Renal Protection Startegies During Surgical Obstetrical Patients Suffuring From Pregnancy Induced Hypertension

Essay
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By

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Abstract

Perioperative Assessment of Renal Functions is very important in patient with PIH Including urine out put (oliguria is considered when urine flow rate less than 0.5 ml/kg /hr, Blood Urea Nitrogen SerumCreatinine level BUN to Creatinine Ratio,creatinine clearance , Filtration Fraction (FF) and Cystatin C . Renal Tubuar function by Urine osmolarity , Urinary sodium level ,Urinary biomarkers as kidney injury molecule -1,interleukin -18 , and neutrophil gelatinase-associated lipocalin.and nephroscreen for predicting acute renal failurea acute renal failure is not

Immediately reversible so so prevention is the most effective way.

Key word

ARF , Ccr ,Bun, Endothelin – 1, hypertension, Perioperative , Renal physiology

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To

My parents,
My wife
My Sons
Youssef & Zeiad

List of Tables

List of Figures

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List Of Abbreviations

ACEI	Angiotensin Converting Enzyme Inhibitor
ADH	Anti Duritic Hormone
ANP	Anti Natruritic Peptide
ARF	Acute Renal Failure
ATN	Acute Tubular Necrosis
Ca	Calcium
CAD	Coronary Artery Disease
CAPD	Contnuous Ambulatory Peritoneal Dialysis
CHF	Chronic Heart Failure
CO	Cardiac Output
CRI	Chronic Renal Impairment
CRRT	Continuous Rnal Replacement Therapy
CVP	Central Venous Pressure
DDAVP	
ECF	Extra Cellular Fluid
ECG	ElectroCardiogram
EDRF	Endothelium derived relaxing factor
ET-1	Endothelin – 1
FF	Filtration Fraction
HCO_3	Bicarbonate
HO-1	Hemoxygenase
H_2O	Water
IHD	Intermittent Haemodialysis
IL-18	Interleukin-18
IPC	Ischemic Preconditioning
IV	Intravenous
K	Potassium
KCL	Potassium Chlorid
KIM-1	Kidney Injury Molecule-1
MAP	Mean Arterial Pressure
MS	Magnesium Sulfate
Na	Sudium
NAC	N-Acetylcystaien
NaCl	Sodium Chlorid
NGAL	Neutrophil Gelatinase Associated Lipocalin
NH_3	Ammonia
NO	Nitric Oxide
NORF	Non Oliguric Renal Failure

PACU	Post Anaethesia Care Unite
PAWP	Pulmonary Artery Wedge Pressure
PD	Peritoneal Dialysis
PIH	Pregnancy Induced Hypertension
RBC	Red Blood Cell
RCIN	Radiocontrast Induced Nephropathy
RRT	Renal Replacement Therapy
SICU	Surgical Intensive Care Unite
SNP	Sodium Nitroprusside
SVR	Systemic Vascular Resistance
TAL	Thick Ascending Limb
TNF	Tumor Necrosing Factor
TXA2	thromboxane A2
UNa	Urinary Sodium
UTI	Urinary Tract Infection
TXA2	thromboxane A2

Aim of the study

Proper preoperative management for obstetrical patients have a Risk for renal impairment by Identification of patients with high risk to Renal failure and Renal protective strategies for these cases .

Introduction

Pregnancy induced hypertension(PIH) is common morbidity during Pregnancy , many patient suffuring from PIH forms :

- Hypertension During Pregnancy.
- Pre-eclampsia.
- Eclampsia .

Renal impairment during pregnancy has many causes as:

- Urinary Tract infection (UTI) which is a very common comorbidity,
- PIH also a common disease during pregnancy,
- shock during Obstetrical Haemarrhages
- surgical complication (eg ...Uretiric injuries)

Renal function tests are needed especially during third trimester And mandatory in PIH patient in preoprative period to diagnose renal Impairment and for management .

