NEUROPHYSIOLOGICAL STUDY OF CRITICAL ILLNESS POLYNEUROPATHY AND MYOPATHY IN CHILDREN ADMITTED TO PEDIATRIC INTENSIVE CARE UNIT

By

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ABSTRACT

Background: *Critical illness polyneuropathy (CIP) and myopathy (CIM) are frequent complication of critical illness in adults and are associated with high morbidity and mortality. Little is known about CIP and CIM among critically ill children regarding incidence, risk factors and outcome.*

Objective: *To detect critical illness polyneuropathy and myopathy in children admitted to pediatric intensive care unit (P.I.C.U) in relation to clinical, laboratory findings and neurophysiological studies.*

Patients and Methods: *This cross sectional observational study was carried out at P.I.C.U of Bab-El shereya university hospital on 100 patients aged from 2months to 15 years during the period from July 2019 to December 2020, they were selected by simple random method. All patients were submitted to medical history taking, complete clinical examination, laboratory investigations and neurophysiological studies including nerve conduction studies (NCS), motor and sensory, and electromyography (EMG).NCS and EMG were done at the seventh day of admission and on discharge. Motor nerve studies included bilateral median and peroneal nerves. Sensory nerves included right median and sural nerves. EMG included right tibialis anterior and gluteus muscles. According to results of NCS and EMG patients were divided into 2 groups; I with abnormal neurophysiological studies and group II with normal neurophysiological studies. The clinical and laboratory profiles of the studied patients had been recorded.*

Results: *29patients (29%) developed CIP/CIM: axonal polyneuropathy (24%) demyelinating polyneuropathy (1%) and myopathy (4%). While remaining patients (71%) showed normal neurorophysiological studies. CIP/CIM was significantly associated with thrombocytopenia, elevated liver enzymes, prolonged prothrombin time, Acidosis, hypocalcemia, hypoalbuminemia, hyperglycemia compared to patients*

with normal studies. Mortality rate and duration of PICU stay were significantly higher among patients with CIP/CIM.

Conclusion: *critically ill children frequently develop CIP/CIM, mostly of axonal polyneuropathy, which prolong duration at PICU specially children on mechanical ventilation with subsequently more invasive procedures and resource utilization.*

Keywords: *Critical illness polyneuropathy and myopathy, nerve conduction studies, electromyography.*

INTRODUCTION

Critical illness polyneuropathy and myopathy (CIP/CIM) is a frequent complication of critical illness, acutely and primarily affecting the motor and sensory axons. This disorder can cause severe limb weakness and prolonged weaning from mechanical ventilation **(Williams et al., 2007)**.

In intensive care unit (ICU) 70% of patients with sepsis or systemic inflammatory response syndrome (SIRS), develop CIP. When further complicated by multiple organ failure (MOF), the incidence increases to up to 100% **(Friedrich O et al., 2015)**.

CIP/CIM are largely unexplored in the critically ill pediatric population. As a result, children who are at risk for acquiring neuromuscular dysfunction due to critical illness are less likely to be identified, evaluated and receive appropriate therapies. Also, little is known about important risk factors and how critical illness polyneuropathy and myopathy impact clinical outcomes **(Field-Ridley et al., 2016)**.

Since delirium and sedation are frequent in PICU patients, reliable bedside examination of neuromuscular function can be difficult. So early studies of critical ill children have relied on electrophysiologic testing to provide a rigorous description of underlying neuromuscular dysfunction **(Bolton C, 2006)**.

AIM OF THE WORK

This study aimed to detect critical illness polyneuropathy and myopathy in relation to clinical examination, laboratory investigations and neurophysiological studies in critically ill children admitted in pediatric intensive care unit of of Bab-El shereya university hospital.

Ethical Consideration:

1. A written informed consent was obtained from parents or the legal guardians before the study.

2. An approval by the local ethical committee was obtained before the study.
3. The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.
4. All the data of the patients and results of the study are confidential & the patients have the right to keep it.

Financial Disclosure / Funding:

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PATIENTS AND METHODS

This cross sectional observational study included 100 critically ill children admitted to Pediatric Intensive Care Unit of Bab-Elshereya University Hospital during the period from July 2019 to December 2020, they were selected by simple rondom method.

