

Review on Spina Bifida

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Abstract: *Spinal bifida is a congenital defect in the spinal cord of the fetus. The most serious forms of infection lead to spinal cord and spinal cord nerve injury, which has a significant negative impact on the life of newborns. Currently, prenatal procedures require abdominal laparoscopic surgery and uterine hysterectomy, which may have serious consequences and risks for mothers. Renal injury and renal failure are among the most serious complications of spinal cord injury. Over the past few decades, a comprehensive treatment strategy has been implemented to minimize renal injuries. Furthermore, most patients dry their urine after primary school.*

Keywords: Spinal bifida

I. INTRODUCTION

Spina bifida is one of the most common neural tube defects in a fetus. It is estimated that one in 1,000 children born in Western Europe suffers from a neural tube defect, with about 140,000 cases worldwide per year.^{1,2} Spina bifida is a non-neurological result of a spinal cord fall during pregnancy. This results in open neural code exposed to amniotic fluids and mechanical stresses directly on neuronal tissues. The most severe form is MMC, and the spinal cord is surrounded by a cystic bag filled with cerebral spinal fluid and a non-septic disease called Myelocysticism.^{3,4} Prenatal and postnatal operations can be considered for the treatment of Spina bifida. In order to prevent infection, post-birth surgery must be performed within 48 hours of birth, and children treated with this method have a 78% survival rate until the 17th year of their lives.^{5,6} Unfortunately, these repairs often require life-long support, resulting in neurological disorders, hydrocephaly and Chiari II mutilations.^{7,8} It has recently been found that minimal invasive approaches to the treatment of MMC are beneficial for uterine surgery and significantly reduce the risks of laparoscopic and open surgery.^{9,10} Neuron tube defects.¹¹

Types of Spina Bifida

Spina Bifida Occulta

occulta spinal cord is the lightest form of spinal cord, resulting from a fracture of one or more vertebrates, but spinal cord and meningitis are still limited to spinal canals. The spinal bifida occulta is a small hole (insinus gland) between two adjacent vertebrates that indicates that the vertebrates are not fused correctly; this defect can be demonstrated by small discs with hair spots or birthmarks on them.^{11, 12}

Spina Bifida Cystica

Spine bifida cystica Serious forms of Spine bifida include extrusion of the spinal cord and/or meningitis due to defects in the vesitonal axis, and are called Spine bifida cystica due to sacs similar to cysts associated with these abnormalities. When the bag contains meningitis and cerebrospinal fluid, spinal cord and spinal cord are in a normal position, this defect is called tOmeningocell. Despite their normal location, meningitis can be characterized by spinal cord abnormalitie.¹⁴

Embryology

The neural tube is a temporary structure formed during embryonic development. In human embryos, the entire process occurs between 17 and 28 days after fertilization.¹⁵ The activities required for the formation of the normal neuronal tube include apoptosis, motion of the neuronal head, proliferation of neuroepithelial cells, contraction of the upper cells and microfilaments and flexion at the dorsolateral bending points.¹⁶

Pathophysiology

The failure of the spinal cord during pregnancy during the third week results in an open neural tube defect (encephaly), an open spinal cord defect (myelomeningocele, or simply spina bifida), or both (craniorachisis), the hypothesis being that the open spinal cord defect and the resulting CSF leakage in the uterus cause a lack of degeneration of the primitive brain ventricular system, resulting in anatomical intracranial abnormalities^{17,18}

Epidemiological

Many epidemiological studies classified Spina bifida and related anencephaly defects and sometimes also encephaly defects under the general term neuronal tube defect (NTD). Box 1 lists pathological conditions that are generally considered to be NTD. The prevalence of NTD births has varied considerably over the past few decades, with still considerable differences between regions. For example, NTD is estimated to be 0.5–0.8 per 1,000 babies in the United States and many European countries.¹⁹ In the United States, for example, Spanish suffer from Spina bifida more frequently.²⁰

Risk Factor

In more than one study, many additional variables are mentioned as risk factors for Spina bifida²¹, and hyperinsulinemia may be associated with obesity and diabetes prior to or concurrently, thereby providing a possible link between the two maternal risk factors. Women who have undergone abdominal surgery to treat abdominal obesity may have increased the risk of having children with spinal cord diseases in women who have undergone abdominal bypass surgery.^{22, 23}