Renal function tests are:

1- Glomerular filteration Rate: Blood urea Nitrogen (BUN), Serum creatinine BUN to creatinine Ratio, Creatinine clearance, Cystatinc, Filtration fraction.

2- Renal tubular function: urine osmolality, sodium conservation , new Urinary biomarkers .

3- Urine output : Renal evaluation of oliguria .

PREGNANCY-INDUCED HYPERTENSION

Pregnancy-induced hypertension (PIH) encompasses a range of disorders collectively and formerly known as toxemia of pregnancy, which includes gestational hypertension (nonproteinuric hypertension), preeclampsia (proteinuric hypertension), and eclampsia.

Occurring in 6% to 8% of all pregnancies, PIH is a major cause of obstetric and perinatal morbidity and mortality. The three principal mechanisms proposed as the etiology of PIH are:

- Vasospasm caused by abnormal sensitivity of vascular smooth muscles to catecholamines.
- Antigen-antibody reactions between fetal and maternal tissues during the first trimester
- that initiates placental vasculitis.
- An imbalance in the production of vasoactive prostaglandins (thromboxane A and prostacyclin), leading to vasoconstriction of small arteries and aggregation of platelets.

Preeclampsia is a multiorgan disease characterized by the development of hypertension with Proteinuria after the 20th week of gestation. It is a disorder of unknown etiology affecting approximately 8% of all pregnancies, with most cases occurring in the first pregnancy.

Hypertension is defined as a sustained systolic blood pressure of at least 140 mm Hg or a Diastolic blood pressure of at least 90 mm Hg. Proteinuria is defined as 300 mg or more of protein in a 24-hour urine collection. Edema may manifest as a recent, rapid weight gain. (1)

Severe preeclampsia includes at least one of the following features:

 Blood pressure: systolic BP 160 mm Hg or higher or diastolic BP 110 mm Hg or higher on two occasions at least 6 hours apart.
 Proteinuria: 5 g of protein or more in a 24-hour urine specimen (or 3+ to 4+ on Semiquantitative urinalysis)
 Oliguria: urine output less than 500 mL in 24 hours
 Cerebral or visual disturbances: headache, blurred vision, or altered consciousness
 Pulmonary edema or cyanosis
 Epigastria or right upper quadrant pain, which may be caused by stretching of Glisson's Capsule by hepatic edema
 Impaired liver function
 Thrombocytopenia: resulting from platelet adhesion to exposed collagen at sites of endothelial damage
 HELLP syndrome: hemolysis, elevated liver enzymes, low platelets
 Evidence of fetal compromise (e.g., intrauterine growth retardation, oligohydramnios) or

Placental abruption. (2)

Eclampsia is defined as convulsions and/or coma not caused by coincidental neurologic disease (e.g., epilepsy), which occurs during pregnancy or the puerperium in a woman whose condition also meets the criteria for preeclampsia.(2)

Risk factors for developing preeclampsia(2)

Hypertensive disease:

- Previous preeclampsia.
- Systolic hypertension during early pregnancy.
- History of chronic hypertension.
- Family history of hypertension during pregnancy.
- Increase in pulse pressure during the first trimester.

Coexisting vascular and endothelial disease:

- chronic renal disease.
- Lupus erythematosus.
- Protein S deficiency.
- Activated protein C resistance.
- Circulating anticardiolipin antibodies.

Obstetric factors:

- African-American race.
- primpara.
- Age older than 40 years.
- History of smoking.
- Obesity.
- Increased trophoblastic mass (e.g., multiple gestation, molar pregnancy).
- Large for gestational age fetus.
- Diabetes.
- Erythroblastosis fetalis.
- Polyhydramnios, particularly in young primigravidas.

