REVIEW OF MANAGEMENT OF ECLAMPSIA

Essay

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By

KHALED SAMIH MOSA BADR

M.B.B.Ch

Under supervision of

Prof. Dr. **HAMDI MOHAMED EL-KABARITY**

Professor of Obstetric and Gynaecology

Faculty of Medicine

Ain Shams University

Dr. **SHERIF MOHAMED SALEH EL-GHETANY**

Assistant Professor of Obstetric and Gynaecology

Faculty of Medicine

Ain Shams University

FACULTY OF MEDICINE

AIN SHAMS UNIVERSITY

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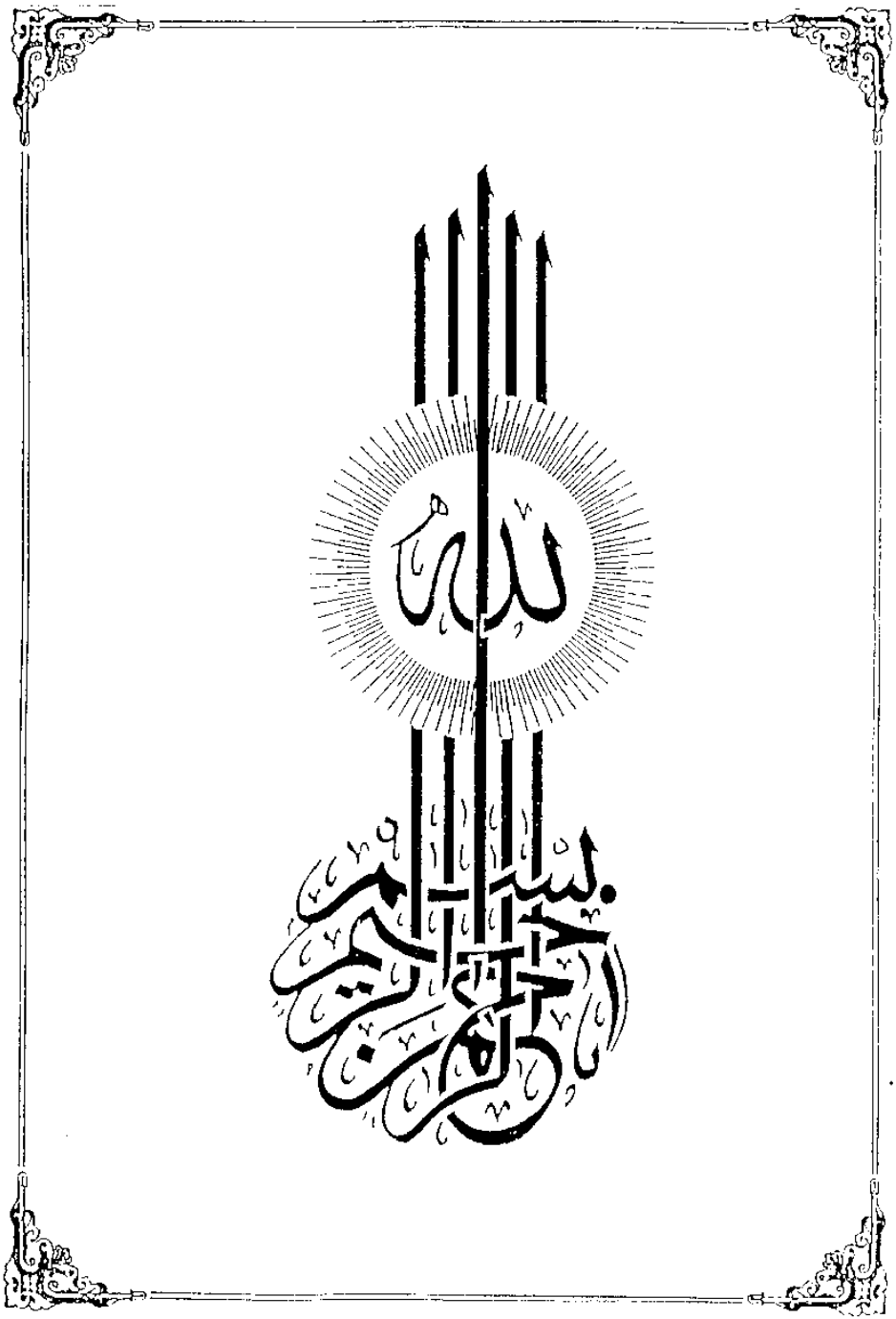

