An Unusual Case of Multi-Organ Failure In Pregnancy

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CASE SUMMARY

DAY 1
28 year old woman, 29 weeks pregnant, self presents with a 3 day history of diarrhoea and vomiting and lower abdominal cramps.

Past medical history:
Nil.
Uncomplicated birth 2008

Examination and observations
Nil to find on examination other than lower abdominal tenderness
RR 17, Sats 99% on air, Temp 37.9, Pulse 110, BP 130/60
Temp 39.1 later on in day, commenced “Sepsis 6” and started on IV Co-Amoxiclav and Metronidazole

Initial Investigations
WCC 8.6
Neutrophils 7.4
Lymphocytes 0.8
CRP 119
Albumin 24
(other routine bloods normal)

DAY 2-8
- Remained tachycardic and pyrexial
- CTPA showed consolidation, no PE
- Worsening upper abdominal pain
- Thoracic USS: no collection
- Day 6 changed to IV Tazocin and Erythromycin
- CRP max 288
- Persistent lymphopenia
- Deranged liver function tests
- Day 7 changed to Amoxicillin, Temocillin and Clindamycin
- Hepatitis and HIV screen negative
- 13 sets of blood cultures: no growth

DAY 9
Acutely confused
CT head normal
Patient underwent emergency c-section and transferred to ICU.
CT Abdomen performed: gross hepatic congestion

NEONATAL
Initially good progress. Antibiotics stopped as no clear bacterial infection.
Baby Day 8:
DIC, fulminant hepatic failure and death.
HSV type 2 PCR positive with high viral load.

Retrospective PCR on blood from mother:
HSV type 2 detected, extremely high level (CT Value 8)

DISCUSSION

Diagnosis: Disseminated HSV infection
Treatment: IV Aciclovir
- Prolonged stay (4 months)
- MRI head normal
- LP: HSV-2. Declined further LPs so monitored viraemia in blood
- Hypoalbuminaemia (<6)
- Pleural effusion requiring drain
- SBP
- C.diff infection
- Depression
- Physiotherapy
- Recovery

Primary disseminated HSV infection is a rare condition which presents with non specific symptoms such as fever, anorexia, nausea, vomiting, abdominal pain and often causes a pneumonia and marked rise in serum transaminase levels without jaundice 1.

It is usually associated with cell mediated immunodeficiency which can be seen in pregnancy (in 2nd and 3rd trimesters). This is thought to be due to immunomodulation rather than immunosuppression, where there is a decrease in CD4, CD8 and NK lymphocytes. This results in susceptibility to, and increased severity of infections that require cell mediated immune response, such as influenza, hepatitis E and malaria 2-3.

Disseminated HSV in pregnancy is uncommon and most cases are due to HSV-2. There is often an absence of mucocutaneous lesions 4. Mortality rates can be as high as 75%, but despite high mortality HSV hepatitis is one of the few treatable causes of acute liver failure 5.

Treatment is with Aciclovir. There is no current consensus on duration of therapy however monitoring of detectable PCR can provide guidance on when cessation of therapy may be appropriate. Liver transplant should be considered and assessment suggested if there are risk factors for progression on presentation such as male gender, advanced age, immunosuppression and significant biochemical liver dysfunction 6.

REFERENCES