SYSTEMIC HYPERTENSION IN NEONATAL INTENSIVE CARE UNIT

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By

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LIST OF ABBREVIATIONS

A II	Angiotensin II
ACE	Angiotensin converting enzyme
ACTH	Adrenocorticotrophic hormone
AGA	appropriate for gestational age
ADPKD	Autosomal dominant polycystic kidney disease
ARPKD	Autosomal recessive polycystic kidney disease
BP	Blood pressure
BPD	Bronchopulmonary dysplasia
BUN	Blood urea nitrogen
BWS	Beckwith Wiedeman syndrome
CLD	Chronic lung disease
CAH	Congenital adrenal hyperplasia
CHD	Congenital heart disease
CHF	Congestive heart failure
CNS	Central nervous systems
CO	Cardiac output
COA	Coarctation of aorta
CT	Computed tomography
DMSA	Dimercaptosucdnic acid
DOC	Deoxy corticosterone
DTPA	Diethylene triamine pentacetic acid
ECG	Electrocardiogram
ECMO	Extra corporeal membrane oxygenation

EFS	Event free survival
FMD	Fibromuscular dysplasia
GI	Gastro-intestinal
HIE	Hypoxic ischemic encephalopathy
HTN	Hypertension
HVA	Homo vanillic acid
IVH	Intraventricular haemorrge
ICHge	Intracranial hemorrghe
LBW	Low birth weight
LDH	Lactate dehydrogenase
LV	Left ventricle
LVH	Left ventricular hypertrophy
MCDK	Multicystic dysplastic kidney
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
Na	Sodium
NAIDs	Non steroidal anti-inflammatory drugs
NICUs	Neonatal intensive care units
NSE	Neuron specific enolase
PKD	Polycystic kidney disease
MIBG	Metaiodobeniyl gunidine
PDA	Patent ductus arteriosus
PPA	Patent ductus arteriosus
PR	Peripheral resistance

RAS	Renal artery stenosis
RVHT	Renovascular hypertension
RVT	Renal vein thrombosis
SGA	Small for gestational age
TPN	Total parental nutrition
US	Ultrasonography
UAC	Umblical artery catheterization
UA	Umblical artery
VMA	Vanillylmandelic acid
VSD	Ventricular septal defect
VLBW	Very low birth weight
WT	Wilms tumor

Abstract

Blood pressure measurement became of great importance in modern neonatal intensive care units not only for detection of hypotension but also for monitoring of hypertension. There are many causes of neonatal hypertension, the most common are renal causes coarctation of aorta, congenital adrenal hyperplasia etc. Methods for blood pressure measurement include indirect non-invasive methods and direct invasive methods. Drug management of neonatal hypertension includes four categories. ACE, B blockers, diuretics and calcium channel blockers.

Our study is a retrospective review done to high light the occurrence of systemic hypertension among neonates admitted to the neonatal intensive care unit of the Children's Hospital, Cairo University, from January ٢٠٠٦ to December ٢٠٠٦.

Key Words: Neonatal hypertension, Renovascular HTN, Coarctation of aorta, BP measurement.

Introduction

Introduction

Recent advances in the ability to identify, evaluate, and care for hypertensive infants, coupled with advances in the practice of neonatology in general, have led to an increased awareness of hypertension in modern neonatal intensive care units (NICUs). ***(Flynn, 2006).***

Hypertension is not commonly diagnosed in newborn infants. The incidence in infants discharged from neonatal units ranges from 0.7% to 2%. ***(Watkinson, 2002).***

Hypertension is defined by a systolic blood pressure in a neonate which is >95 percentile for age and sex in 3 separate occasions for infants of similar gestational or postconceptual age and size. ***(Watkinson, 2002).***

Hypertension in newborn infants primarily is of renal origin, although cardiac, endocrine, and pulmonary causes have been described as well. ***(Flynn, 2006).***

The gold standard for blood pressure measurement is an appropriately calibrated intra-arterial catheter. However, for babies who do not have or require invasive monitoring the most frequently used technique is via an oscillometric manometer (e.g. Dinamap). Blood pressure should be taken when babies are quiet and not feeding (systolic BP is 5mmHg lower in sleeping babies) with appropriate sized cuff. ***(Flynn, 2006).***

Management should proceed in a stepwise fashion. First reduce or withdraw treatment that may raise blood pressure. If investigations reveal a surgically remediable cause, the timing of surgery depends not just on the nature of the lesion but also on the severity of the hypertension, the benefits and risks of medical treatment, and the age and weight of the baby. ***(Flynn, 2006).***

Aim of work

The aim of this study is to describe a standardized protocol for identifying hypertension in the neonate and to characterize the relationship of blood pressure with gestational age, postconceptual age, and birth weight and the need for blood pressure measurement in neonate who have left neonatal intensive care units. We also aim to explore the causes of the most cases of neonatal hypertension and to describe potential complications related to specific antihypertensive agents or too-rapid reduction in blood pressure.

REVIEW OF LITERATURE

Chapter 1

Physiology of neonatal circulation

Physiology of neonatal circulation

I. The Fetal Circulation:

In the adult circulation the right heart, the lungs, left heart and systemic circulation are arranged in series. Blood flow through each of these elements is identical, and equals cardiac output. In the fetal circulation, the right and left ventricles each pump blood into the arterial circulation in parallel In the fetus, the placenta provides for gas and metabolite exchange. The lungs do not provide gas exchange, and vessels in the pulmonary circulation are vasoconstricted. Three cardiovascular structures unique to the fetus are important for maintaining this parallel circulation: the ductus venosus, foramen ovale, and ductus arteriosus. ***(Eldestone, 1980)***.

The total fetal cardiac output (the combined ventricular output of both the left and right ventricles) amounts to about 450mL/kg/min. Approximately 65% of descending aortic blood flow returns to the placenta; the remaining 35% perfuse the fetal organs and tissues in the human fetus, which has a larger percentage of blood flow going to the brain, right ventricular output is probably closer to 1.3 times left ventricular flow. Thus, during fetal life the right ventricle is not only pumping against systemic blood pressure but is also performing a greater volume of work than the left ventricle is. ***(Rudolph, 1985).***

In preterm fetal, combined ventricular output is greater. (Approximately 550 ml/kg/min) ***(Iwamoto et al., 1989).***