Haematology cases

Dr Sara Hall
PathWest
Female Case

Clinical details

• 52 year old female
• “Flow for clonal B-cells.”
• Sample: blood

Full blood count

<table>
<thead>
<tr>
<th></th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>143</td>
<td>115-160 g/l</td>
</tr>
<tr>
<td>MCV</td>
<td>84</td>
<td>80-100 fl</td>
</tr>
<tr>
<td>WCC</td>
<td>4.7</td>
<td>4.0-11.0</td>
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<tr>
<td>PLT</td>
<td>202</td>
<td>150-400</td>
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<tr>
<td>Neuts</td>
<td>2.4</td>
<td>2.0-7.5</td>
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<tr>
<td>Lymphs</td>
<td>1.7</td>
<td>1.2-4.0</td>
</tr>
<tr>
<td>Monos</td>
<td>0.5</td>
<td>0.2-1.0</td>
</tr>
<tr>
<td>Eos</td>
<td>0.12</td>
<td>0.2-1.0</td>
</tr>
<tr>
<td>Basos</td>
<td>0.07</td>
<td>0.0.2</td>
</tr>
</tbody>
</table>
Further clinical history

• Multiple lesions: Class-switch type cutaneous marginal zone B-NHL (electron beam radiotherapy).
• “difficult to explain” widespread low volume FDG avid nodes with monocytoid B-cell hyperplasia. Reactive? EBV driven.
• Lymphadenopathy since her mid 20’s
Flow cytometry: Blood

- 34% lymphocytes
  - 72% T-cells (CD4:CD8 29:30)
  - 10% B-cells (polyclonal)
  - 14% NK cells

WCC 4.7 X 10^9/L
Lymphs 1.7 X 10^9/L
No monoclonal B-cell population.
DN αβT cells
20% of T cells
15% of lymphocytes

so what????
Autoimmune Lymphoproliferative syndrome (ALPS)

- Genetic defect in lymphocyte apoptosis resulting in accumulation of lymphocytes
- Clinical hallmarks
  - Chronic non-malignant lymphadenopathy
  - Splenomegaly
  - Autoimmune manifestations
  - Multi-lineage cytopenias (immune and sequestration): HA>ITP>Neutropenia
- Commonly presents in childhood, but can present later
- Increased risk of lymphoma.
Revised diagnostic criteria and classification for the autoimmune lymphoproliferative syndrome (ALPS): report from the 2009 NIH International Workshop

Joao B. Oliveira,³ Jack J. Blesing,² Umberto Danzani,³ Thomas A. Fleisher,¹ Elaine S. Jaffe,⁴ Michael J. Lenardo,⁵ Frederic Rieux-Laucat,⁶ Richard M. Siegel,⁷ Helen C. Su,⁸ David T. Teachey,⁹ and V. Koneti Rao¹⁰

Table 2. Revised diagnostic criteria for ALPS

Required
1. Chronic (> 6 months), nonmalignant, noninfectious lymphadenopathy or splenomegaly or both
2. Elevated CD3⁺ TCRβ⁺ CD4⁺ CD8⁻ DNT cells (> 1.5% of total lymphocytes or 2.5% of CD3⁺ lymphocytes) in the setting of normal or elevated lymphocyte counts

Accessory
Primary
1. Defective lymphocyte apoptosis (in 2 separate assays)
2. Somatic or germline pathogenic mutation in FAS, FASLG, or CASP11

Secondary
1. Elevated plasma sFASL levels (>200 pg/mL) OR elevated plasma interleukin-10 levels (>20 pg/mL) OR elevated serum or plasma vitamin B₁₂ levels (> 1500 ng/L) OR elevated plasma interleukin-18 levels > 500 pg/mL
2. Typical immunohistological findings as reviewed by an experienced hematopathologist
3. Autoimmune cytopenias (hemolytic anemia, thrombocytopenia, or neutropenia) AND elevated immunoglobulin G levels (polyclonal hypergammaglobulinemia)
4. Family history of a nonmalignant/noninfectious lymphoproliferation with or without autoimmunity

A definitive diagnosis is based on the presence of both required criteria plus one primary accessory criterion. A probable diagnosis is based on the presence of both required criteria plus one secondary accessory criterion.
No access to advanced molecular or functional testing?

