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FEVERS PRESENTING BY COMA IN EMBABA FEVER HOSPITAL

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THESIS

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Presented by

Dr. MAGDY ABDEL-RAZEK HASAN
M.B., B.CH

د ما رکیت مرکب می مورد

Supervisors

Prof. Dr. MOHAMED FATHY ABDEL-WAHAB
Prof. of Tropical Medicine
Ain Shams University

Prof. Dr. MUBARK MOHAMED HUSSEIN

Ass. Prof. of Tropical Medicine

Ain Shams University

FACULTY OF MEDICINE AIN SHAMS UNIVERSITY

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C O N T E N T S

		Page			
-	INTRODUCTION AND AIM OF THE WORK	ı			
,	REVIEW OF LITERATURE				
	- Coma	2			
	- Meningitis	8			
	- Encephalitis	13			
	- Hepatic coma	16			
	- Diabetic coma	21			
	- Renal failure	26			
	- Leukaemia	30			
	- Heat stroke	33			
-	MATERIAL AND METHODS	36			
-	RESULTS	41			
-	TABLES				
_	DISCUSSIONS 47				
_	SUMMARY	55			
-	- REFERENCES 61				
_	- ARARTC SHMMARY				

INTRODUCTION AND AIM OF THE WORK

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Introduction

Fever presenting by coma is a challenging problem in fever hospitals as it deserves special considerations from the diagnostic and prognostic point of view. Coma may be either a late development of a disease, the nature of which has been suggested by other symptoms or an early prominent feature of the case.

Aim of the work

Study of the clinical picture, diagnosis and management of fevers presenting by come in Embaba Fever hospital, Giza.

- REVIEW OF LITERATURE

COMA

Pathophysiology:

The pathophysiologic basis of coma is either mechanical destruction of crucial areas of the brainstem or cerebral cortex (anatomic coma) or global disruption of brain metabolic processes (metabolic coma). Coma of metabolic origin may be produced by interruption of energy substrate delivery (hypoxia, ischemia, hypoglycemia) or by alteration of the neurophysiologic responses of neuronal membranes (drug pralcohol intoxication, epilepsy or acute head injury) (Ropper and Martin, 1987).

In general, unconsciousness here is usually the result of cerebral oxygen deficiency, disturbance of cerebral metabolism or abnormalities associated with alteration of electrical patterns of cerebral cortex (Adams, 1966).

The brain is markedly dependent on continous blood flow and delivery of oxygen and glucose. Normal resting cerebral blood flow is approximately 55 ml per 100 gm per minute. When it becomes 25 ml per 100g per minute, the EEG becomes diffusely slowed (Typical of metabolic encephalopathies) and at 15 ml per 100g per minute, brain electrical activity ceases.

If all other conditions such as temperature and arterial oxygenation remain normal, cerebral blood blow less than 10 ml per 100 g per minute causes irreversible brain damage (Ropper and Martin, 1987).

Consciousness:

Arousal and awareness (Caronna and Simon, 1979). Arousal (Simply being awake) is a primitive state managed by reticular activating system extending from the medulla to thalamus. Awareness is requiring an intact cerebral cortex to interpret the sensory input and respond accordingly (Scherer, 1986).

The comotosed patient is neither awake nor aware (Plum and Posner, 1980).

The vegetative state in this case, the patient is awake but not aware. The brain stem functions without intellect (Caronna and Simon, 1979). The locked in syndrome is intact mind locked into an immobilized body (Scherer, 1986).

Coma Grading :

Crade I: Patient drowsy but responds to verbal

commands.

Crade II: Patient unconscious but responds to minimal

stimulus.

Crade III: Patient unconscious but responds to maximal

painful stimulus.

Crade IV: Patient unconscious and does not respond to

any stimulus.

(Krapez and Cole, 1977).

Level of Consciousness:

The Glasgow coma scale is useful in predicting the outcome, the chance of good recovery or moderate disability improved with increasing score (Rwiza and McLarty, 1987).

	Sco	re Times h.
Eye opening	4	Spontaneous .
•	3	To noise
-	2	To pain
-	1	None
Best verbal response	5	Oriented
-	4	Confused
-	3	Inappropriate words
• ·	2	Incomprehensive sounds
-	1	None
Best motor response	6	Obeys
.	5	Localises pain
, -	4	Withdraws
-	3	Flexion to pain
-	2	Extension to pain
-	ı	None

(Rwiza and McLarty, 1987).

The Glasgow coma scale has been mainly used for predicting outcome in patients with head injury (Jennett et al., 1977). This scale is found to be equally useful in patients in nontraumatic coma (Rwiza and McLarty, 1987).

If the usefulness of this scale is established, it will enable doctors working in the tropics in crowded wards to predict prognosis (Rwiza and McLarty, 1987).

Along with the scale, important bedside observations such as blood pressure, pulse and pupillary size can be incorporated (Teasdale et al., 1975).

The scale can therefore serve a dual function of assessing the level of consciousness as well as helping in estimation of the prognosis (Rwiza and McLarty, 1987).

Management:

Coma is a serious medical emergency requiring undivided attention and the application of every possible therapeutic measure until death or recovery (Plum and Levy, 1978). An intensive care unit with basic lifesupporting facilities, such as a reliable supply of oxygen, intubation sets with occasional ventilation for limited periods and an electrocardiographic monitoring machine, would be adequate.

Most important however is meticulus observation of basic nursing routines for the unconscious patients and awareness of those causes of coma such as meningitis, cerebral malaria and diabetic coma, for which specific and highly effective remedies are available (Rwiza and McLarty, 1987).

Fevers Presenting by Coma

According to Mackenzie (1973), fever with coma may be classified into two main groups:

- A) Cases in which coma is a late development of a disease, the nature of which has been suggested by other symptoms:
 - 1. Severe infection including typhoid fever, typhusfever, measles, scarlet fever, all forms of
 pneumonia, malaria, black water fever, septicaemia
 pyaemia, yellow fever and Weil's disease.
 - 2. Infections of nervous system including all forms of meningitis, encephalitis and cerebral abscess.
 - 3. Cerebral tumors, primary or secondary.
 - 4. Endogenous intoxications and metabolic diseases, cholaemia, thyroid storm or diabetes.

- B) Coma occuring as the presenting sign in a previously healthy subject, or in one about whom no medical data are known:
 - 1. Cerebrovascular accidents.
 - 2. Head injuries.
 - 3. The acute effects of drugs and poisons.
 - 4. Cerebral infections as fulminating meningococcal meningitis, cerebral malaria or rupture of cerebral abscess into the ventricles.
 - 5. Haemorrhage into cerebral tumour.
 - 6. Post epileptic coma.
 - 7. Heat stroke.