

Review Study

Association of NTDs with maternal and only mother serum vitamin B12 Association-A Review Study

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Abstract

Neural tube defects (NTDs) are one of the commonest birth defects in our clinical practices. Maternal nutritional factors are associated with increased chance of development of NTDs in offspring. Periconceptional folic acid supplementation can prevent up to three fourths of neural tube defects (NTDs). Some studies reported that there might be an association between maternal serum vitamin B12 levels and NTDs. Dietary vitamin B12 deficiency is a common problem in the Indian subcontinent including Bangladesh because of inadequate dietary intake, dietary habit and/or malabsorption. There is scarcity of study to establish relationship of vitamin B12 and NTDs.

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It would be beneficial to see the association of vitamin B12 and NTDs in a Bangladeshi population, which may help in deciding about vitamin B12 supplementation as a preventive measure of NTDs.

Keywords: NTDs; Maternal and Neonatal Serum; Vitamin B12 level

Introduction

Neural tube defects (NTDs) are a group of serious birth-defects that arise when the neural tube fails to develop into the brain and spinal cord during the first month of pregnancy. The neural tube usually closes by day 28 of prenatal life, and its incomplete or incorrect closure results in malformations called neural tube defects (NTDs) [1]. There is a spectrum of severity of NTDs from anencephaly, which is incompatible with life beyond the neonatal period, to spina bifida occulta, which may be asymptomatic. In the United States, the incidence of NTDs was estimated at 1 per 1,000 deliveries, anencephaly at 0.6-0.8 per 1,000 live births, and open spina bifida as 0.5-0.8 per 1,000 live births [2]. The incidence varies from 1/100 live births in certain regions of China to about 1/5000 live birth in Scandinavian countries. In Bangladesh prevalence of NTDs is 4.7 per thousand live birth. Some hospital based study showed, NTDs was found in 13.33% and 11.32% [3] of total birth defects enrolled their studies. Etiology of NTDs is considered multifactorial, with genetic, environmental and nutritional factors playing some role [4]. The nutritional status of pregnant women may influence the vulnerability to NTDs in the fetus. The search for other etiological factors, particularly nutritional factors, has been continuing. Maternal nutritional risk factors are associated with increased likelihood of development of NTDs in offspring including: folate deficiency in the periconceptional period, low maternal vitamin B12. Folic acid can prevent up to three fourths of neural tube defects (NTDs). Some studies suggest that nutrients other than folate may also be essential to neural tube closure and have a potential role in risk reduction, with vitamin B12 mentioned most often [5]. Some study showed, low maternal vitamin B12 level increase risk of NTD. Vitamin B12 is a necessary cofactor for two enzymes in DNA synthesis: folate-dependent methionine synthesis and folate-independent methyl malonyl CoA mutase. Maternal serum vitamin B12 status prior to and during pregnancy is strongly associated with cord blood concentrations of vitamin B-12 and total homocysteine as well as infant stores of the vitamin at birth. Moreover, fetal vitamin B12 accumulation during gestation is the major determinant of the B12 status of the newborn and of stores in infancy. Therefore, this hospital-based study was conducted with the objective to see the association of NTDs with maternal and neonatal serum vitamin B12 level.

NTDs with maternal and neonatal serum vitamin B12 level

NTDs represent one of the most common congenital malformations in neonate worldwide. They constitute a heterogeneous group of disorders that occur during the first few weeks of gestation involving specific elements of the neural tube and its derivatives [6]. The central nervous system (CNS) appears at the beginning of the third week as a slipper-shaped plate of thickened ectoderm, the neural plate, in the

mid-dorsal region in front of the primitive node. Its lateral edges soon elevate to form the neural folds.

Pathophysiology

Two distinct processes appear to be involved in the formation of the neural tube: primary neurulation and secondary neurulation (i.e. canalization). The neural plate and the notochord are formed during early embryonic development. The neural groove develops by the third gestational week. Subsequently, the neural folds form bilaterally (Figure 1).