Inclusion criteria:

1. Age: 2months -15 years.
2. Patients admitted to pediatric intensive care unit more than 7days.
3. Critically ill patients admitted due to different causes such as sepsis, respiratory diseases, cardiac diseases or multiorgan failure.

Exclusion criteria:

1. Patients have previous myopathy or polyneuropathy due to any cause e.g. diabetes mellitus, thyroid dysfunction.
2. Patients admitted to pediatric intensive care unit less than 7days.

Methodology:

All patients were subjected to detailed history, clinical examination and in-depth neurological examination. Also 3 severity scores were counted, namely the Pediatric Risk of Mortality (PRISM), sequential organ failure assessment (SOFA) score and respiratory score to assess illness severity.

Investigations:

A. Laboratory:

- Complete blood count by Sysmex x5-800 (Sysmex Corporation, Japan).
- C-reactive protein (quantitative assessment) by latex agglutination test (TURBOX plus Orion Diagnostica, Finland).
- Blood urea, serum creatinine, ALT and AST by BIOBASE, Automatic Chemistry Analyzer

(BIOBASE Corporation, China).

- PT, PTT and INR by BIOBASE Auto Coagulation Analyzer BK1000B (BIOBASE Corporation, China).
- Arterial blood gases, Na and K by Cobas b 221 system, USA.

B. Neurophysiological studies: NEUROMYAN machine used in both NCS and EMG. Motor nerves included bilateral median and peroneal nerves and sensory nerves included right median and peroneal nerves. EMG included both right tibialis and gluteus maximus muscles. NCS and EMG done at 7th day of admission and on discharge. According to the results patients were divided into two groups:I with abnormal neurophysiological studies and II with normal neurophysiological studies.

Axonal polyneuropathy was diagnosed when the nerve conduction study in 2 or more nerves showed that (1) CMAP amplitudes are <80% of lower limit of normal (2) the sensory nerve action potential amplitudes are <80% of lower limit of normal (3) the nerve conduction velocities are normal or near normal, without conduction block. Also, EMG showed (a) denervation potential (fibrillation, positive sharp waves) in distal muscle and (b) reduced recruitment of motor potential in distal muscle. Acute demyelination polyneuropathy was diagnosed when the nerve conduction study showed (i) prolonged distal motor latency >115% of the upper limit of normal, (ii) decreased conduction velocity<90% of lower limit of normal (iii) conduction block **(Stevens RD et al., 2009)**.

CIM is diagnosed when needle EMG shows short duration, low amplitude motor potential with early or normal full recruitment in proximal muscles (gluteus maximus). **(Latronico N, Bolton CF, 2011)**.

Statistical analysis: Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 23. The quantitative data were presented as mean, standard deviations and ranges when parametric and median, inter-quartile range (IQR) when data found non-parametric. Also qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test and/or Fisher exact test when the expected count in any cell found less than 5. The comparison between groups regarding quantitative data and

non-parametric distribution was done by using Mann-Whitney test.

The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant as the following:

P-value > 0.05: Non significant (NS).

P-value < 0.05: Significant (S).

RESULTS

Our results will be demonstrated in the following tables and figures.

Table (1): demographic and clinical characteristics of studied patients

		Group I (N=29)	Group II (N=71)	T test	P-value
Age (months)		48.83±24.48	51.96±25.97	0.556	0.580
Sex	**Male n (%)**	13(44.8)	41(57.7)	1.383	0.240
	Female n (%)	16(55.2)	30(42.3)		
Duration at PICU (days)		26.17 ± 5.85	15.79 ± 4.64	9.401	0.001*
Mechanical ventilation n (%)		23 (79.3%)	37 (52.1%)	6.349	0.012*
Drugs n (%)	Sedation	19 (65.5%)	11 (15.5%)	24.538	0.001*
	Analgesia	5 (17.2%)	8 (11.3%)	0.648	0.420
	N.M blockers	12 (41.4%)	7 (9.9%)	13.289	0.001*
	Steroids	17 (58.6%)	21 (29.6%)	7.369	0.007*
Severity scores	Respiratory score	6.55 ±1.74	5.63±1.76	2.374	0.020*
	PRISM score	26.76±5.77	17.31±5.61	7.579	0.001*
	SOFA score	13.17±3.09	8.03±2.15	9.497	0.001*
Mortality n (%)		7 (24.1%)	6 (8.5%)	4.483	0.034*

