Format Corrections

Fundamentals of Veterinary Clinical Pathology
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This listing: August 14, 2004
Previous listings: March 8, 2003

Pages 293 – 294: Error in outline levels *March 8, 2003*

Version in 1st and 2nd printings from bottom of page 293 to middle of page 294

D. Increased [UN] in serum or plasma (azotemia) (Table 8.2)
E. Decreased [UN] in serum or plasma (Table 8.4)
    1. Disorders that cause decreased urea synthesis
        a. Hepatic insufficiency
            (1) Extensive hepatocellular disease that results in marked reduction in functional hepatic mass (> 80% loss) and thus sufficient decrease in urea synthesis to cause a decreased [UN] and a corresponding increase in [NH_4^+]
            (2) Portosystemic shunt (congenital or acquired)
                (a) Less NH_4^+ is delivered to hepatocytes from the intestines.
                (b) There is less uptake of NH_4^+ by hepatocytes because of decreased functional hepatic mass due to atrophy, necrosis, or fibrosis.
        b. Urea cycle enzyme deficiencies (congenital, extremely rare)
    2. Disorders that cause increased renal excretion of urea
        a. When less H_2O is resorbed in the proximal tubules (e.g., due to glucosuria), less of the filtered urea is resorbed in the proximal tubules because the resorption of H_2O creates the concentration gradient for urea resorption.
        b. In central and nephrogenic diabetes insipidus, reduced ADH activity or response in the medullary collecting tubules results in decreased resorption of both urea and H_2O.
    3. Consequence: The amount of urea in the renal interstitial fluid may diminish. Because about 50% of the medullary hypertonicity is normally due to urea, the urea deficit may contribute to a reduced concentration gradient, impaired renal concentrating ability, and thus polyuria.

Correction: reduction in one outline level for this section

III. Increased [UN] in serum or plasma (azotemia) (Table 8.2)
IV. Decreased [UN] in serum or plasma (Table 8.4)
    A. Disorders that cause decreased urea synthesis
        1. Hepatic insufficiency
            a. Extensive hepatocellular disease that results in marked reduction in functional hepatic mass (> 80% loss) and thus sufficient decrease in urea synthesis to cause a decreased [UN] and a corresponding increase in [NH_4^+]
            b. Portosystemic shunt (congenital or acquired)

(1) Less NH_4^+ is delivered to hepatocytes from the intestines.
(2) There is less uptake of NH_4^+ by hepatocytes because of decreased functional hepatic mass due to atrophy, necrosis, or fibrosis.

2. Urea cycle enzyme deficiencies (congenital, extremely rare)

B. Disorders that cause increased renal excretion of urea
1. When less H_2O is resorbed in the proximal tubules (e.g., due to glucosuria), less of the filtered urea is resorbed in the proximal tubules because the resorption of H_2O creates the concentration gradient for urea resorption.
2. In central and nephrogenic diabetes insipidus, reduced ADH activity or response in the medullary collecting tubules results in decreased resorption of both urea and H_2O.

C. Consequence: The amount of urea in the renal interstitial fluid may diminish. Because about 50% of the medullary hypertonicity is normally due to urea, the urea deficit may contribute to a reduced concentration gradient, impaired renal concentrating ability, and thus polyuria.

Pages 512 – 513: Error in outline levels *August 14, 2004*

Version in 1st, 2nd, and 3rd printings at top of page 513

C. Defective absorption of cobalamin in ileum
1. Diseases that damage ileal mucosa: inflammation, villous atrophy, cytotoxic drugs, resection
2. Congenital deficiency of the receptor for the intrinsic factor/cobalamin complex in giant schnauzers;[16,17] a similar disorder may occur in border collies.[18]

D. Severe cobalamin deficiency in a cat with methylmalonic acidemia: Cause of the deficiency was not established but clinical evidence supported a congenital defect in cobalamin absorption.[19]

Corrected*: increase one outline level for this section*

2. Defective absorption of cobalamin in ileum
a. Diseases that damage ileal mucosa: inflammation, villous atrophy, cytotoxic drugs, resection
b. Congenital deficiency of the receptor for the intrinsic factor/cobalamin complex in giant schnauzers;[16,17] a similar disorder may occur in border collies.[18]

3. Severe cobalamin deficiency in a cat with methylmalonic acidemia: Cause of the deficiency was not established but clinical evidence supported a congenital defect in cobalamin absorption.[19]