Elevated TCRαβ DNT’s + elevated secondary biomarker (B12, sFASL, IL-10, IL-18)

Predicts FAS mutation with post-test probability of 85-97%

Elevated Vitamin B_{12} Levels in Autoimmune Lymphoproliferative Syndrome Attributable to Elevated Haptocorrin in Lymphocytes

TrueSight One sequencing panel

• 4,811 genes associated with known clinical phenotypes

• Targeted bioinformatics analysis of sub-panels of genes is based on a specific clinical phenotype.

• All reported variants are Sanger confirmed.
Massively Parallel Sequencing via TruSight One

Clinical Notes: ? Autoimmune lymphoproliferative syndrome.

Result: Variant likely to be pathogenic

Variant(s): LRG_13411(FAS): c.[749G>T];[749=] p.[(Arg250Leu)];[(Arg250=)]


Interpretation:

TruSight One panel sequencing, with bioinformatically targeted analysis of genes associated with autoimmune lymphoproliferative syndrome (ALPS) (genes listed above), has identified a heterozygous missense variant in the FAS gene, c.749G>T p.(Arg250Leu), in this patient.

The c.749G>T variant has not been observed in the gnomAD population variant database, indicating that it is not a common variant in the examined populations, and in silico analyses predict the variant to be disease causing. The variant is located in 2 functionally important domains, altering a highly conserved amino acid. The c.749G>T variant has been reported as pathogenic in LOVD, based on 1 submission. In addition, this variant overlaps the amino acid residue of a well described pathogenic variant (c.748C>T, p.R250X). Furthermore, two other missense variants which overlap the same amino acid (c.749G>A, p.R250Q and c.749G>C p.R250P) have been identified in patients with ALPS (Kuehn HS et al., 2011 J Immunol 186(10):6035-6043).

Therefore, the c.749G>T p.(Arg250Leu) FAS variant is classified as likely pathogenic particularly as it is consistent with the clinical information provided for this patient. Further supporting evidence such as functional studies or segregation/de novo data will be required to upgrade this variant.

Referral of this patient to Genetic Services is recommended for genetic counselling.

Resources:
None

Method:
Secondary analyses: Illumina BWA Enrichment (v2.1.1) Tertiary analyses: Illumina BWA (2.0.5), Cartagenia. Variant classification: ACMG guidelines (Richards et al. Genet Med 2015; 17(4):405-24). Reported variants are confirmed by an orthogonal technology. Test sensitivity: SNP >99%, INDELs >80%, mosaicism as low as 12.5% <70%.

RefSeq:
Cited in comments for reported variants.

Note:
At least 95% of targeted regions covered to average depth of at least 20 fold. Method does not detect epigenetic changes, large repeat expansions, variation in untargeted regions and large scale structural rearrangements.

Product Info:

Disclaimer:
Clinically relevant variants interpreted using existing evidence-future interpretation may change. Result assumes correct sample identity.
ALPS is genetic defect in apoptosis
Programmed cell death

- 1842 Karl Vogt (Neuchâtel)
  - development of the tadpole in the midwife toad. Notochord cells disappeared to be replaced by vertebral cells.
- 1885 Walther Flemming (Kiel)
  - Chromatolysis
- 1951 Glucksmann (Strangeways lab, Cambridge)
  - Planned cell death is normal and necessary
- 1972 John Kerr et al.
  “Apoptosis”
APOPTOSIS: A BASIC BIOLOGICAL PHENOMENON WITH WIDERANGING IMPLICATIONS IN TISSUE KINETICS

J. F. R. KERR*, A. H. WYLLIE AND A. R. CURRIE†

From the Department of Pathology, University of Aberdeen

Received for publication April 1972

* On study leave from the University of Queensland. Present address: Department of Pathology, University of Queensland Medical School, Herston, Brisbane, Australia, 4006.
* We are most grateful to Professor James Cormack of the Department of Greek, University of Aberdeen, for suggesting this term. The word "apoptosis" (ἀπόπτωσις) is used in Greek to describe the "dropping off" or "falling off" of petals from flowers, or leaves from trees. To show the derivation clearly, we propose that the stress should be on the penultimate syllable, the second half of the word being pronounced like "ptosis" (with the "p" silent), which comes from the same root "to fall", and is already used to describe drooping of the upper eyelid.
Apoptosis

• “a distinctly different mode of cellular death with ultra-structural features that are consistent with an active, inherently controlled phenomenon”

• 20-30 billion cells per day in an adult human!
Apoptosis—essential for normal immune system development

• Adaptive immunity
  – developmental stages of **positive and negative selection** in the thymus and bone marrow (e.g. affinity maturation, deletion of autoreactive B and T cells).
  – the **killing** of infected cells by cytotoxic lymphocytes in the periphery
  – Activation induced cell death (**AICD**) - removal of lymphocytes that have performed their function i.e. the threat has been neutralised.