Data are expressed as number and percentage except severity scores and duration at PICU which expressed as mean ± SD. This table demonstrates that there is high statistically significance difference between groups regarding duration at PICU, PRISM score, SOFA score, using some drugs (N.M blockers, steroids and sedatives). Also there is statistically significance mortality in patients with CIP/CIM and incidence of CIP/CIM among patients on mechanical ventilation.

Table (2): Incidence of abnormal neurophysiological studies in relation to causes of admission among the studied patients

Cause of admission	Group l (n=29)	Group II (n=71)	T test	P value
Sepsis n (%)	18 (62.1)	26 (36.6)	5.412	0.020*
Respiratory n (%)	9 (31)	27 (38)	0.437	0.509
Others n (%) e.g, cardiac diseases, trauma	2 (6.9%)	18 (25.4)	4.383	0.063

Data are expressed as number and percentage. This table demonstrates that there is statistically significance incidence of abnormal neurophysiological studies among patients with sepsis.

Table (3): laboratory findings of the studied patients

	Group I			Group II			t. test	p. value
WBC (cells/mcl)	16.64	±	6.05	15.14	±	4.76	1.324	0.188
HB (gm/dl)	10.07	±	2.20	10.54	±	1.99	1.030	0.305
PLT (cells/dl)	118.24	±	47.52	180.73	±	57.84	5.148	0.001*
ALT(IU/l)	118.66	±	48.24	53.86	±	19.82	9.562	**0.001***
AST(IU/l)	110.07	±	55.00	49.58	±	26.32	7.446	**0.001***
PT (scondes)	19.55	±	3.83	14.98	±	3.52	5.732	**0.001***
INR	1.69	±	0.42	1.20	±	0.40	5.417	**0.001***
Albumin(g/dl)	3.00	±	0.78	4.38	±	0.60	9.568	**0.001***
Urea(mg/dl)	23.14	±	7.21	22.65	±	6.64	0.327	0.745
Creatinine (mg/dl)	0.83	±	0.35	0.72	±	0.35	1.382	0.170
CK (U/l)	105.07	±	34.02	196.61	±	33.95	1.130	0.261
RBS (mg/dl)	192.21	±	32.22	127.90	±	38.48	7.929	**0.001***
PTT (second)	39.59	±	9.05	38.51	±	8.45	0.568	0.572
Na (Meq/l)	126.55	±	5.68	132.96	±	10.22	3.175	**0.002***
K(Meq/l)	3.98	±	0.85	3.94	±	0.73	0.225	0.823
Ca (mg/dl)	7.98	±	0.99	9.55	±	0.93	7.501	**0.001***
CRP	50.48	±	25.46	26.96	±	16.02	5.560	**0.001***
PH	7.24	±	0.06	7.33	±	0.07	5.834	**0.001***
P co2 (mmHg)	43.93	±	12.36	43.45	±	10.31	0.199	0.842
Hco3 (mmHg)	19.72	±	3.43	20.92	±	3.17	1.665	0.099

Data expressed as mean ± SD.

WBC; white blood cells, HB;haemoglobin, PLT; platelets, ALT; alanine transaminase. AST; aspartate transeaminase, PT; prothrombin time. INR; international normalization ratio, CK; creatine kinase, RBS; rondome blood suger, Na; sodium.K;potassium.Ca; calcium, CRP; c reactive protine.

This table shows high statistically significance difference between the studied groups regarding platelets count, liver enzymes, PT, INR, albumin, calcium, CRP and PH.