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INTRODUCTION

INTRODUCTION

Eclampsia continues to be important public health problems in childbearing period. Even in recent studies, eclampsia remains one of the leading causes of maternal mortality in many parts of the world (Moodley et al., 1983; Redman, 1988) and was associated with increase risk of abruptio placenta (Sibai et al., 1984), disseminated I.V. coagulation, acute renal failure and cerebral hemorrhage (Cunningham and MacDonald, 1989).

In addition, eclampsia reduces uteroplacental perfusion which places the fetus at high risk of intrauterine growth retardation, preterm birth and perinatal mortality (Cunningham and MacDonald, 1989).

Eclampsia is diagnosed when convulsions, not caused by any cioincidental neurological disease such as epilepsy, develop in a pregnant woman who has clinical criteria for pre-eclampsia (American College of Obs. & Gyn., 1986).

The word eclampsia is derived from a Greek word by Hippocrates to designate a fever of sudden onset. The word means a flash and is indicative of fulminating character of the disease which has come to be generally known as eclampsia.

Although pre-eclampsia and eclampsia have been studied extensively worldwide, their cause remains unknown (Saftalas et al., 1990).

Most likely, eclampsia ultimately results from cerebral vasospasm (Seehan and Lynch, 1973) or cerebral oedema (Chesley, 1978).

The management of eclampsia aims to stop the convulsions and prevent recurrence, control the blood pressure, correct fluid and electrolyte imbalance and deliver the patient promptly.

Anticonvulsant and antihypertensive drug therapy should protect the woman and her fetus from deleterious effects of convulsions, but should not expose either to addtional risks from therapy (Crowther, 1990). There is uncertainty as to the most effective anticonvulsant. Magnesium sulphate is favoured by some (Sibai et al., 1981; Pritchard et al., 1984), diazepam (Thakkar and Wacha, 1978) and phenytoin (Moore and Redman, 1987) by others.

Excellent results have been reported for all these anticonvulsant drugs with respect to reducing maternal and perinatal mortality. When eclampsia develops before or during labour the outlook for both mother and baby is worse the longer delivery as Menon (1961) in Madras and Lopez-Llera

(1967) in Mexico have shown.

Delivery should, therefore, effected promptly by whatever means caries the best prognosis for mother and baby.

Menon (1961); El-Kabarity et al. (1985) showed cecsarean section to be safe when performed at right time and after intensive care management of eclampsia.

However, after introduction of electronic fetal monitoring and efficient induction of labour by excalating doses of oxytocin it would seems reasonable nowadays to attempt vaginal delivery in favourable cases (near term, cervix "ripe") as soon as fits are controlled (Lawson, 1982).

The aim of our essay is to review the protocoles of management of eclampsia and to varify the prons and cons in these different lines of treatment.

HISTORICAL REVIEW

The term eclampsia, according to Delee was introduced by Boisser de Savages in 1760 and derives from the Greek ward Ekauttel used by Hippocrates to denote a sudden fever, which means a shining froth (Theobald, 1955).

Hippocrates mentioned that in pregnancy drowsiness with headache accompanied by heaviness and convulsions, prognosis is generally bad (Chadwick and Mann, 1950).

Mauriceau (1672-1709) was the first to differentiate between epilepsy and eclampsia. Castilli in 1682 translated the ward eclampsia which he defined as "a brightness, shining or lightness".

The correlation between eclamptic convulsion and the development of oedema, proteinuria and hypertension was not clear until the nineteenth century.

In 1843, Simpson pointed out that albuminuria and oedema were almost inevitable forerunners of eclampsia but he thought eclampsia was a manifestation of nephritis.

Browne and Dixon (1978) mentioned that the unsatisfactory term "toxaemia of pregnancy" was based on the idea that it is due to an unknown circulating toxin.

