



Nipah Virus and Bacteria Infections: Cardiac Complications in Maternal Health

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Editorial

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Abstract

As a zoonotic virus, the nipah virus (NiV) can pass from people to animals. A novel paramyxovirus called NiV, which is related to the Hendra virus and has spread globally, has had tremendous impact on people's life. Numerous investigations have shown that NiV and SARS-CoV, the zoonotic virus that was responsible for the 2002 outbreak of severe acute respiratory syndrome, have a lot in common biologically. While NiV alone can also cause myocardial damage, pre-existing cardiovascular illness appears to be associated with worse results and an increased risk of death in individuals with the virus. Emerging infections include both newly emerging and re-emerging infections that have an increasing occurrence over the past few decades or threaten to do so. A new infectious agent entering the community, a previously unknown circulating agent being discovered, or the discovery that an infection is the true cause of a noncommunicable disease.

Keywords: Nipah Virus; Bacteria; Heart; Gestational Diabetes; iPSC-Derived Cardiomyocytes

Introduction

We highly value diversity, equity, and inclusion across a range of categories, such as color, ethnicity, sex, age, region, and more published in Circulation [1]. Even though Circulation frequently incorporates significant research on disparities across issues, for the past several years Circulation have featured an several issue specifically on disparities, which has allowed Rishi et al to compile a complementary collection of Original Research Articles, Research Letters, Frame of Reference pieces, and Editorials. This year, they are delighted to release their third annual themed issue, which offers important insights into the community, policy, and health system issues that influence cardiovascular health disparities across the country.

Scientists and medical professionals have worked to improve IVF (in vitro fertilization) as a method of treating

infertility for the past 20 years. However, numerous clinical studies are looking at the possibility that gestational diabetes can reverse the glucose intolerance that was initially noticed during pregnancy. With proof suggesting the benefits of such treatment, if any, were derived from cardioprotective paracrine substances generated by stem cells as opposed to early phases of embryonic development, a new paradigm was subsequently established. Researchers have subsequently attempted to explore circadian gene expression and maternal health in relation to cardiovascular safety [2]. It is noteworthy that differences are seen in congenital heart disease outcomes as well as in atherosclerotic cardiovascular disease. Using multicenter data from the Fetal Heart Society Research Collaborative, Lopez et al. discover that Black infants with a prenatal diagnosis of hypoplastic left heart syndrome had a roughly 2-fold higher risk of dying than White infants. These findings add to a growing body of information showing that Black infant death rates in the US

are unacceptable high [3].

Gestational Diabetes and Complications

According to the Gunderson et al study, Black and White women without a history of gestational diabetes who developed impaired glucose tolerance or overt diabetes within 15 years of giving birth had a progressively higher relative risk of coronary artery calcification in middle age (1.5- to 2.2-fold) compared to women who maintained normoglycemia. Women with normoglycemia, impaired glucose tolerance (prediabetes), or overt diabetes within 15 years of pregnancy had a relative risk of coronary artery calcification in midlife that was two times higher than women without a history of gestational diabetes who maintained normoglycemia. Maintaining normoglycemia may not reduce the risk of atherosclerotic cardiovascular disease in women who have had gestational diabetes in the past. Through routes including insulin resistance and decreased insulin secretion that promote atherogenic plaques independent of dysglycemia, a history of gestational diabetes may include underlying vascular alterations and negatively affect the development of cardiovascular disease. These results add to the accumulating evidence that better risk stratification of women for the early prevention of atherosclerotic cardiovascular disease is required among those with a history of gestational diabetes [4].

According to the Lane-Cordova et al study, Preterm birth or a small-for-gestational-age infant in the past increased the risk of atherosclerotic cardiovascular disease (ASCVD) in late middle age and increased the likelihood that a woman would experience significant vasomotor symptoms of menopause. In adjusted analyses of white women, a history of a preterm birth/small for gestational age infant and vasomotor symptoms were linked to an increased risk of ASCVD compared to no exposures. Women were more likely to have ASCVD risk scores below 7.5% if they also had vasomotor signs of menopause, preterm birth, or a small for gestational age child. The risk of ASCVD later in life was linked to reproductive events across the life course, particularly in white women. To better assess ASCVD risk, clinicians should ask patients about their complete reproductive history, including information on pregnancy and menopause [5].