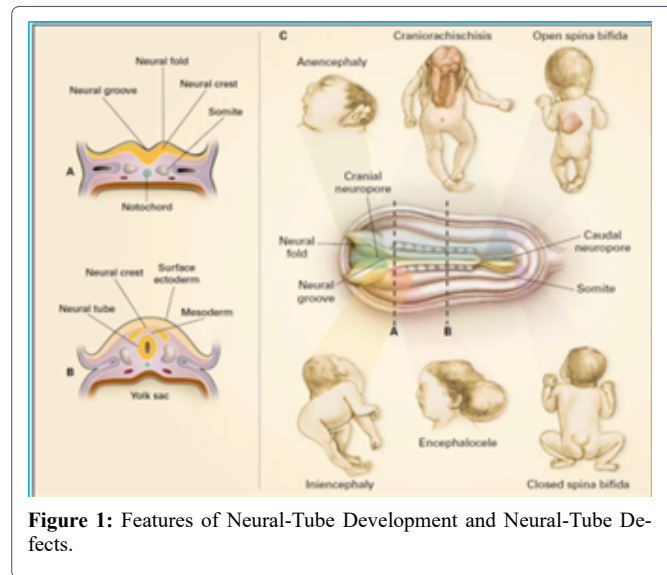


Figure 1: Features of Neural-Tube Development and Neural-Tube Defects.

Features of Neural-Tube Development and Neural-Tube Defects [7]. Panel A shows a cross section of the rostral end of the embryo at approximately three weeks after conception, showing the neural groove in the process of closing, overlying the notochord. The neural folds are the rising margins of the neural tube, topped by the neural crest, and demarcate the neural groove centrally. Panel B shows a cross section of the middle portion of the embryo after the neural tube has closed. The neural tube, which will ultimately develop into the spinal cord, is now covered by surface ectoderm (later, the skin). The intervening mesoderm will form the bony spine. The notochord is regressing. Panel C shows the developmental and clinical features of the main types of neural-tube defects. The diagram in the center is a dorsal view of a developing embryo, showing a neural tube that is closed in the center but still open at the cranial and caudal ends. The dotted lines marked A and B refer to the cross sections shown in Panels A and B. Shaded bars point to the region of the neural tube relevant to each defect. In anencephaly, the absence of the brain and calvaria can be total or partial.

Genetic disorders

While some neural tube defects may arise as a result of gene-environment interaction, there are a number of identified syndromes that also can give rise to neural tube defects. Some 60 syndromes that have a neural tube defect component together with other birth defects been identified. Each of the different types of neural tube defects may arise as a result of one or more of these syndromes. There is also evidence that non-syndrome neural tube defects may sometimes also have a genetic source, based on familial and twin studies.

Environmental exposures

Since neural tube defects are rare birth defects, it is hard to definitively associate an environmental exposure to a specific NTD. However, a number of studies have identified exposures that may cause neural tube defects. A selection of these is listed in Table 1, to give an idea of the range of exposures in the first trimester pregnancy that may give rise to neural tube defects. Occupational exposure to glycol ethers, pesticides, drinking water (hard water), cured meat, bilighted potato containing high concentrations of nitrates and lead are among the important exposures found to be associated with NTD. Maternal tea consumption has also been described in the etiology of NTD [8].

Maternal factors

General conditions

There are a number of maternal conditions that may contribute to an increased risk of an NTD-affected pregnancy. It was found that the incidence of NTD among infants of women who had a fever in the 1st trimester to be higher than control afebrile women. Though it was not clear whether the exposure to high temperatures or to the bacteria or virus is the cause of the maldevelopment of the neural tube [9]. Women with diabetes were found to be four times associated with the pregnancies involving neural tube defects (95 percent confidence interval [3.1, 5.1]) in comparison to the non-diabetics. However, the investigators found a much smaller odds ratio of 1.3 (95 percent confidence interval [1.0, 1.6]) of neural tube defect pregnancies among women with gestational diabetes. Obese women were found to be 3.5 times more, affected by NTD in comparison to non-obese. Maternal psychological stress may increase the risk of neural tube defects. It was reported that women who experienced death of someone close, job loss, separation or divorce - either themselves or someone close to them in the very early stages of pregnancy had an odds ratio of 1.5 (95 percent confidence interval [1.1, 2.1]) [10].

