A rare but devastating complication of PD

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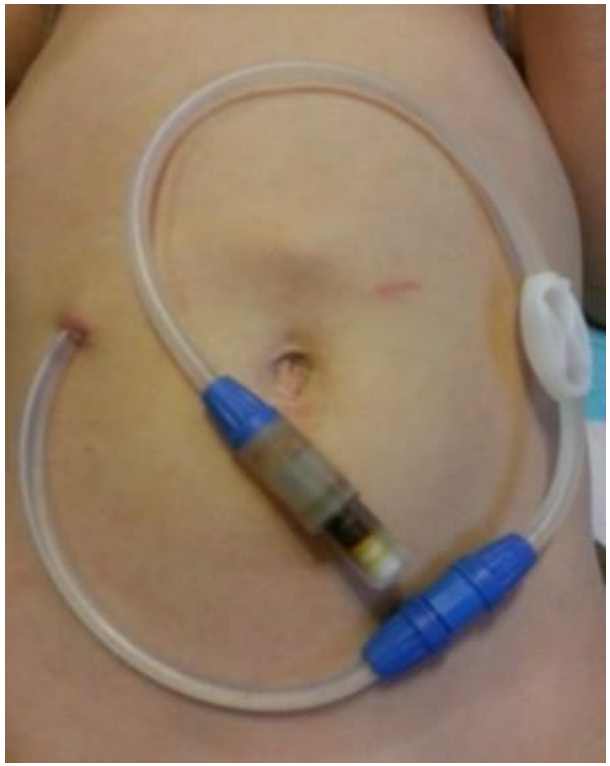


Outline

- Case
- Complications
- Literature
- Key learning points
- Summary

Case

- 9m M
- Antenatal Dx R MCDK, L dysplastic kidney
- Term LSCS
- Renal impairment from birth, Mx conservatively
- 4/52 became anuric -> nephrostomy inserted L dysplastic kidney
- No improvement in renal function -> PD catheter, automated PD

Further PMHx

- CMPI
- Hypothyroidism - likely 2^{o} to iodine on PD site dressing
- Incarcerated R inguinal hernia - repaired 3m

Sequence of Events

- 8m presented to CAU, UHW with D&V
- BP 94/40
- ↓ 40g in weight since clinic appt 5/7 prior
- Mx initially continuous NG dioralyte, before recommencing Peptijunior feeds 12h later
- D/C home at 48h

Continued…

- 2/7 later, suspected infected cannula site on his foot

-> commenced on PO Flucloxacillin by GP

2nd Presentation to CAU

- 1/52 after d/c, presented to CAU with a 48h Hx of "vacant episodes" & lethargy

O/E:

A: Patent

B: RR 48, sats 100%

C: CRT 3s, HR 170, BP 70/42

D: drowsy but responsive to pain

BM 5.9

T 37.5^{o}C

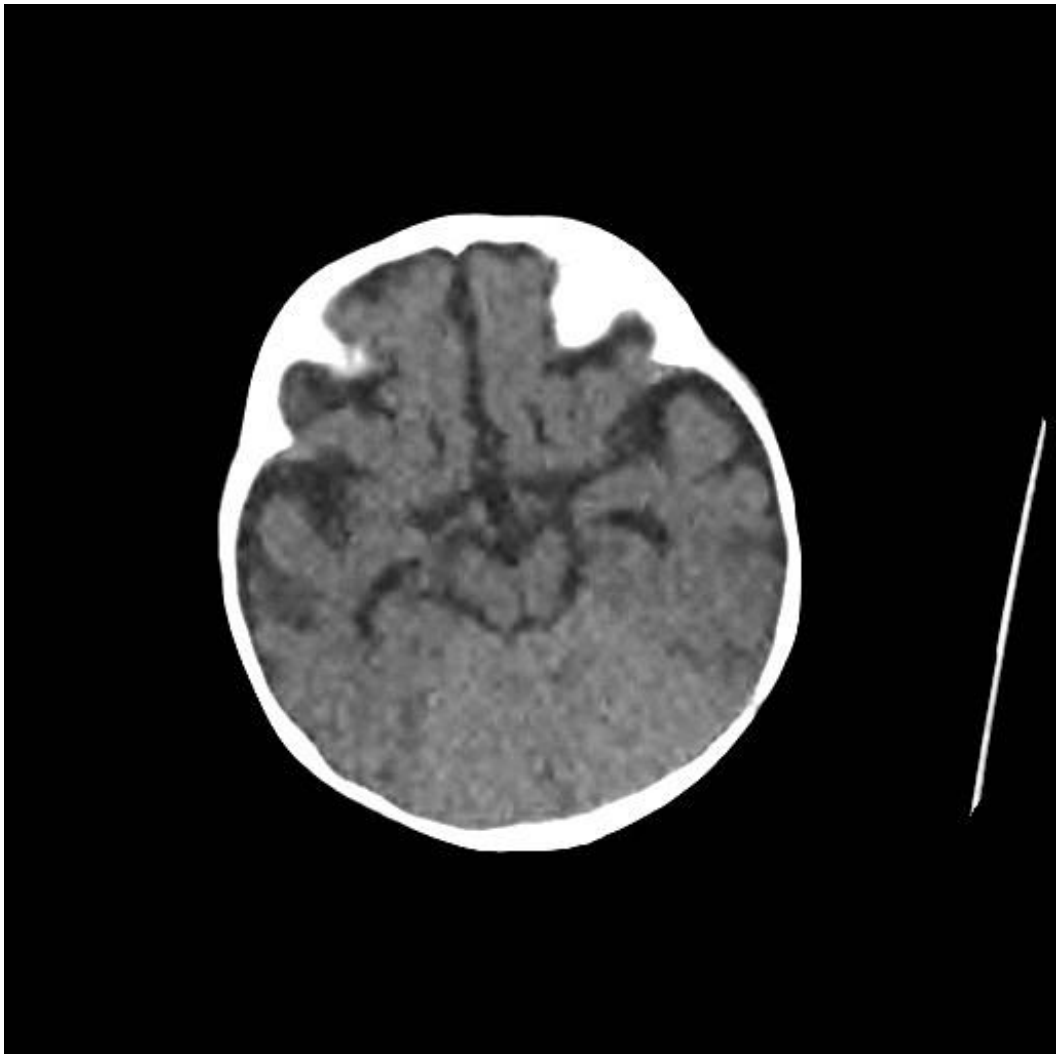
Initial Mx

- Fluid bolus 10ml/kg 0.9% Saline
- Full septic screen
- High dose IV Flucloxacillin
- Admitted to Pelican Ward

Investigations

- CRP 43
- Hb 130 WCC 22.9 Neut 13.6 Plt 191
- Cr 282 Ur 13.2
- BC –ve
- CSF –ve
- PD –ve
- Concerns about ongoing vacant episodes & ↓responsiveness -> CTB