Weinstein (1982) said when hepatic pain is associated with abnormal liver function tests (e.g. elevated liver enzymes), low platelet count (probably due to DIC) and hemolysis, the condition is known as "HELLP syndrome".

As pointed out by Chesley (1971) every one from allergist to zoologist had proposed a theory and suggested rational therapy based upon his theory, including mastectomy, oopherectomy, renal decapsulation, trephination and all sorts of medical regimens.

Before the mid-nineteenth century, the maternal case mortality rate from eclampsia ranged from 22 % to 47 % and the perinatal mortality rate was something over 66% (Chesley, 1978). As Chesley says, during this period the treatment consisted of "purge, puke, plaster and phlebotomy".

During the second half of the nineteenth century heavy sedation with narcotics and anesthetics became the standard mode of therapy. The maternal mortality rate centered around 22 % during this period. Early in the twenteenth century, the Russian Stroganoff introduced the concept of prophylaxis against recurrent seizures and halved the maternal case mortality rate (to 10 %) by using morphine, chloral hydrate and light chloroform anesthesia (Richard, 1984).

PREVALENCE AND TYPES

. The incidence of eclampsia shows a wide seasonal and geographical variations together with nutritional status and economic standard of the patient.

. Eclampsia occurs in as many as 1 to 3 per thousand pregnancies delivered in large obstetric centres. Since the incidence of PIH in such institutions is typically between 5% to 10 % of all pregnancies, eclampsia could be judged to complicate roughly 1 in 20 to 1 in 100 cases of PIH. This kind of computation, however is of little value to the individual practitioner (Richard, 1984).

. The liability of eclampsia is very much greater during the first pregnancy than any other subsequent pregnancies, about 70 % of cases occuring in primigravida (Percival, 1976). The old primigravidae are more likely to have chronic hypertension which predisposes to pre-eclampsia and eclampsia. Very young teen age primigravidae are also at high risk (Duenholter et al., 1975).

. The liability of eclampsia in multiple pregnancy is four to five times greater than singleton pregnancy. About 90 % of cases occur after the twenty fourth week of normal pregnancy and 50 % between thirty sixth and full term. But

eclampsia had been observed as early as sixteen to eighteen weeks with vesicular mole where no foetus was present at all. This occurrence is explained by the fact that eclampsia and pre-eclampsia occur more often with hyperactive placenta than with a small degenerative one. Other example of hyperplacentosis are diabetes mellitus with pregnancy and multiple fetuses (Page, 1939).

In 64 % of cases, the convulsions begin before the onset of labour and the condition termed antepartum eclampsia and in 19 % during the process of delivery and the condition is termed intrapartum eclampsia. In 17 %, the convulsions begin early after delivery and the condition is termed post partum eclampsia.

Post partum eclampsia occurs at late as 48 hours after delivery, but most obstetricians believe that post partum eclampsia can not occur more than 24 hours after delivery. There were few cases in whom fits occured for the first time after 24 hours and in whom careful investigations failed to reveal any other cause. One of them a 17 years old Pakistanian, developed fits on the sixth day of the puerperium, from which she subsequently died, and autopsy revealed the characteristic changes associated with eclampsia (Brown et al., 1987).

ETIOLOGY :

The etiology of eclampsia is unknown but most investigators believe that cerebral vasospasm and cerebral oedema are important factors (Richard, 1984).

Cerebral oedema :

Over 70 years ago Zangemeister (cited by Chesley, 1978) concluded that cerebral oedema was the cause of eclampsia; he opened the skulls of three living eclamptic women and found the dura to be tense and hard. Then he opened the dura, a large amounts of fluids escaped and the convulsions subsided. Indeed the modern technique of computed axial tomographic (CAT) scanning has identified substantial cerebral oedema in some eclamptic women (Beeson and Duda, 1982; Gaitz and Bamford, 1982).

It is an extension of this central process of oedema that is thought to produce the occasional case of transient blindness associated with eclampsia (Beeson and Duda, 1982; Pritchard and MacDonald, 1980).

Cerebral vasospasm :

Other authors such as Sheehan and Lynch 1973) attribute eclampsia principally to cerebral vasomotor disturbances. Vasospasm has long been recognised as a critical component of PIH and eclampsia. Coronary artery spasm in fact, may be an