Maternal age

The literature reports associations between maternal age and the incidence of neural tube defects. Prevalence of NTD-affected pregnancies was found to be decreased with increasing maternal age. Meanwhile some other authors have found a U-shaped distribution of neural tube defects in relation to maternal age.

Maternal diet

Folic acid is a nutrient that is clearly needed for proper neural tube development. In 1992, the U.S. Centers for Disease Control and Prevention recommended that all women of childbearing age consume 0.4 mg of folic acid daily. This routine supplementation prevents the occurrence of NTD by more than 50% when it is taken before conception and continued throughout the first trimester of pregnancy. Women who have had a child with an NTD the recommendation for daily consumption of folic acid is 4000 µg. This supplementation in addition to folate in the diet before and during early pregnancy resulted in 71% reduction of recurrence of NTDs. The recommendation originated in research indicating that -inadequate levels of folic acid in the first weeks of pregnancy increased the risk of having a baby with a neural tube defect. In 1996, the U.S. Food and Drug Administration agreed that enriched cereal -grain products must be fortified with folic acid, as a means to reduce the rate of neural tube defects in the United States. There is evidence to suggest that excess lead (Pb) is causally related to NTD specially anencephaly [11,12]. Besides,

deficiencies of vitamin B12, zinc, selenium and have been described to be associated with increased incidence of NTD [13].

Vitamin B12 and Neural Tube Defects

Vitamin B12 deficiency was first described in 1849, and was considered to have a fatal outcome until 1926 when a diet of liver, high in vitamin B12, was shown to slow the disease process.

Function

Vitamin B12 also known as cobalamin, comprises a number of forms including cyano-, methyl-, deoxyadenosyl- and hydroxy-cobalamin. The cyano form, which is used in supplements, is found in trace amounts in food [14]. The other forms of cobalamin can be converted to the methyl- or 5-deoxyadenosyl forms that are required as co factors for methionine synthase and L-methyl-malonyl-CoA mutase. Methionine synthase is essential for the synthesis of purines and pyrimidines. The reaction depends on methyl cobalamin as a co-factor and is also dependent on folate, in which the methyl group of methyltetrahydrofolate is transferred to homocysteine to form methionine and tetrahydrofolate. Therefore vitamin B12 is also necessary for erythropoiesis and essential for normal neurodevelopment [15].

Food Sources

Vitamin B12 is synthesized by certain bacteria in the gastrointestinal tract of animals and is then absorbed by the host animal. Vitamin B12 is concentrated in animal tissues, hence, vitamin B12 is found only in foods of animal origin [16]. Foods that are high in vitamin B12 include: liver, kidney, beef and lamb, chicken, eggs, fish and milk, curd and dairy foods.

Recommended dietary allowance (RDA)

3 µg for adult, 0.5-1.5 µg for children and 4 µg for pregnancy and lactation.

Absorption

Vitamin B12 from dietary intake is bound to proteins and is released by hydrochloric acid that is secreted by the parietal cells from the stomach. The free form of vitamin B12 is then immediately bound to haptocorrins, which are glycoproteins secreted by the stomach and salivary glands. These haptocorrins protect vitamin B12 from chemical denaturation in the stomach. The parietal cells secrete intrinsic factor that binds to vitamin B12 and enables its active absorption. When the contents of the stomach enter the duodenum, the haptocorrins become partly digested by the pancreatic proteases. This causes them to release their vitamin B12. Immediately after they are released, they get bound to intrinsic factor in the duodenum. The vitamin B12-intrinsic factor complex then proceeds to the ileum, where it is absorbed by specific ileal receptors [17] (Figure 2). Metabolic reaction.