When a woman enters menopause, her periods (menstruation) cease. Most frequently, it is a typical, natural body transformation that happens between the ages of 45 and 55. A woman can no longer get pregnant after menopause. A chronic infection of the heart's lining or valves that is largely bacterial in origin and can either be culture-positive or culture-negative is known as infectious endocarditis (IE). IE has been linked to *Staphylococcus* and *Streptococcus*

species most frequently. However, reports of IE being caused by other rare organisms like nutritionally variant *Streptococcus*, *Bartonella spp.*, *Coxiella burnetii*, *Tropheryma whippelii*, HACEK (*Haemophilus aphrophilus*, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, *Kingella kingae*) group and some of the fungi are on a rise. Rare organisms such *Abiotrophia defectiva*, *Gemella*, *Fingoldia*, *Propionibacterium humerusii*, and *Haemophilus parainfluenzae* have also been the cause of sporadic cases that have been documented in the literature Yallowitz, et al. [6]. The cardiovascular system can be impacted by a wide variety of infectious pathogens, including those that can develop spontaneous epidemics or spread through biological attack. Numerous of these infectious diseases have been present in specific regions of the world for millennia, either sporadically (e.g., botulism, tularemia) or endemically (e.g., viral hemorrhagic fevers), while others are relatively recent introductions (e.g., severe acute respiratory syndrome, or SARS, or Nipah virus) [7].

Potential Obstacles to Successful Clinical Application

Cardiomyocytes can be produced effectively and repeatedly from induced pluripotent stem cell (iPSC) technology thanks to recent advancements [8]. For the following reasons, using iPSCs in cell therapy is highly appealing. First, the vast self-renewal and differentiation capability of iPSCs enables the production of a significant quantity of the iPSC-derived cardiomyocytes (iPSC-CMs) necessary for a successful cell treatment. Second, because iPSCs are capable of self-renewal, desired genetic modifications can be made to them before differentiation [9]. Third, iPSC-CMs demonstrate beating cardiomyocyte characteristics, and it has been demonstrated that they engraft in the host heart and electromechanically pair to nearby native cardiomyocytes [10]. The adoption of allogenic iPSC-CMs may also assist avoid any potential immunogenicity of the transplanted cells because iPSCs are created from the somatic cells of the patient [11]. Zhu, et al.'s observation of ongoing myocardial regeneration in iPSC-CMs provides more evidence for the long-lasting efficiency of genetically engineered cell cycle activation. Although more investigation is required to fully comprehend the long-term implications of CCND2 overexpression in iPSC-CMs, Zhu et al.'s method of genetically resetting cardiac cell cycle activity appears to be a promising method for effective myocardial regeneration [12]. In a nonhuman primate model employing pigs, Mattapally et al. recently described allogeneic transplantation of iPSC-CMs. Although they saw the transplanted iPSC-CMs engraft successfully, recipient pigs experienced brief episodes of ventricular tachycardia [13].

Conclusion

To encourage screening and adherence to guideline-concordant care, health systems need to give priority to methods that advance equality in their local communities and offer a complete support system that includes multidisciplinary team members (social workers, community health workers). Researchers can test programs to enhance risk factor screening (for example, faith-based screening, pharmacist-led interventions in Black barbershops), address health-related social needs (for example, food insecurity, housing instability), and improve environmental factors that contribute to poor cardiovascular outcomes (for example, a lack of green space for regular exercise). We hope that the pieces in this editorial have brought attention to ongoing discrepancies and have sparked initiatives to alter the current situation so that the future “where we go next” is one where cardiovascular health parity is attained.

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