Short-term costs of preeclampsia to the United States health care system



Warren Stevens, PhD; Tiffany Shih, PhD; Devin Incerti, PhD; Thanh G. N. Ton, MPH, PhD; Henry C. Lee, MD; Desi Peneva, MS; George A. Macones, MD; Baha M. Sibai, MD; Anupam B. Jena, MD, PhD

BACKGROUND: Preeclampsia is a leading cause of maternal morbidity and mortality and adverse neonatal outcomes. Little is known about the extent of the health and cost burden of preeclampsia in the

OBJECTIVE: This study sought to quantify the annual epidemiological and health care cost burden of preeclampsia to both mothers and infants in the United States in 2012.

STUDY DESIGN: We used epidemiological and econometric methods to assess the annual cost of preeclampsia in the United States using a combination of population-based and administrative data sets: the National Center for Health Statistics Vital Statistics on Births, the California Perinatal Quality Care Collaborative Databases, the US Health Care Cost and Utilization Project database, and a commercial claims data set.

RESULTS: Preeclampsia increased the probability of an adverse event from 4.6% to 10.1% for mothers and from 7.8% to 15.4% for infants while lowering gestational age by 1.7 weeks (P < .001). Overall, the total cost burden of preeclampsia during the first 12 months after birth was \$1.03 billion for mothers and \$1.15 billion for infants. The cost burden per infant is dependent on gestational age, ranging from \$150,000 at 26 weeks gestational age to \$1311 at 36 weeks gestational age.

CONCLUSION: In 2012, the cost of preeclampsia within the first 12 months of delivery was \$2.18 billion in the United States (\$1.03 billion for mothers and \$1.15 billion for infants), and was disproportionately borne by births of low gestational age.

Key words: health care cost burden, hospital admission, maternal morbidity, maternal mortality, perinatal morbidity, preeclampsia, preterm birth

Preeclampsia is among the top 6 causes of maternal mortality, severe maternal morbidity, and adverse neonatal outcomes in the United States and globally. 1-4 Within the United States, the incidence of preeclampsia has risen steadily over the past 3 decades, from 2.4% of pregnancies in 1980 to 3.8% of pregnancies in 2010.^{5,6} The incidence of preeclampsia is rising, in part, because of recent trends to delay pregnancy to a later age and the increased rate of obesity in pregnant women in the United States.6,7

The classic definition of preeclampsia is characterized by hypertension plus proteinuria; however, preeclampsia can occur in the absence of proteinuria.^{8,9} In this case, hypertension may be accompanied by systemic organ involvement such as cerebral symptoms, abnormal coagulation, and liver involvement.¹⁰

The clinical manifestations of preeclampsia could develop at <28 weeks fetal condition.¹¹ The pathophysiological abnormalities in preeclampsia are characterized by endothelial dysfunction, 12-15 altered immunological adaptation, exaggerated inflammatory response, 16-19 increased coagulation with abnormal thrombin production, 20-22 increased oxidative stress, 23-25 genetic predisposition, 26 and reduced proangiogenic factors in association with increased antiangiogenic substances. 12,27-30 Indeed, studies have found that the levels of placental growth factor are reduced,³¹

(very early-onset preeclampsia), at

28–33 weeks (early-onset preeclampsia)

or after 34 weeks gestational age (GA). In

addition, the clinical effects of pre-

eclampsia may be limited to the mother

(usually after 37 weeks of GA), the fetus,

or both. Consequently, maternal and

perinatal complications from pre-

eclampsia will depend on GA at onset,

severity of maternal manifestations, and

It has been suggested that the imbalance in proangiogenic and antiangiogenic factors is responsible for hypertension, proteinuria, and increased

whereas the levels of soluble fms-like

tyrosine kinase-1^{32,33} and soluble endoglin³⁴ are elevated at the time of

diagnosis of preeclampsia.

capillary permeability. As a result, the levels of these factors and the ratio between soluble fms-like tyrosine kinase-1 and placental growth factor correlate with the severity of preeclampsia as well as the latency period from diagnosis until delivery.

