Royal Cornwall Hospitals NHS
NHS Trust

Brain or the kidneys?
Nephrogenic Diabetes Insipidus with loss of Pituitary brightness on MRI

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Background:

❖6 month old boy with chronic vomiting and severe weight faltering (birth weight 50th to 75th centile dropped to 0.4th centile)

❖originally attributed to gastro-oesophageal reflux was admitted after a period of poor urine output and found to have severe hypernatraemia (Na 168mmol/l, K 4.1mmol/l, Urea 16.2mmol/l, Creatinine 54 umol/l) with high plasma osmolality (330mosm/kg) and inappropriately low urine osmolality (130mOsm/Kg).

❖Renal USS was normal with slightly small kidneys.

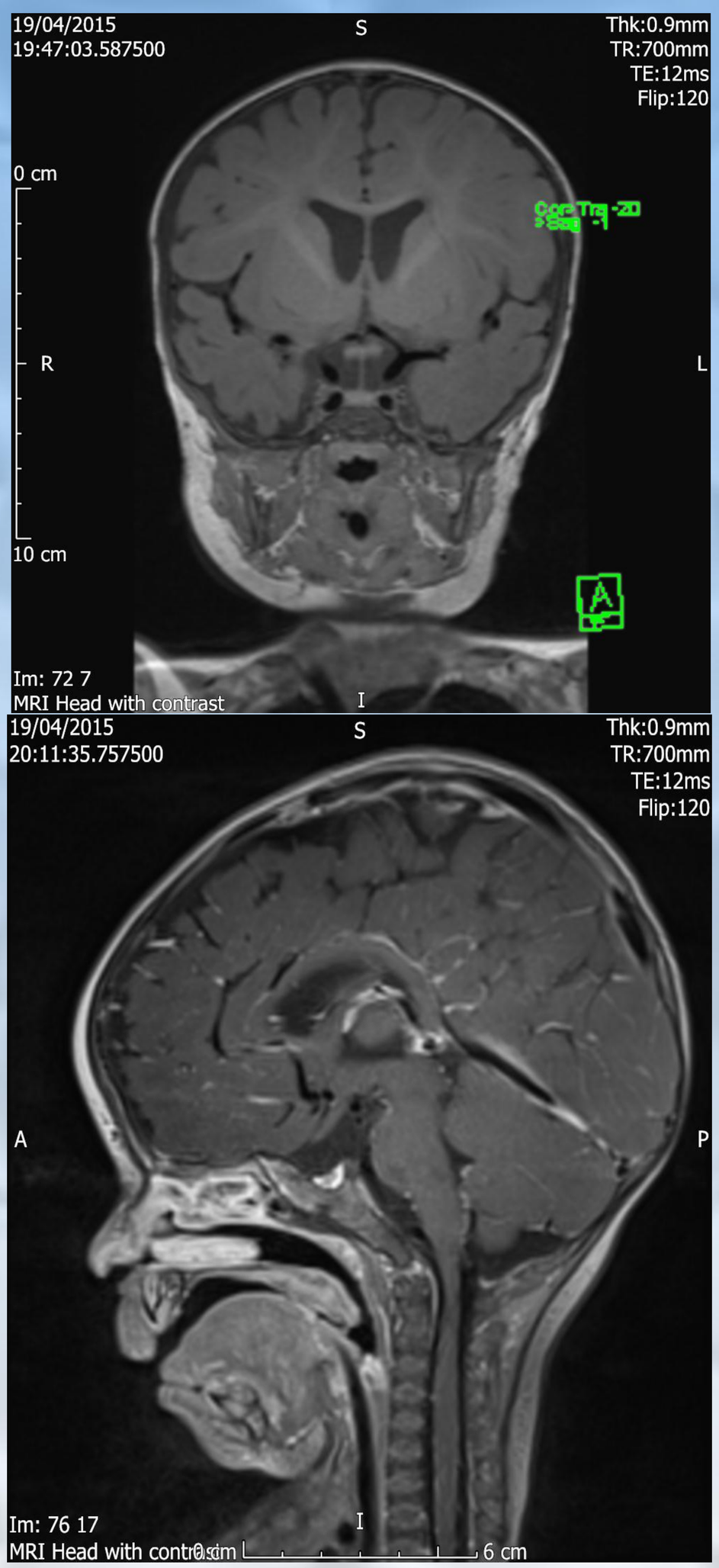
Discussion:

❖Thyroid function tests were TSH 2.7 miu/l & free T4 9.2 pmol/l suggesting a pituitary problem and so hydrocortisone, thyroxine and DDAVP were commenced. Urine output was high at up to 8ml/kg/hour even after DDAVP introduction with eventual rise in Sodium to 187mmo/l.

❖An MRI Brain showed loss of posterior pituitary bright signal with prominent sulci (felt to be secondary to marked dehydration) supporting a diagnosis of central DI. DDAVP was continued, as the initial poor response to DDAVP was blamed on the acute renal insult. Electrolytes improved, this was felt to be due to fluid management rather than DDAVP response as polyuria persisted.

Methods:

Medline, Pubmed search



Results:

❖DDAVP trial:
- ❖Pre-DDAVP osmolalities: Urine : 143mOsmol/kg, Serum: 301mOsmol/kg.
- ❖Post DDAVP urine osmolality: 142mOsmol/kg.

❖ A Standard Synacthen test showed normal peak Cortisol response (789nmol/l) and repeat Thyroid function tests were TSH 3.39 miu/l & free T4 17.4 pmol/l so Hydrocortisone and Levothyroxine were stopped (previously borderline FT4 was ascribed to sick euthyroid).

❖A diagnosis of Nephrogenic DI was made and DDAVP was stopped. Chlorothiazide and Amiloride have produced gradual improvement of polyuria and weight on low Renal solute formula alongside liberal intake of water (180mls/kg/day). Indomethacin was discontinued because of vomiting.

Conclusion:

❖ The loss of posterior pituitary bright spot is not always indicative of cranial DI as it can occur after exhaustion of vasopressin reserves in a child with Nephrogenic DI.

References

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