• Two pathways
  1. **extrinsic** (**FAS**- death receptors and ligands)
  2. **Intrinsic** (mitochondrial). Intracellular release of pro-apoptotic members of the BCL2 family.
FAS=CD95
FADD= FAS Associated Death Domain

ALPS-related syndromes

Price et al
BLOOD. 27 MARCH 2014 • VOLUME 123, NUMBER 13
Genes Mutated in ALPS

- **unknown**: 23%
- **CASP10**: 6%
- **somatic FAS**: 20%
- **FASLG**: 1%
- **germline FAS**: 50%

ALPS-FADD rare
ALPS-FAS

- Most common is heterozygous dominant FAS mutation
  - Production of mutant FAS prevents trimerisation of the DISC
- Recessive forms
  - Absent mutant FAS produced
    - Large homozygous deletions of FAS gene.
    - Non-sense mutations
  - Heterozygous carriers are symptomatic
    - homozygotes (deletional) or compound heterozygotes often have a severe phenotype.
- Incomplete penetrance – discovery of somatic heterozygous dominant mutations in the DN T-cells only. These account for 15% of cases.

NB. FAS mutation ≠ ALPS. Clinical penetrance in <60%

DISC = Death inducing signal complex
Clinical considerations

Increased risk of lymphoma

• Hodgkin and non Hodgkin NHL
• 14 fold and 51 fold increase risk respectively

Autoimmune disease

• Haematologic
• Non-haematologic
  – Glomerulonephritis
  – Hepatitis
  – GBS
  – Uveitis. Iridocyclitis
  – Others

Panel Sequencing Shows Recurrent Genetic FAS Alterations in Primary Cutaneous Marginal Zone Lymphoma

Katja Maurus1,2, Silke Appenzeller1, Sabine Roth1,2, Jochen Kuper3, Simone Rost4, Svenja Meierjohann5,6, Panagiota Arampatzı6, Matthias Goebeler2,7, Andreas Rosenwald1,2, Eva Geissinger1,2 and Marion Wobser8

ALPS-FAS morbidity and mortality

1. Sepsis post-splenectomy
   – Splenectomy now not recommended
2. Lymphoma
   – Often refractory
   – Challenge in distinguishing the benign from the malignant.

Natural history of autoimmune lymphoproliferative syndrome associated with FAS gene mutations

Susan Price,1 Pamela A. Shaw,2 Amy Seitz,3 Gyan Joshi,4 Joie Davis,1 Julie E. Niemela,5 Katie Perkins,6 Ronald L. Hornung,6 Les Folio,7 Philip S. Rosenberg,8 Jennifer M. Puck,9 Amy P. Hsu,3 Bernice Lo,1 Stefania Pittaluga,10 Elaine S. Jaffe,10 Thomas A. Fleisher,6 V. Koneti Rao,1 and Michael J. Lenardo1

BLOOD, 27 MARCH 2014 • VOLUME 123, NUMBER 13
Questions?
Comments?
Take home points

• Always have a systematic way of looking at T-cell populations. Don’t pretend that the ones you can’t explain are not there!

• “paediatric” diagnoses can be made well into adulthood
Case 2

Clinical details

- 66 yo r female
- “? Myeloproliferative neoplasm”

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<thead>
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<th></th>
<th>Full blood count</th>
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<tr>
<td>Hb</td>
<td>111</td>
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<tr>
<td>MCV</td>
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<td>90</td>
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<td>Neuts</td>
<td>30.2</td>
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<tr>
<td>Blasts</td>
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<tr>
<td>Promyelo</td>
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<tr>
<td>Myelos</td>
<td>9.5</td>
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<tr>
<td>Metas</td>
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</table>