CTB Image



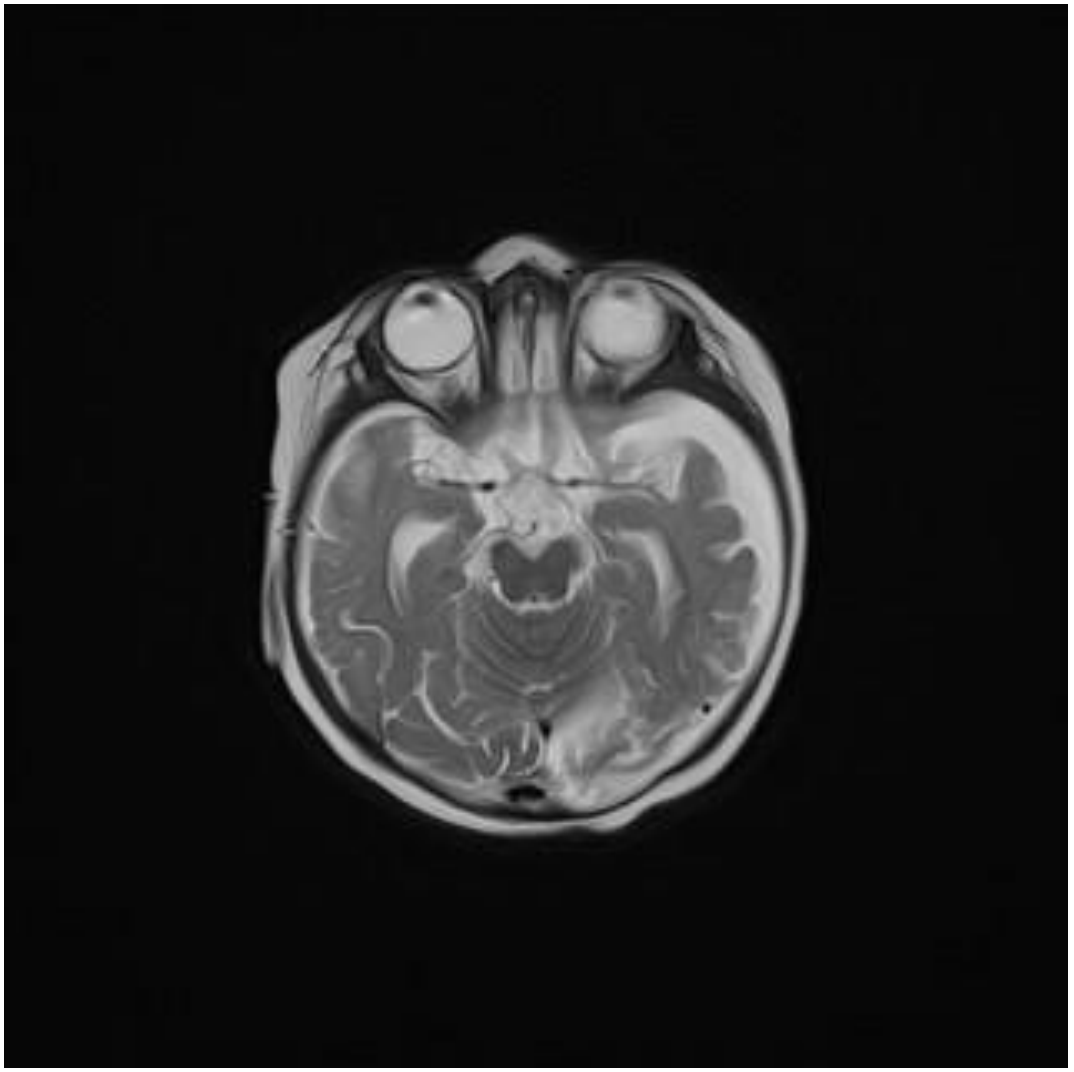
1/52 later…..

- Respiratory distress, profound hypotension (systolic of 20)
- Multiple fluid boluses T/F PCCU, I&V, Quadruple inotropes
- Hydrocortisone to maintain his MAP >35
- NPA: Rhinovirus +ve

Back on the ward….

- D/c from PCCU 1/52 later, MAP BP 35-40
- Concerns raised regarding vision, lack of fixing & following
- Also noted to have ↓ movement, ↑ tone & reflexes on R-side
- Commenced on Na supplements and Midodrine to optimise BP (Stabilised at 75-80 systolic)
- MRI Brain