The pathogenesis of preeclampsia

Preeclampsia is most likely a disease of heterogeneous causes of both maternal and placental origin.

a. Immunologic factors

Immunologic disorders may arise from an abnormal maternal-fetal antigen-antibody response or from the contents of seminal fluids; spermatozoa may cause antibody formation or prostaglandin may initiate uterine vasoconstriction. The fetus acquires 50% of its genes from the father, which represents in part a paternal allograft that interacts with maternal tissue as fetal trophoblast migrates into the maternal decidua after implantation. Migration normally occurs in two phases. Trophoblasts displace the muscular structure of the maternal spiral arteries before 20 weeks of gestation, causing their adrenergic denervation and converting them from high-resistance to low resistance vessels. At the same time, biochemical adaptations occur in the maternal Vasculature, with an increased dominance of endothelium-dependent vasodilators, prostacyclin (Prostaglandin I [PGI]), and nitric oxide (NO). In preeclampsia, the second wave of trophoblastic migration fails. This failure can result in a high-resistance, low-flow uteroplacental circulation and consequent placental ischemia and hypoxia. These changes may represent an aberrant Immunologic mechanism.(3)

b. Genetic factors

A familial tendency toward preeclampsia exists in some populations, and it may result from a recessive genetic inheritance.(2)

c. Endothelial factors

Vascular endothelial damage or dysfunction is the common pathologic feature of preeclampsia and occurs in the placental decidual vessels and renal microvasculature. Endothelial cell dysfunction in response to unknown factors may cause a hormonal imbalance in women with preeclampsia .

The metabolic end products of normal vascular endothelium include PaO2 and Endothelium derived relaxing factor (EDRF), which is either NO or a related nitrosyl substance.

Both PaO2 and EDRF are potent vasodilators. In patients with preeclampsia, the failure of trophoblast to invade the uteroplacental vascular bed may encourage an increased production of free radicals and lipid peroxides by the decidual lymphoid tissue. As a result, an imbalance occurs between the production of the vasoconstrictor thromboxane A2 (TXA2), which is derived from platelets, and the production of endothelium-derived PaO2. This imbalance results in a

reduced perfusion of the intervillous space. Some investigators have concluded that hypoxia induced impairment of NO production by the syncytiotrophoblast results in uteroplacental insufficiency and production of a toxin responsible for the clinical manifestations of preeclampsia.(3)

It has been shown that production of endothelin 1(ET-1), which is the most potent endogenous vasoconstrictor and produced mainly by vascular smooth muscle cells as well as by the vascular endothelium, is markedly increased in preeclampsia and correlates inversely with NO production. Therefore, the imbalance between NO and ET-1 may play a significant role in the pathophysiology of preeclampsia, Fetoplacental blood vessels, namely, the human placental chorionic plate arteries, constrict in response to ET-1.(3)

Moreover, there is now some evidence that amniotic concentrations of ET-1 is elevated in pregnancies associated with preeclampsia. Margarit et al. found a statistically significant increase in ET-1 concentration in the aminionic fluid of women at 17 weeks' gestation who developed preeclampsia as compared to those who did not develop preeclampsia. By the second trimester, higher levels of ET-1 have also been shown in the amniotic fluid of women with preterm premature rupture of the membranes(4).

d. Platelet factors

In mild preeclampsia, serotonin (5-HT) released from aggregating platelets interacts with endothelial 5-HT1 receptors, resulting in the release of prostacyclin and NO (EDRF). The released prostacyclin induces angiotensin II release, improving uteroplacental perfusion. In early-onset severe preeclampsia, damaged uteroplacental vessels cannot respond to 5-HT1 effects. Instead, serotonin interacts with 5-HT2 receptors on vascular smooth muscle cells, inducing vasoconstriction. Platelet-derived serotonin also activates 5-HT2 platelet receptors, establishing a positive feedback loop and intensifying platelet aggregation. The loss of 5-HT1 receptors prevents stimulation of angiotensin II release.(3)

e. Calcium

In normal pregnancy, the intracellular free calcium concentration increases slowly, but this increase is significantly greater in the third trimester in women with preeclampsia. The increase in cytoplasmic calcium levels is enhanced by angiotensin II, and the enhancement is greater in women with preeclampsia compared with normotensive women.

This response to angiotensin II occurs long before signs of preeclampsia become evident and is a sensitive indicator of its subsequent development.(5)