Primary Adult Hypolactasla

Pages with reference to book, From 55 To 57

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The disaccharide lactose is the principle carbohydrate in the mammalian milk. In order to be absorbed and metabolized, it must be hydrolysed to its constituent monosaccharides i.e. glucose and galactose. The ability to hydrolyse dietary lactose is dependent upon the presence of a beta-galactosidase, lactase, which is found in the brush border of the enterocyte. High intestinal lactase activity is present during the suckling period in almost all mammalian species. After the time of weaning, its concentration declines by about 90% and is maintained at low levels thereafter[1]. Like other mammals, human infants have high concentration of intestinal lactase at birth which falls after the time of weaning resulting in a state called 'primary adult hypolactasia,. There is considerable variation in the onset of primary adult hypolactasia in various human populations[2]. The fall in lactase activity generally occurs between two and seven years[3]. Some human adults, however, retain the high concentration of intestinal lactase which is generally referred to as 'hereditary persistence of high intestinal lactase activity'. These individuals can hydrolyse large amounts of lactose and, thus can digest milk and other dairy products without any untowaid symp toms. In contrast, subjects with low intestinal lactase activity can absorb only small amounts of lactose. After ingestion of lactose, they usually complain of Juilness, rumbling sounds. and dis tension of abdomen followed by increased flatus production and watery diarrhoea. Evidence from family sioudies has shown taL this variation in intestinal lactase activity constitutes a genetic polymorphism, and persistence of liigh intestinal Jactase activity is due to an autosomal dominant allele.[4-6] Although it is not possible to induce lactase activity by giving lactose rich diet[7], the continued milk ingestion during early childhood might delay, for a few years, the onset of post-weaning hypolactasia[8,9].

Racial differences in the incidence of primary adult hypolactasia have been observed in different parts of the world. A high incidence is found among Atricans, Asians, American Negroes and Amerindians; on the other hand, Caucasians and certain nomadic tribes in Africa and Asia show a low incidence of primary adult hypolactasia.[10,11] By employing different techniques, frequency of primary adult hypolactasia among Pakistani adults is reported to be 45%[12] and 60%[13] respectively. It seems that primary adult hypolactasia is a relatively more ancient phenotype in the evolution of man and the allele for persistence of high intestinal lactase activity has attained the present high frequencies in some populations by natural selection.[11,14]

The definite diagnosis of lactase deficiency can be made by assay of lactase in jejunal biopsy specimen[15] This also allows histological examination of the specimen and assay of other brush border enzymes. Due to severe inconvenience for the subject and the necessity of special equipment for this method, indirect methods such as oral tolerance test[16] and breath hydrogen test[17] have been developed. Though oral tolerance test is a relatively simple and safe method, it is less sensitive as compared to others since it is influenced by velocity of gastric emptying and by the glucose metabolism of the subject. The breath hydrogen test is a reliable and noninvasive method, though it requires equipment for gas chromatography which is expensive and not widely available in Pakistan. This handicap, however, can be overcome by sending the breath samples in cans to a distant laboratory for analysis. This method is perhaps ideal for field studies.

Primary adult hypolactasia should not be confused with congenital lactase deficiency and secondary lactose malabsorption. While primary adult hypolactasia is a physiological state and the most commonly observed type, the other two are pathological entities. Congenital lactase deficiency is a rare

disorder[18] and the patients suffer from diarrhoea and fail to thrive if their diet includes lactose. Secondary lactose malabsorption is due to an acquired deficiency of lactase resulting from damage to the small intestinal mucosa in various diseases. Acute and chronic gastroenteritis protein-calorie malnutrition, coeliac disease, tropical sprue, cystic fibrosis of the pancreas and extensive gastrointestinal operations are common causes of secondary lactase deficiency[19] Secondary lactase deficiency is generally transient and is accompanied by distinct histological changes in the intestinal mucosa and low activities of other brush border enzymes as well.

As lactose malabsorption can complicate clinical picture in a number of disease entities, physicians must be familiar with different aspects of lactose malabsorption. Possibility of lactose malabsorption should be considered in children with unexplained diarrhoea and recurrent abdominal pain[20-22] Lactose malabsorption may also masquerade as irritable bowel syndrome.[23] As about half of our population cannot hydrolyse lactose. cases of peptic ulcer and other diseases should be advised large amounts of milk with caution.

There are different opinions regarding desirability of milk consumption on the part of lactose malabsorbers. The question is of immense practical importance as milk is included in nutritional programmes of developmg countries by national and international agencies to control malnutrition. It is also frequently used in relief programmes. Some workers suggest that milk consumption should not be encouraged in lactose malabsorbers as it leads to inadequate absorption of a variety of important nutrients[24-26]. Others, on the other hand, argue that small or modest quantities of milk can be tolerated and can be nutritionally useful to lactose malabsorbers[27-29] Before long term effects of milk consumption in lactose malabsorbers are evaluated, it seems that the latter advice should be followed. In view of recent data on primary adult hypolactasia in Pakistani human populations, heavy emphasis on milk consumption in our culture should be reconsidered. Undue concern is shown by parents if the children refuse to drink milk without realizing that they may be lactose malabsorbers, Older children and adults with hypolactasia should be reassured that lactose malabsorption is not a pathologic condition and they should be encouraged to consume lowlactose dairy products such as curd, cheese and butter.

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