<table>
<thead>
<tr>
<th>Parameter</th>
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</tr>
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<tbody>
<tr>
<td>Hb</td>
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<td>0-0.2</td>
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<td>0-0.2</td>
</tr>
<tr>
<td>Metas</td>
<td>0-0.2</td>
</tr>
</tbody>
</table>
CT
Blood film

Leucoerythroblastic, dysplastic neutrophils, 2% blasts.
BM aspirate- aparticulate

Myelogram:
Blasts: 2%
Pros:  10%
Myelos: 1%
Metas:  22%
Neuts:  40%
Eryth:  7%
Lymphs: 13%
Monos:  4%
Plasma <1%
Eos: <1%
Basos: 1%
M:E 11.4
Reticulin

Masson’s trichrome
Other

- Iron stain
  - No ring sideroblasts

- Flow cytometry
  - Blast gate: 3%. All haematogones (5.5% of total nucleated cells)
  - No monoclonal or aberrant T-cell population
So far

• Hyperplastic and dysplastic granulopoiesis
• Blasts not increased, monocyte % not increased
• Splenomegaly
• Interesting large demarcated areas on the trephine with mixed cell infiltrate and collagen fibrosis.
• Differential diagnoses?
• Further investigations?
Cytogenetics
• Karyotype: 46XX
• FISH results:
• Nuc ish (CHIC2, PDGFRB, FGFR1, JAK2, ABL1, BCR)x2 [100].

Preliminary Molecular studies
• JAK2, CALR exon 9, MPL W515L/K mutations NOT detected.
• RQ-PCR BCR-ABL 0.000%
Immunohistochemistry

MPO

CD117

Tryptase
Differential Diagnoses

• Systemic mastocytosis with an associated haematologic neoplasm (SM-AHN)
  
  – SM-aCML
  
  – SM-MDS/MPN-U
  
  – SM- CMML – monocytes <10%
  
  – SM-CNL – too dysplastic, no CSF3R mutation.
### SM diagnostic criteria

<table>
<thead>
<tr>
<th>Major SM criterion</th>
<th>Multifocal dense infiltrates of MCs (≥15 MCs in aggregates) in BM biopsies and/or in sections of other extracutaneous organ(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor SM criteria</td>
<td>a. &gt;25% of all MCs are atypical cells (type I or type II) on BM smears or are spindle-shaped in MC infiltrates detected on sections of visceral organs</td>
</tr>
<tr>
<td></td>
<td>b. <em>KIT</em> point mutation at codon 816 in the BM or another extracutaneous organ</td>
</tr>
<tr>
<td></td>
<td>c. MCs in BM or blood or another extracutaneous organ exhibit CD2 and/or CD25</td>
</tr>
<tr>
<td></td>
<td>d. Baseline serum tryptase level &gt;20 ng/mL (in case of an unrelated myeloid neoplasm, item d is not valid as an SM criterion)</td>
</tr>
</tbody>
</table>

If at least 1 major and 1 minor or 3 minor SM criteria are fulfilled, the diagnosis of SM can be established.
aCML, BCR-ABL1− negative: diagnostic criteria

- **Blood**
  - WCC 60.0
  - 78% neutrophils and precursors
  - 4% monocytes
  - 2% blasts

- **Marrow**
  - 73% neutrophils and precursors
  - 4% monocytes
  - 2% blasts
<table>
<thead>
<tr>
<th>MDS/MPN-U diagnostic criteria</th>
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<tbody>
<tr>
<td>Myeloid neoplasm with mixed myeloproliferative and myelodysplastic features not meeting the WHO criteria for any other myelodysplastic/myeloproliferative, myelodysplastic or myeloproliferative neoplasm.</td>
</tr>
</tbody>
</table>

- <20% blasts

- Clinical and morphologic features of one of the categories of MDS

- Clinical and morphologic myeloproliferative features manifesting as a plt count of ≥450 X 10^9/L associated with bone marrow megakaryocytic proliferation +/- a WCC ≥ 13 x 10^9/L. *

- No recent history of cytotoxic or growth factor therapy that could explain the myelodysplastic/myeloproliferative features.

- No PDGFRA, or PDGRFB, or FGFR1 rearrangement and no PCM1-JAK2.