Diagnostic

Biochemical diagnosis

Prenatal diagnosis was first made possible in the early 1970s when high concentrations of AFP were found in amniotic fluid samples from encephalitis and MMC pregnant women.^{24, 25} AFP measurements in amniotic fluid samples can be useful in high-risk cases, but the probability of a 1% miscarriage after amniocentesis limited its more general application. In MMC, an increase in AFP concentration was observed in maternal serum samples.²⁶

In the late 1980s, the bipolar diameter of pregnancy was very small, and the symptoms of “lemon” and “banana” (Figure 4E-H) were described, as well as the advances in ultrasound and noninvasive diagnosis of MMCs and other TNSs.²⁷

Prevention

Folic acid prevention of NTD was described as a success in modern public health.³⁰ Nearly 40 years ago, Smithells and his colleagues found that women's diet and postpartum blood levels were relatively low for certain micronutrients, including folate,^{31,32}. Folic acid-containing multivitamin supplements reduced the risk of recurrence of NTD in women undergoing previous pregnancy.^{33,34} The underlying mechanisms by which folic acid helps reduce the risk of recurrence of NTD are Some countries have also observed a reduction in the prevalence of NTD after implementing voluntary folic acid supplements or strengthening programmes.³⁵

Management

POSTNATAL SURGERY

Management and postnatal surgery Neonates with spinal cord disabilities are best managed by beginning with imaging of the central nervous system and then measuring serial head to evaluate the growth rate of the head and the need for shunting. Almost all babies with chest injuries need ventricle-peritoneal shunts, while about 85 per cent of lumbar injuries and about 70 per cent of sacral injuries need shunts.³⁶

FOTAL SURGERY

Pregnant mothers with MMC diagnosis are subject to extensive prenatal tests and are considering surgery. These include pregnancy assessment, gene or chromosome screening (see section III), ultrasound for assessment of lower

extremity function, identification of foot disorders and estimation of spinal column levels, and cervical echocardiography for fetal echocardiography, and ultra-fast MRI for assessment of spinal cord damage, fibrosis, or other abnormal brain abnormalities.³⁷

II. CONCLUSION

SB is one of the most common congenital defects in the central nervous system, and continues to develop efforts to understand cell mechanisms of embryonic development. Although important steps have been taken to understand the phases of neurosis, we are not yet able to correct the deficits that cause NTD. We currently hope that new therapies and optimal medical and surgical management will improve the results and quality of life of these patients until they understand the complete biological basis of embryonic development, focusing on prevention rather than intervention, a choice of treatment.

REFERENCES

- [1]. C. E. Eduardo Castilla Vice-Chairperson Lorenzo D Botto, M. K. Bakker, V. Carlo Mirabello, P. Mastroiacovo, and S. Zezza Emanuele Leoncini Priscilla Carcione Lucia Mazzanti, "Annual report 2011 (with data for 2009)," International clearinghouse for birth defects surveillance and research (ICBDSR), Tech. Rep., 2010. [Online]. Available: www.icbdsr.org
- [2]. A. J. Copp, N. S. Adzick, L. S. Chitty, J. M. Fletcher, G. N. Holmbeck, and G. M. Shaw, "Spina bifida," Nature Reviews Disease Primers, vol. 1, 4 2015
- [3]. M. Meuu, C. Meuli-Simmen, G. M. Hutchins, C. D. Yingling', K. M. Hoffman, M. R. Harrison, and N. S. Adzick, "In utero surgery rescues neurological function at birth in sheep with spina bifida," NATURE MEDICINE, vol. 1, no. 4, 1995.
- [4]. M. Meuli and U. Moehrlen, "Fetal Surgery for Myelomeningocele: A Critical Appraisal," European Journal of Pediatric Surgery, vol. 23, no. 2, pp. 103–109, 2013.
- [5]. A. J. Copp, N. S. Adzick, L. S. Chitty, J. M. Fletcher, G. N. Holmbeck, and G. M. Shaw, "Spina bifida," Nature Reviews Disease Primers, vol. 1, 4 2015.
- [6]. Lee-Yang, Leonard J. Paulozzi, and C. Wong, "Survival of infants with spina bifida: a population study, 1979–94," Pediatric and perinatal epidemiology, pp. 374–378, 2001.
- [7]. J. P. Bruner, W. O. Richards, N. B. Tulipan, T. L. Arney, and D. Nashville, "Endoscopic coverage of fetal myelomeningocele in utero," Am J Obstet Gynecol, pp. 153–158, 1999
- [8]. Pippa Oakeshott, Gillian M Hunt, Alison Poulton, and Fiona Reid, "Open spina bifida: birth findings predict long-term outcome," Archives of Disease in Childhood, vol. 97, pp. 474–476, 2012.
- [9]. M. C. Dewan and J. C. Wellons, "Fetal surgery for spina bifida," Journal of Neurosurgery: Pediatrics, vol. 24, no. 2, pp. 105–114, 2019
- [10]. T. Vandebroek, M. Ourak, C. Gruijthuisen, A. Javaux, J. Legrand, T. Vercauteren, S. Ourselin, J. Deprest, and E. VanderPoorten, "MacroMicro Multi-Arm Robot for Single-Port Access Surgery," International Conference on Intelligent Robots and Systems (IROS), 2019.
- [11]. Campbell LR, Dayton DH, Sohal GS. Neural tube defects: a review of human and animal studies on the etiology of neural tube defects. Teratology 1996;34:171-
- [12]. Moore KL, Persaud TV. The developing human: clinically oriented embryology. Philadelphia (PA): Saunders; 1993.
- [13]. Muller F, O'Rahilly R. Human embryology and teratology. 2nd ed. New York: Wiley-Less Publications; 1996.
- [14]. Moore KL, Persaud TV. The developing human: clinically oriented embryology. Philadelphia (PA): Saunders; 1993.
- [15]. Volpe, J.J. Neural Tube Formation and Prosencephalic Development. In Neurology of the Newborn; Saunders: London, UK, 2008; Volume 889, pp. 5–8. ISBN 9781416039952.
- [16]. Blom, H.J.; Shaw, G.M.; den Heijer, M.; Finnell, R.H. Neural Tube Defects and Folate: Case Far from Closed. Nat. Rev. Neurosci. 2006, 7, 724–731