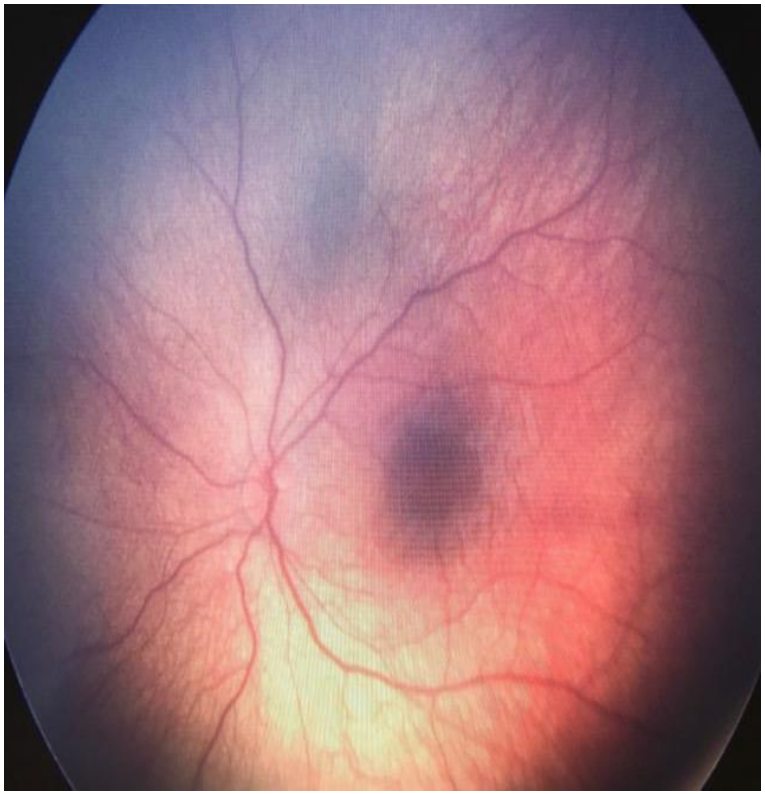
MRI Brain Image

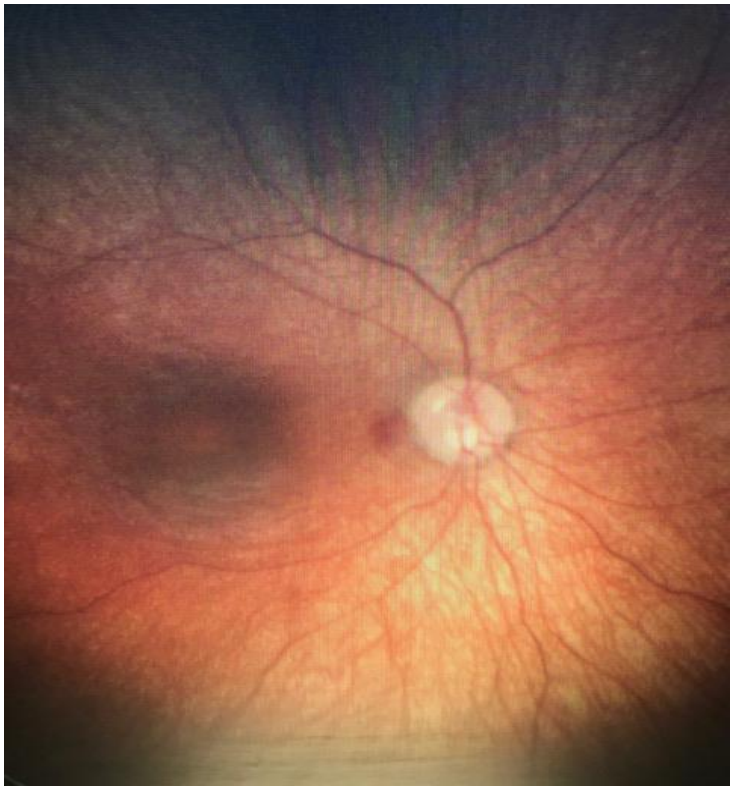


Ophthalmology Ax

- AION of LE and a significant degree of cortical visual impairment
- Commenced on low dose Aspirin (later discontinued due to gastric irritation)
- VEP testing which confirmed the findings
- Registered as severely visually impaired

Ophthalmology Images





On Discharge

- Resp: SVIA
- CV: Weaning dose of Midodrine
- Renal: Automated PD
- Neuro: Great improvement in mvt of RUL/RLL
- Ophthalmology: Registered as severely visually impaired

Summary of Events

- TWO distinct episodes of profound hypotension
- Supported by Ophthalmology findings
- Pale, cupped optic nerve LE suggests a delayed presentation
- ?occurred during episode of D&V prior to admission - could account for 'vacant episodes'
- Likely cortical visual impairment occurred following profound hypotension prior to PCCU

Literature

- AION rare in children
- Has previously been described in those on APD with hypotension 1%

Mechanism:

- 1. Hyponatraemic hypovolaemia related to 1^{o} disease
- 2. Ultrafiltration causing net Na loss

Continued…

- Hypoperfusion of post ciliary arteries – supply optic nerve head -> AION

- Vidal et al, Dufek et al

Key Learning Points

- Patients on APD complex to manage
- Profound hypotension is a rare but devastating SE of APD
- Early recognition of water & salt depletion is essential to help prevent ischaemia

Summary

- Case, sequence of events
- Imaging
- Literature
- Key learning points

Thank you