*isolated del(5q) are excluded
NGS panel

**Clinical Indication** – Systemic mastocytosis with associated haematologic neoplasm

**Sample Type** – Bone marrow aspirate

**Histological Features** – Haemodilute and panacillary aspirate. Mast cell aggregates with atypical morphology, marked granulocytic hyperplasia with left shift and disorganised erythropoiesis noted on the trephine, consistent with a diagnosis of systemic mastocytosis with an associated haematologic neoplasm, favoured atypical CML (refer to PathWest report, Lab ID P18-1270029M)

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**NGS PANEL REPORT**

<table>
<thead>
<tr>
<th>ASSAY</th>
<th>MYELOID GENE PANEL</th>
<th>MUTANTS DETECTED</th>
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</thead>
<tbody>
<tr>
<td>Genes</td>
<td>ASXL1 (exon 12), BRAF (exon 15), CALR (exon 9), CBL (exon 8, 5), CSF3R (exon 14, 17), DNMT3A (exon 23), EZH2 (exon 2-20), FLT3 (exon 14, 15, 20), GATA2 (exon 4, 5), IDH1 (exon 4), IDH2 (exon 4), JAK2 (exon 12, 14, 16), JAK3 (exon 13), KIT (exon 8, 10, 11, 17), KRAS (exon 2, 3, 4), MPL (exon 10), NPM1 (exon 11), NRAS (exon 2, 3, 4), RUNX1 (exon 4-9), SETBP1 (exon 4), SRSF2 (exon 14, 15, 19), SRSF2 (exon 1), TET2 (exon 2-21), TP53 (exon 2-11), U2AF1 (exon 2, 8), WT1 (exon 7, 8, 9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CEBPA NOT PERFORMED</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FLT3-ITD NOT PERFORMED</td>
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</table>

**SUMMARY**

Mutations in KIT, KRAS, ASXL1 and TET2 were detected in this bone marrow aspirate sample consistent with the diagnosis of systemic mastocytosis with an associated haematologic neoplasm. Please correlate with morphological, immunophenotypic and cytogenetic features.

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**VARIANTS DETECTED**

<table>
<thead>
<tr>
<th>Gene</th>
<th>Variant</th>
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<tbody>
<tr>
<td>KIT</td>
<td>NM_000222.2: c.2447A&gt;T; p.(Asp816Val)</td>
</tr>
<tr>
<td>KRAS</td>
<td>NM_033360.2: c.493G&gt;A; p.(Ala164Thr)</td>
</tr>
<tr>
<td>ASXL1</td>
<td>NM_015338.5: c.2702dup; p.(Ser803Ilefs*3)</td>
</tr>
<tr>
<td>TET2</td>
<td>NM_001127058.2: c.5541G&gt;A; p.(Trp18471)</td>
</tr>
</tbody>
</table>

---

**Rare. 5% MDS**

? aCML V

MDS/MPN-U

50-60% of aCML and CNL

20-30% of SM, 30% MDS.

mutTET2 + KitD816V = aggressive SM
Atypical chronic myeloid leukemia is clinically distinct from unclassifiable myelodysplastic/myeloproliferative neoplasms

Sa A. Wang,1 Robert P. Hasserjian,2 Patricia S. Fox,3 Heesun J. Rogers,4 Julia T. Geyer,5 Devon Chabot-Richards,6 Elizabeth Weinzierl,7 Joseph Hatem,8 Jesse Jaso,1 Rashmi Kanagal-Shamanna,1 Francesco C. Stingo,3 Keyur P. Patel,1 Meenakshi Mehrotra,1 Carlos Bueso-Ramos,1 Ken H. Young,1 Courtney D. Dinardo,6 Srdan Verstovsek,9 Ramon V. Tiu,10 Adam Bagg,8 Eric D. Hsi,4 Daniel A. Arber,7 Kathryn Foucar,6 Raja Luthra,1 and Attilio Orazi5

