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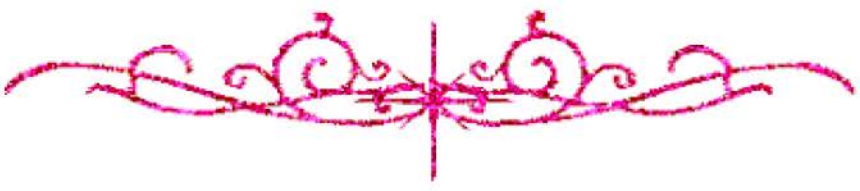

بالرسالة صفحات لم ترد بالأصل

CHANGES IN AUDITORY P300 EVENT RELATED POTENTIALS IN UREMIC PATIENTS UNDERGOING HEMODIALYSIS

Thesis submitted to the Faculty of Medicine,
Alexandria University,
In partial fulfillment of the requirements for
The master degree of
INTERNAL MEDICINE

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ACKNOWLEDGEMENT

I would like to express my deepest gratitude to Prof. Dr.**ESAM ELDEEN HASAN ELKASHEF**, Professor of Internal Medicine, and Chairman of Nephrology Unit, Faculty Of Medicine, University of Alexandria, for his great support.

I would like to present special appreciation to Prof. Dr. **MONA IBRAHIM MORAD**, Professor of Otolaryngology, (Audiology Unit), Faculty of Medicine, University of Alexandria for scientific supervision and continuous guidance.

Thanks to Dr. **AHMED GABER ADAM**, Lecturer of Internal Medicine, Faculty of Medicine, University of Alexandria for his great help.

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INTRODUCTION

INTRODUCTION

Nervous system dysfunction is a major complication of end stage renal disease. In uremia, there is an accumulation of toxic end products of metabolism, primarily of proteins and their derivatives, secondary to decreased excretion. The actual pathophysiological and biochemical mechanisms of the uremic syndrome still remain obscure.

The relationship between the degree of elevation of urea and creatinine and the extent of neurologic impairment is highly variable. The rapidity of development of uremia is a factor: the more sudden the rise, the lower the levels of azotemia that are associated with symptoms. Once azotemia develops, the symptoms if uremia do correlate in a general way with the degree of biochemical disturbance as hyper – or hypokalemia hyper – or hyponatremia, hypocalcemia, hyperphosphatemia, hyperparathyroidism, acidosis, dehydration, and the effects of hypertension. None of these abnormalities is the primary cause of uraemic encephalopathy.

The clinical picture known as uremic encephalopathy, is fundamentally characterized by cognitive, neuromuscular and behavioural disturbances. [1]

The symptomatology is initially characterized by cognitive and behavioural disturbances; the patient complains of reduced attention and concentration, mnestic deficiency, and ideomotor slowing; only later on do neurological signs such as trmors, myoclonias and possible convulsions appear. [1]

Nowadays, it is possible to examine the cognitive functions using the Event – Related Potential P300. The P300 is a neurophysiological method which gives an indication of the attentive functions and short term memory, since it gives an objective measurement of the cerebral activity during cognitive tasks. [2]

Evoked potentials represent stable cortical sequences of averaged negative and positive EEG peaks within a period of several hundred milliseconds, elicited by appropriate sensory stimuli (stimulus – related) or complex tasks (event – related P300). Figures 1,2.

P3 wave (P300) is the third positive wave in the event – related potential (ERP). It appears around 300 msec after stimulus onset (Thus, it is also referred to as P300). Event – related auditory P300

evoked potentials are generated following a binaurally presented tone discrimination with frequent (80%) tones of 1000 Hz and rare (20%) target tones of 2000 Hz. The subjects are instructed first to hear the tones and daydream (passive task), then to count the less frequent tones and press on the button of a hand counter (active discrimination task). Uremic patients received passive and active tasks before and after dialysis treatment.

