Regular values for** Na, K, LDH, ALT, AST, AP, abumine, Hb, platelets, troponin I, TSH, IgM, IgA, urine status and sediment.

- Microbiology:

Negative for malaria, HSV (PCR-CSF), rickettsiosis, brucellosis, tularemia, syphilis, salmonellosis, trypanosomiasis, schistosomiasis, worm ova. BC negative.

- CSF:

Pathologically elevated CSF/serum quotient for albumin with 10,5 x 10^3 (8 x 10^3), correlates with light disruption of blood-brain-barrier. No pleocytosis. No intrathecal Ig-production.

Results II

- **Imaging**
- Abdominal US
- CT-Thorax
- MRI-Cerebrum
- MRI-Spine/Aorta

∑ No signs of vasculitis

- FDG-PET/CT: cutane FDG-uptake (exanthema)

- **Other results:**
- ECG
- TEE
- ENT / eyes / OMS

∑ no signs of vasculitis / infectious focus or MI

- Skin biopsy: discrete inflammatious infiltrates by granulocytes in corium and subcutaneous fat (exanthema)

Differential diagnoses:

- **febrile ID with unknown origin during immunosuppressive therapy**
- **or acute exacerbation of vasculitis**
- **or febrile drug reaction to azathioprin**

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$\sum$ **FUO**

Follow up:

1.) exclusion of MI and cerebral infarction

2.) discontinuation of Aza, empirical antibiotic treatment (pip+comb+moxi) for 2 d -> no resolution, progress of paresis

3.) broader antibiotic treatment (vanco+mero+fluco), high dose glucocorticoids (250 mg predni)

4.) fever↓, CRP↓, general aspect↑, stable paresis

5.) post-hospital: glucocorticoid-induced myopathie, change to MMF, persistent hyoimmunoglobulinemia

Discussion:

Diskussion

Exacerbation of vasculitis:

+ history

+ steroids

- exacerbation during immunosuppression

- PET

ID:

+ laboratory findings

+ immunosuppression

- PET / imaging

- antibiotics

Conclusion

- In up to 43,9 % it is impossible to uncover the underlaying reason for FUO (1).
- In these cases it might make sense to empirically treat a potentially underlaying rheumatic condition and/or an ID in order to prevent more severe complications.

(1) **Gaeta, G. B., F. M. Fusco, and S. Nardiello.** 2006. Fever of unknown origin: a systematic review of the literature for 1995-2004. Nucl Med Commun **27:**205-11.