After entering the cell, folic acid, possibly with the aid of folate receptors, is involved in the transfer of carbon atoms that are used to synthesize nucleotides or, through the conversion of homocysteine to methionine, for the methylation of a variety of substrates. These processes are regulated by numerous molecules, including enzymes and vitamins other than folic acid (e.g., vitamins B6 and B12). The activity of some enzymes, such as methionine synthase, may be influenced by other enzymes, such as methionine synthase reductase. Enzymes and factors that may be important in neural-tube development are

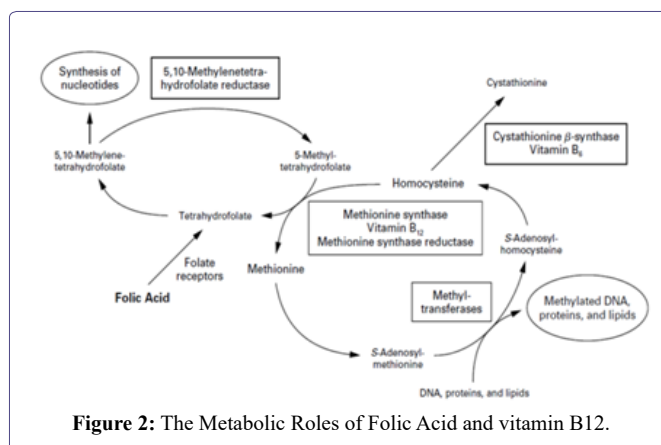


Figure 2: The Metabolic Roles of Folic Acid and vitamin B12.

indicated by boxes. Cobalamin is involved as a coenzyme in 2 enzymatic reactions. In the first enzymatic reaction, methionine synthetize produces methionine from homocysteine in the cytosol. In the second enzymatic reaction adenosylcobalamin is involved as a coenzyme in the production of methylmalonyl-co-enzym-A (methylmalonyl-CoA) to succinyl-CoA in the mitochondrion. A vitamin B12 deficiency results in elevated concentrations of homocysteine and methylmalonic acid in serum [17].

Vitamin B12 deficiency and pregnancy

Reported prevalence's of deficient plus low serum B-12 concentrations reported in Latin America are typically around 40% and reach 60 to 80% in some populations in Africa and Asia, affect all ages, and are associated with low animal source food consumption [18]. From the first through the third trimester of pregnancy there is a gradual decline in the serum concentration of vitamin B12 due to hemodilution, hormonal changes, alterations in the concentration of vitamin B12 binding proteins, and placental transport of vitamin B12 to the fetus. The lowest concentration of vitamin B12 is seen at 32 weeks of pregnancy and before delivery it increases again to reach a normal level after birth. The concentration of the active part of vitamin B12, holotranscobalamin, remains unchanged during pregnancy [19].

Types of Neural Tube Defects

The main types of neural tube defects (NTDs) are described below. The corresponding codes from the International Classification of Diseases -Ninth Revision Clinical Modification (ICD-9-CM) also are provided.

Anencephaly

Anencephaly (ICD-9-CM 740.0) occurs when the cephalic end of the neural tube fails to close, resulting in the absence of a major portion of the brain, skull and scalp. Many infants with anencephaly are delivered stillborn; those born alive are usually blind, deaf, unconscious and unable to feel pain. If the infant is not stillborn, then he or she will usually die within a few hours or days after birth.

Antenatal diagnosis and preventive aspects

The incidence of NTDs is declining throughout the world following the introduction of ultrasound examination and measurement of maternal serum alpha fetoprotein (MSAFP), amniotic fluid alpha fetoprotein (AFAFP) and amniotic fluid acetylcholinesterase (AFACHe) [20]. MSAFP is widely offered as a screening test for the detection of

NTDs in pregnancy in addition to a careful examination of the fetal brain and spine with ultrasound during an anomaly scan at 18-20 wks gestation. USG is used both as a screening test and as a follow up test after positive results an MSAFP screening. AFAFP and AFACH_E are employed primarily as a confirmation test [20].