WHO 2008 criteria

-MDS/MPN 2004-2012 CMML, JMML, RARS-T excluded
-Total n=134
-aCML n=65
<table>
<thead>
<tr>
<th></th>
<th>aCML (n=65)</th>
<th>MDS/MPN-U (n=69)</th>
<th>P value</th>
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<tbody>
<tr>
<td>WBC $\times 10^9$/L MR</td>
<td>40.8 (13.8-227.1)</td>
<td>19.4 (1.5-98.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Increased LDH</td>
<td>48/57 (84.2%)</td>
<td>41/63 (65.1%)</td>
<td>0.0168</td>
</tr>
<tr>
<td>Platelets MR</td>
<td>87 (7-974)</td>
<td>190 (9-1040)</td>
<td>0.0020</td>
</tr>
<tr>
<td>Blood myeloid precs (%)</td>
<td>17 (10-65)</td>
<td>4 (0-45)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Blood blasts % MR</td>
<td>2 (0-17)</td>
<td>0 (0-13)</td>
<td>.0009</td>
</tr>
<tr>
<td>Dysgranulopoiesis</td>
<td>100%</td>
<td>52%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>M:E</td>
<td>9.4 [0.8-100]</td>
<td>6 [0.5-63]</td>
<td>0.0110</td>
</tr>
<tr>
<td>Organomegaly</td>
<td>29/65 (44.6%)</td>
<td>15/66 (22.7%)</td>
<td>0.0080</td>
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</tbody>
</table>

MR median range
N=177 total

comments/questions?
Case 3

### Clinical details

- 86 year old man
- Presents to ED with fever and LUQ abdominal pain
- Cholangitis secondary to common bile duct stones.
- Deranged LFT’s

### Coagulation screen

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal Range</th>
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<tr>
<td>PT</td>
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<td>10-14s</td>
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<tr>
<td>INR</td>
<td>1.3</td>
<td>0.8-1.2</td>
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<tr>
<td>APTT</td>
<td>26</td>
<td>23-35s</td>
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<tr>
<td>Fibrinogen</td>
<td>5.2</td>
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<tr>
<td>Platelets</td>
<td>173</td>
<td>150-400</td>
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</tbody>
</table>
Medical history
• Chronic renal failure
• Obstructive sleep apnoea
• IHD-CABGS 2002
• PVD with angioplasty
• Coronary angiogram 1988
• PPM 2002
• Appendicectomy
• Osteoarthritis
• Diverticular disease

Regular Medications
• Oral B12
• somac
• aspirin
• simvastatin
• spironolactone
• Oxazepam,
• ostelin
• amiodarone
• bicor
• lisinopril
• tobramycin eye drops
- Ceftriaxone + Metronidazole  IV
- Sphincterotomy
- Augmentin DUO (10 Days)
- More Ceftriaxone + Metronidazole  IV
- Still sick
## Coagulation profile

<table>
<thead>
<tr>
<th>Date</th>
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<th>21/05/09</th>
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<tbody>
<tr>
<td>PT (10-14s)</td>
<td>17</td>
<td>56</td>
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<tr>
<td>INR (0.8-1.2)</td>
<td>1.3</td>
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<tr>
<td>APTT (23-35s)</td>
<td>26</td>
<td>90</td>
</tr>
<tr>
<td>Fibrinogen (2.0-4.5)</td>
<td>4.1</td>
<td>4.2</td>
</tr>
<tr>
<td>Platelets (150-400)</td>
<td>175</td>
<td>163</td>
</tr>
</tbody>
</table>

Is this result real??
Pre-analytic checks

- Correct patient
- Correct tube
- Clot check
- Correct volume in tube
Smell the content of the tube

Part II project ?
What next?

TCT- normal

Mixing studies: no correction of 1:1 with normal plasma.
## Factor levels

<table>
<thead>
<tr>
<th>Factor</th>
<th>Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>FII (U/ml)</td>
<td>0.78</td>
</tr>
<tr>
<td>FV</td>
<td>0.04</td>
</tr>
<tr>
<td>FVII</td>
<td>0.57</td>
</tr>
<tr>
<td>FVIIIc</td>
<td>1.44</td>
</tr>
<tr>
<td>FIX</td>
<td>0.90</td>
</tr>
<tr>
<td>FX</td>
<td>0.66</td>
</tr>
<tr>
<td>FXI</td>
<td>0.73</td>
</tr>
<tr>
<td>FXII</td>
<td>0.69</td>
</tr>
<tr>
<td>PT (10-14s)</td>
<td>35</td>
</tr>
<tr>
<td>APTT (24-40s)</td>
<td>80</td>
</tr>
<tr>
<td>Inhibitor level</td>
<td>5.9BU/ml</td>
</tr>
<tr>
<td>LAC</td>
<td>ND</td>
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</tbody>
</table>