The clinical presentation of preeclampsia is highly variable and may be characterized by sudden progression requiring delivery within hours or days, or it could remain stable and progress very slowly over weeks. Nevertheless, pregnancies complicated by preeclampsia are associated with substantial maternal and neonatal complications. These complications could happen acutely with complete resolution, or the mother and infant may be left with residual injury. In addition, neonates who survive are at increased risk for longterm deficits related to residual injury or as a result of fetal programming in utero. Moreover, some women who survive may be left with residual deficits as well as the effects of maternal vascular programming (Table 1).

From an epidemiological perspective, preeclampsia is growing at a rate more rapid than diabetes, ischemic heart failure, Alzheimer's disease, obesity, and chronic kidney disease, 35-37 diseases for

0002-9378/\$36.00 © 2017 Elsevier Inc. All rights reserved. http://dx.doi.org/10.1016/j.ajog.2017.04.032



Related editorial, page 235.



Click Supplemental Materials under article title in Contents at org

TABLE 1

Burden of early-onset preeclampsia in the United States

Major maternal morbidity and mortality

Major perinatal morbidity and

mortality

Acute: death and intensive care unit complications

- Eclampsia, stroke
- Pulmonary edema
- Myocardial ischemia
- Admission to intensive care unit
- Renal injury-failure with or without dialysis
- Abruption, disseminated intravascular coagulation
- Liver dysfunction, hematoma

Long-term: residual morbidity

- Neurological deficit
- Renal failure requiring dialysis
- Cardiomyopathy

Maternal programming caused by preeclampsia

- Coronary artery disease
- Chronic hypertension
- Metabolic syndrome
- Renal insufficiency
- Stroke
- Retinal dysfunction
- Premature death

Acute: death and neonatal intensive care hospitalization complications

- Respiratory distress syndrome and bronchopulmonary dysplasia
- Intraventricular hemorrhage, periventricular leukomalacia
- Necrotizing enterocolitis, late sepsis
- Retinopathy of prematurity
- Prolonged neonatal intensive care hospitalization

Long term: residual morbidity

- Cerebral palsy, neurological deficits, seizure disorder
- Learning disabilities
- Blindness and hearing deficits
- Chronic lung disease
- Chronic heart disease

Fetal programming: fetal origin of adult disease

- Metabolic syndrome
- Stroke, chronic heart disease, and others

Stevens. Short-term costs of preeclampsia in the US. Am J Obstet Gynecol 2017.

which substantial research and treatment funding have been allocated. This is notable because the rate of growth of early-onset preeclampsia in the United States has recently exceeded the rate in other high-income countries as well as several low- and middle-income countries.³⁸ Additionally, existing treatment options for preeclampsia are limited, despite both the scale of the disease burden and its high growth rate.

Several studies have estimated separately the burden of preeclampsia on mothers and infants, ³⁹⁻⁴³ while others have examined overall trends in preeclampsia by either maternal age at onset or by disease severity. 44-47 No study has simultaneously estimated the overall health and cost burden of preeclampsia by GA among both mothers and infants in the United States within the first 12 months after delivery.⁴⁸ This study estimated the annual epidemiological and health care cost burden of preeclampsia to both mothers and infants in the United States in 2012.

Materials and Methods Study design and data sources

We conducted a retrospective cohort study using secondary analysis of multiple data sets to estimate the national clinical and economic burden of preeclampsia in the United States. We required data that were nationally representative, longitudinal, linked mothers to their infants and included International Classification of Disease, ninth revision, codes to ascertain both outcomes and costs during the 6 months leading to the birth event and the 12 months after

Because no single data source met all of these requirements, we combined 5 of the data sets we reviewed (Appendix 1). To obtain mother-infant linkage and GA by week, we used data sources obtained by California Perinatal Quality Care Collaborative: the California Office of Statewide Health Planning and Development (OSHPD) data set linked to vital statistics and birth certificate data.49 The California OSHPD data provide administrative and billing information on nearly all hospitalizations in California and contains follow-up data on infant and maternal outcomes. Only 2% of births are not linked to vital statistics in the cases of home births without hospitalization, births in

Download English Version:

https://daneshyari.com/en/article/5675757

Download Persian Version:

https://daneshyari.com/article/5675757

<u>Daneshyari.com</u>