Clinical assessment

Examination of the newborn baby should be performed by a clinician with appropriate experience. The width and length of the spinal lesion should be measured and its position relative to the spinal column recorded. Within the cystic lesion is the neural plaque, the maldeveloped spinal cord that is exposed on the surface. Immediately after birth, the nerve roots can be clearly seen through the CSF within the sac: they usually run horizontally or even upward because the physiological cranial migration of the cord was impeded during fetal life. The neural plaque may occupy the centre, or the upper or lower part of the whole lesion. A large plaque with a high upper level carries a bad prognosis, with extensive paralysis and analgesia [2].

Paralysis

There is a wide range of paralysis possible in the legs, from none to complete. Typically, the paralysis is of lower motor neuron type (flaccid) [2]. Knowledge (and a chart) of the segmental innervations of the leg muscles is helpful and the neurological level of paralysis may be ascertained in each leg separately and charted. The neurological level does not necessarily coincide with the vertebral level. All muscles below the uppermost affected segment may be paralyzed [21]. Another pattern also occurs, especially in thoracolumbar lesions, with flaccid paralysis of muscles deriving their innervations from the affected segments followed by reflex movements in muscles below the affected segments [22]. Reflex activity, which later disappears and may be followed by spasticity, should not be mistaken for useful voluntary movement. Stimulation of the legs may provoke reflex movements and mislead the examiner; it is better to stimulate the upper half of the body to elicit active movement in unaffected muscle groups.

The sphincters and the urinary tract

Almost all babies with myelomeningocele have paralysed urethral sphincters [2]. In the absence of normal sphincter control, urine can be expressed. An intravenous urethrograni or a renal ultrasound and a micturating cysto-urethrogram will show whether there is hydronephrosis, retention of urine, trabeculation or diverticulac in the bladder and/or ureteric reflux. The expressibility of the bladder and the residual urine can also be demonstrated. Other renal defects, such as single kidney, horseshoe kidneys; pelvic kidney and double pelvis and ureters are some ten times commoner than in infants without NTDs. The anus is not visible in normal babies without separating the buttocks, but when the muscles of the perineum and of the rectal sphincters are paralysed it is on the surface and patulous. Perianal rugae may still be present. The anal sphincter cannot contract on perianal stimulation or grip a small finger inserted into the rectum.

Hydrocephalus

The maximum head circumference must be measured and charted and related to the baby's weight and gestational age. Cranial ultrasound is an essential early investigation in myelomenmgocele and can reveal dilated ventricles before the head circumference enlarges. Ultrasound, computed tomography (CT) or MR imaging can also reveal the associated Chiari malformation, which can cause bulbar paresis. Treatment is insertion of a ventricular shunt.

Diagnostic evaluation

During the first hospitalization, the following tests should be done on most children with meningomyelocele. Scheduling these tests will vary depending on each situation [23].

Biochemical analysis

A 2- 3 ml blood sample was obtained from each subject of both groups by venipuncture and collected in a capped polypropylene tubes were labeled with the allocated number. Serum was separated by centrifugation (4000 rpm for 5 minutes), and haemolysed sample was discarded. The level of serum vitamin B12 was expressed in pgm/ml. Cut offs value for B12 that represented deficiency (<200 pg/ml), marginal deficiency (200-300 pg/ml), normal status (>300 pg/ml) [24].

Conclusion

Maternal folate deficiency can increase the probability of a fetus to have NTDs as compared to pregnancies with normal folate. Finding of lower folate levels in case mothers than in control mothers would have supported the use of folate supplements to prevent NTDs in low-risk populations. This finding focuses attention to the importance of nutritional factors in addition to genetic role in the etiology of neural tube defects.

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