P300 latency is abnormally delayed in conditions involving generalized cognitive impairment such as dementia, schizophrenia, hepatic encephalopathy and chronic renal failure. [2]

CHRONIC RENAL FAILURE

Definition: Chronic renal failure is irreversible deterioration in renal function. The ensuing impairment of the excretory and endocrine functions of the kidney leads to the development of the clinical syndrome of uremia. [3]

Etiology of chronic renal failure: [3]

(I) Congenital and inherited diseases:

1. Polycystic kidney disease. (Infantile or adult)
2. Alport's syndrome.
3. Congenital hypoplasia.

(II)Vascular diseases:

1. Arteriosclerosis.
2. Vasculitis (Scleroderma, polyarteritis nodosa, systemic lupus erythematosus).

(III) Glomerular diseases:

1. Proliferative glomerulonephritis (proliferative GN).
2. Crescentic GN.
3. Membranous GN.
4. Mesangio capillary GN.
5. Glomerulosclerosis.
6. Secondary GN (amyloidosis, polyartertis nodosa, sysemic lupus erythematosus, diabetic glomerulosclerosis).

(1V) Interstitial diseases:

1. Chronic infective interstitial nephritis (chronic pyelonephritis).
2. Vesico-ureteric reflux.
3. Tuberculosis.
4. Analgesic nephropathy.
5. Nephrocalcinosis.
6. Schistosomiasis.
7. Unknown origin.

(V) Obstructive uropathy:

1. Causes in the lumen:

   Stone, blood clot, caseous or necrotic debris, and sloughed papilla.

2. Causes in the wall:

   a. Tumour.

   b.Fibrosis: following trauma, stone, instrumentation, surgery or infection (tuberculosis, schistosomiasis, gonorrhea).

   c. Congenital bladder neck obstruction.

   d. Urethral valves.

3. External causes: -

   a. Aberrant renal artery crossing upper ureter.

   b. Retroperitoneal tumour.

   c.Retroperitoneal fibrosis (idiopathic, leaking aortic aneurysm, drug induced, neoplastic).

   d. Secondary

   d. Prostatic enlargement.

   e. Phimosis.

   f. Accidental ligation of ureter at operation.

4. Functional:

   Congenital neuromuscular defects (pelvi-ureteric junction, bladder neck). [3]

<u>Pathophysiology and clinical picture of chronic renal failure (CRF)</u>

(I) Water, Electrolyte, and Acid base metabolism in uremia: [4]

(1) Potassium:

In advanced CRF, the serum potassium concentration tends to be higher than normal, even though body stores of potassium may be reduced. Hyperkalemia can be accentuated by oliguria, trauma, surgery, anaesthesia, blood transfusion, or increased dietary intake.

It can produce serious cardiac abnormalities, but many patients are asymptomatic until cardiac arrest occurs. Occasional patients

complain of muscle weakness or parasthesias. The major warning signs are those detected by electrocardiography and include peaked T waves and prolongation of the P-R interval and QRS complex. [4]

(2) Sodium:

The kidney has a remarkable ability to maintain total body sodium within normal limits until the end stages of CRF.

A) Sodium wasting:

Some patients with CRF have salt-losing nephropathy and may lose sodium chloride to the point of extracellular volume contraction and hypotension. Renal diseases associated with salt wasting include pyelonephritis, medullary cystic disease, hydronephrosis and interstitial nephritis. [4]

B) Sodium retention:

Many patients with CRF are unable to rapidly increase sodium chloride excretion to appropriate levels with increases in dietary intake. These patients have the physical findings of expanded extracellular fluid volume: hypertension, peripheral edema, pulmonary vascular congestion and cardiomegaly. [4] Volume overload worsens hypertension and thus accelerates all forms of CRF. [5]

(3) Acid - Base Balance: [4]

The kidney normally regulates blood pH within narrow limits by reabsorption (proximal tubule) and regeneration (distal tubule) of bicarbonate by secretion of protons into the urinary fluid. The total quantity of acid that can be excreted is a function of the amount of buffer that is excreted and the net rate of proton secretion .The excreted buffers may be filtered (phosphate) or generated (ammonia). In chronic renal disease, there is progressive reduction in net ammonia secretion.

Metabolic acidosis develops when exogenous intake and endogenous production of acid exceed renal net acid excretion. In chronic metabolic acidosis, extrarenal buffering mechanisms become involved, including bone salts and intracellular buffers. Loss of bone buffer stores contributes to the development of osteomalacia and renal osteodystrophy. [6]

(4) Calcium: [4]

The total serum calcium concentration in CRF is significantly lower than normal, although usually above 7.5 mg per deciliter.