Management of Acute Portal Systemic Encephalopathy

Pages with reference to book, From 133 To 134

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Portal systemic encephalopathy (PSE) is a syndrome composed of few non-specific reversible signs which include disturbances of mental state and neuromuscular abnormalities, portal systemic shunting, E.E.G. abnormalities, foetor hepaticus and hyperventilation. Raised blood ammonia levels is the most specific sign which confirms the diagnosis of PSE.

The precipitant, usually a protein, either endogenous i.e., bleeding in the gut, or exogenous, a proteinous food should be identified and stopped. Diuretics, sedatives, tranquilizers and hypoglycaemia also precipitate PSE. They should be avoided and corrected respectively.

Ammonia content should be reduced by the use of Catharsis and enema. Prolonged Catharsis can also precipitate coma by producing dehydration, hypokalemia and hyponatremia. Large volume of saline by mouth helps in the gut cleansing (Levy et al., 1976), but it should be used cautiously in cirrhotics. Acetic acid 0.25-1 .0% as one litre enema is good in trapping ammonia by its local action (Conn and Lieberthal, 1980). Soap suds and alkaline enemas should never be used, as they drive the ammonia from the lumen into the blood.

Dietary proteins should not be restricted to less than 10-20 grams/day more than a day or two; as protein catabolism may worsen the situation. If possible, tube feed the patient on a low protein blanderized diet. Increase the protein content as the encephalopathy improves. A constant 10% glucose infuson will check the obligatory protein loss and assure adequate carbohydrate intake all day.

Ammonia production can be suppressed by using antioiotics. Oral use of non absorbable antibiotics e.g., neomycin, in patients on a low or protein free food showed a decrease in the blood ammonia levels and recovery in PSE patients (Sherlock et al., 1956; Atterbury et al., 1976). It is also useful in the treatment of chlorthiazide induced PSE. Neomycin can also be used as enema in 2-4 grams initial dose. Absorbable antibiotics as ampicillin, tetracycline and metronidazole also reduce blood ammonia level (Meyers and Leiber, 1976; Morgan et al., 1982). Ampicilim is the drug of choice in azotemia induced PSE. Use of lactobacillus acidophilus in the suppression of ammonia formation is well known, hut it does not work well and rapidly in PSE.

Removal of ammonia from the blood has been tried by dialysis. Hemodialysis is effective in fulminant hepatic failure (Doyle, 1962; Keynes, 1968) and in azotemia induced PSE. Peritoneal or hemodialysis is of little or no value in the treatment of nitrogenous PSE. Cation exchange resins are helpful. Lactulose enemas are quite effective in the removal of ammonia from the body.

Biochemical cohversion of ammonia to less toxic substances has been tuied with Arginine, protamine and mono amine oxidase inhibitors but no fruitful results were seen. Arginine and glutamate together (Young et al., 1959; Tobe, 1961) are helpful in lowering ammonia levels and perhaps recovery from nitrogenous PSE. Ornithine and Ketoglutarate need clinical evaluation in the treatment of PSE.

L. Dopa in a controlled trial was associated with a rise in the oxygen consumption by the brain, but DEG failed to show any improvement. Use of L dopa even in most desperate conditions is unjustfied (Conn and Lieberthal, 1980).

Supportive therapy in the form of prevention and control of infection, treatment of fever by inducing hypother.mia, correction of anaemia by transfusions and avoidance of 02 inhalations, should he carried out.

pH abnormalities e.g., metabolic alkalosis due to hypokalemia should he treated by high doses of potassium choloride. Respiratory alkalosis followed by metabolic acidosis due to prolonged and profound hyperventilation is best treated by rebreathing the expired air. Intravenous alkali in PSE patients is dangerous as ammonia toxicity increases in alkaline pH (Conn and Lieberthal, 1980).

Coma inducing agents e.g. sedatives, hypflotics and tranquilizers should be avoided. Diaphenhydramine (Benadryl) can be used for sedation. Phenobarbitone and oxazepam are good tranquilizers for PSE patients.

Desperate situations demand desperate sol u-tions and steroid therapy is often the last resort of the desperate physician. In Conn and LieberthaI's (1980) opinion corticosteroid therapy has no role in the maflagement of PSE.

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