- [17]. Botto LD, Moore CA, Khoury MJ, Erickson JD. Neural-tube defects. *N Engl J Med* 1999;341:1509-19.
- [18]. McLone DG, Knepper PA. The cause of Chiari II malformation: a unified theory. *PediatrNeurosci* 1989;15:1-12
- [19]. International Center on Birth Defects. International Clearinghouse for Birth Defects Monitoring Systems (ICBDMS), Annual Report 2011 with Data for 2009. International Center on Birth Defects; 2011.
- [20]. Canfield MA, et al. Anencephaly and spina bifida among Hispanics: maternal, sociodemographic, and acculturation factors in the National Birth Defects Prevention Study. *Birth Defects Res A ClinMolTeratol.* 2009; 85:637–646.
- [21]. Little J, Elwood JM. Epidemiology of neural tube defects. In: Kiely M, ed. *Reproductive and Perinatal Epidemiology*. Boca Raton: CRC Press, 1991: 251–336.
- [22]. Haddow JE, Mitchell LE, Kloza EM, Thanhauser D. Neural tube defects after gastric bypass. *Lancet* 1986; 1: 1330.
- [23]. Martin L, Chavez GF, Adams MJ, et al. Gastric bypass surgery as maternal risk factor for neural tube defects. *Lancet* 1988; 1: 640–41.
- [24]. Dennis M, Landry SH, Barnes M, Fletcher JM. A model of neurocognitive function in spina bifida over the life span. *J IntNeuropsychol Soc.* 2006; 12:285–296.
- [25]. Brock DJH, Sutcliffe RG. Early prenatal diagnosis of anencephaly. *Lancet.* 1972; 300:1252–12
- [26]. Amniotic fluid acetylcholinesterase electrophoresis as a secondary test in the diagnosis of anencephaly and open spina bifida in early pregnancy Report of the Collaborative Acetylcholinesterase Study. *Lancet.* 1981; 318:321–324. [No authors listed.]
- [27]. Wald NJ, et al. Maternal serum-alpha-fetoprotein measurement in antenatal screening for anencephaly and spina bifida in early pregnancy. Report of UK collaborative study on alphafetoprotein in relation to neural-tube defects. *Lancet*
- [28]. Campbell S, Pryse-Davies J, Coltart TM, Seller MJ, Singer JD. Ultrasound in the diagnosis of spina bifida. *Lancet.* 1975; 305:1065–1068. [PubMed: 48732]
- [29]. Biggio, JR Jr; Owen, J.; Wenstrom, KD.; Oakes, WJ. Can prenatal ultrasound findings predict ambulatory status in fetuses with open spina bifida? *Am J Obstet Gynecol.* 2001; 185:1016–1020. [PubMed: 11717624]
- [30]. Buyukkurt S, et al. Prenatal determination of the upper lesion level of spina bifida with threedimensional ultrasound. *FetalDiagnTher.* 2013; 33:36–40. [PubMed: 22986465]