“CHANGE IS THE LAW OF LIFE….”
- JOHN F KENNEDY, 1953

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Conflict of Interest: Nil
CLINICAL CASE

- 19 yr old woman
  - Several weeks lethargy
  - Partner carrying to bed/toilet
  - 14kg weight loss in 3/12

- PMH
  - Anxiety

- DH
  - Propranolol

- Multiple GP visits
  - 20/6/14 vomiting
  - 26/6/14 vomiting
  - 01/07/14 TSH 14 pmol/L ↗
    Na⁺ 127 mmol/L ↓
    K⁺ 6 mmol/L ↑
  - 04/07/14 Started Thyroxine
  - 14/07/14 Unable to walk
ADMITTED TO LOCAL DGH

Observations
- Temp 39°C
- HR 120 SR
- BP 125/77
- GCS 11/15

Bloods
- Na⁺ 113 mmol/L ↓
- K⁺ 7 mmol/L ↑
- Ur 24 mmol/L ↑
- Cr 276 µmol/L ↑
- cCa 3 mmol/L ↑
- Hb 101 g/L ↓
- WCC 10 x10⁹/L

ABG
- pH 7.4
- pO₂ 12 kPa
- pCO₂ 3 kPa
- BE -8.4 mmol/L
- HCO₃ 17 mmol/L
- Lactate 1.8 mmol/L

DIAGNOSIS?
- Adrenal antibodies positive
- Other pituitary axes normal
PRIMARY ADRENAL INSUFFICIENCY
“ADDISON’S DISEASE”¹

¹Addison T, 1855
PROGRESS

- Initial treatment
  - Thyroxine stopped
  - iv Hydrocortisone
  - iv Fludrocortisone
  - iv Aciclovir + Cefotaxime

- Transfer to HDU
  - Invasive monitoring
  - Norad + Dobutamine
FURTHER PROGRESS

- Hypotensive and rising lactate despite inotropes + 8L iv fluid
- CT Abdo normal
- Echo
  - LVEDD 3.9cm
  - LVEF 10%
  - Normal valves + RV
- Transfer to regional unit
TRANSFERRED TO WYTHENSHAWE CTCCU

- ABG on 40% O2
  - pH 7.26
  - pO2 11 kPa
  - pCO2 2.8 kPa
  - HCO3 12 mmol/L
  - Lactate 5.7 mmol/L

- Differential diagnoses
  - Addison’s with concomitant acute fulminant myocarditis
  - Addisonian cardiomyopathy\(^1\)\(^-\)\(^7\)

- Bloods
  - Cr 139 µmol/L
  - AST 900 IU/L
  - INR 2.5
  - Trop 1693 ng/L

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1Krishnamoorthy A, ASAIO J 2013
2Sherlock M, Clin Endocrinol 2008
3Wolff B, Int J Cardiol 2007
4Wiltshire EJ, J Paediatr Child Health 2004
5Conwell L, J Paediatr Endocrinol Metab 2003
6Afzal A, Can J Cardiol 2000
7Derish M, Intensive Care Med 1996
MECHANICAL CIRCULATORY SUPPORT

- INTERMACS Profile 2 ("sliding on inotropes")
- IABP
- Milrinone, Noradrenaline and Vasopressin
POST-ECMO PROGRESS

- Myocardial biopsy
  - Oedematous
  - No infiltrate / fibrosis
  - No amyloid / iron / fungi

- Likely Addisonian cardiomyopathy

- Long-term HeartWare LVAD for "bridge-to-recovery" on day 4

- 25/7 ITU admission

- Re-explored for low flow - external compression of RV by sternum

- E.coli pneumonia
DISCHARGED
6 WEEKS POST-LVAD

• Discharge medications
  Aspirin 300mg  Warfarin (INR 2-3)
  Lansoprazole 30mg  Bisoprolol 10mg
  Candesartan 8mg  Spironolactone 25mg
  Hydrocortisone 25mg  Fludrocortisone 50mcg

• Plan
  • Await myocardial recovery then for LVAD explant

• Outpatient progress
  • Minimal LVAD support required (pump speed approx 2700rpm)
  • Walking along sea front in Blackpool and Scarborough
  • Weight normalised at 65kg
HAEMORRHAGE
3 MONTHS POST-LVAD

- HPC
  - LVAD “low flow alarm”
  - Drowsy
  - Menorrhagia

- Investigation
  - Hb 5 g/L
  - INR 3.6
  - Normal CT head

- Management
  - 4 unit transfusion
  - Gynae review
  - Norethisterone
CVA
8 MONTHS POST-LVAD

- March 2015
  - Admitted to DGH with LUL weakness
  - INR 3
  - Aspirin changed to Clopidogrel

- Recovery assessment delayed
RECOVERY ASSESSMENT - ECHO
10 MONTHS POST LVAD

“ON” PUMP

“OFF” PUMP
RECOVERY ASSESSMENT - ECHO
10 MONTHS POST LVAD

“ON” PUMP

“OFF” PUMP
14min mod Bruce protocol

Respiratory Quotient 1.02

VO₂ max 19 ml/kg/min (47% predicted)
RECOVERY ASSESSMENT - RHC

- PA mean (mmHg)
- Wedge mean (mmHg)
- Cardiac Index (L/min/m²)

Graph showing differences in measurements between On Pump (2700rpm) and Off Pump (1800rpm) conditions.
REPEAT RECOVERY ASSESSMENT
18 MONTHS POST-LVAD

![Graph showing data on PA mean (mmHg), Wedge mean (mmHg), and Cardiac Index (L/min/m²) on and off pump. The graph illustrates changes in measurements over time.]
**TRANSPLANT CONSIDERATION**

- Insufficient recovery
  - LVAD explant not feasible

- 100% HLA sensitisation from previous pregnancies

- 0% chance of transplant match
  - Transplant not feasible

- LVAD destination therapy by default

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1Kirklin JK, J Heart Lung Transplant 2015
2JHLT 2016;35(10):1149
DRIVELINE ISSUES
14 MONTHS POST-LVAD

• Itchy, sore driveline, skin breakdown
• Multiple swab +ve Staph Aureus
• Multiple antibiotics
LVAD EVIDENCE BASE

Improved survival\textsuperscript{1, 2}  
Improved functional capacity\textsuperscript{3}  
Improve QoL\textsuperscript{3}

\textsuperscript{1}Rose EA, NEJM 2001;345:1435  
\textsuperscript{2}Slaughter S, NEJM 2009;361:2241  
\textsuperscript{3}Rogers JG, JACC 2010;55:1826  
\textsuperscript{4}Kirklin J, J Heart Lung Transplant 2015;34:1495
LVAD EVIDENCE BASE

Improved survival\textsuperscript{1,2}

30% complication-free survival at 12 months\textsuperscript{4}

\textsuperscript{1}Rose EA, NEJM 2001;345:1435
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\textsuperscript{3}Rogers JG, JACC 2010;55:1826
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SUMMARY

• MCS improves survival in selected patients with severe heart failure

• Durable LVADs indicated as bridge to transplant or recovery (destination therapy currently under consideration in UK)

• Intensive monitoring of LVAD recipients to minimise complications

• Exit strategy from MCS should always be considered