Table (4): Motor nerves conduction study's findings among patient

		Group I			Group II			t. test	p. value
left peroneal nerve	DL (seconds)	4.79	±	0.93	4.55	±	1.11	0.990	0.325
	Amplitude (mv)	2.41	±	1.10	12.55	±	2.61	20.159	**0.001***
	CV(m/s)	48.98	±	5.51	52.34	±	6.11	2.564	**0.012***
	F wave latency (sec)	37.25	±	9.91	38.10	±	11.42	0.345	0.731
left median nerve	DL (seconds)	3.00	±	0.57	3.05	±	0.70	0.304	0.762
	Amplitude (mv)	2.93	±	0.61	10.94	±	2.47	17.224	**0.001***
	CV(m/s)	50.08	±	9.77	53.71	±	5.18	2.418	**0.017***
	F wave latency (sec)	18.17	±	3.92	17.27	±	4.07	1.019	0.311
right peroneal nerve	DL (seconds)	4.21	±	1.18	4.33	±	1.12	0.476	0.635
	Amplitude (mv)	2.17	±	0.73	12.20	±	1.94	27.051	**0.001***
	CV (m/s)	51.82	±	9.48	55.17	±	5.36	2.240	**0.027***
	F wave latency (sec)	33.32	±	9.46	34.63	±	9.97	0.598	0.551
right median nerve	DL (seconds)	3.32	±	0.59	3.23	±	0.66	0.694	0.490
	Amplitude (mv)	3.09	±	0.52	10.90	±	1.88	21.958	**0.001***
	CV (m/s)	50.98	±	9.34	54.72	±	5.83	2.415	**0.018***
	F wave latency (sec)	17.17	±	2.90	16.37	±	4.09	0.965	0.337

Data expressed as mean ± SD, DL: distal latency. CV: conduction velocity

This table demonstrates that there is high statistically significant difference between studied geoups regarding amplitude and statistically significant difference regarding conduction velocity in all studied nerves.

Table (5): sensory nerves conduction study's findings among studied patients

		Group I			Group II			t. test	p. value
Rt sural	**PL (seconds)**	3.45	±	0.66	3.74	±	4.84	0.318	0.751
	CV (m/s)	44.59	±	4.95	47.56	±	6.12	2.321	**0.022***
	Amplitude (uv)	3.33	±	1.04	7.86	±	0.99	20.460	**0.001***
Rt median	**PL (seconds)**	3.28	±	0.53	3.46	±	0.61	1.452	0.150
	CV(m/s)	49.09	±	7.01	52.15	±	5.59	2.301	**0.024***
	Amplitude (uv)	8.38	±	1.16	23.39	±	4.27	18.618	**0.001***

PL:peak latency. CV:conduction velocity. Data are expressed as mean±SD.

This table demonstrates that there is high statistically significant difference between groups regarding amplitude and statistically significant difference regarding conduction velocity.

Table (6): EMG of right tibialis anterior muscle among studied patients

		Group I	Group II	X^2	P-value
Denervation	No	4(13.8%)	71(100%)	**10.201**	**0.001***
	Yes	25(86.2%)	0(0%)		
Motor unit	Normal	29(100%)	71(100%)	-	-
	Polyphasic	0(0%)	0(0%)		
Interference	Normal	4(13.8%)	71(100%)	**81.609**	**0.001***
	Decreased	25(86.2%)	0(0%)		
	Early recruitment	0(0%)	0(0%)		

Data expressed as number (percentage).

This table demonstrates that there is high statistically significant difference between groups regarding denervation and decreased interference.

Table (7): EMG of right gluteus Maximus muscle among studied patients

		Group I	Group II	X^2	P-value
Denervation	No	29(100%)	71(100%)	-	-
	Yes	0(0%)	0(0%)		
Motor unit	Normal	25(86.2%)	71(100%)	**10.201**	**0.001***
	Polyphasic	4(13.8%)	0(0%)		
Interference	Normal	25(86.2%)	71(100%)	**10.201**	**0.001***
	Decreased	0(0%)	0(0%)		
	Early recruitment	4(13.8%)	0(0%)		

Data expressed as number (percentage).

This table demonstrates that 4 patients developed myopathy in the form of small polyphasic motor unit with early recruitment.

Table (8): Pattern of neurophysiological studies among the studied patients

	N	%
Normal	71	71
Axonal polyneuropathy	24	24
Demylinating polyneuropathy	1	1
Myopathy	4	4
Total	100	100

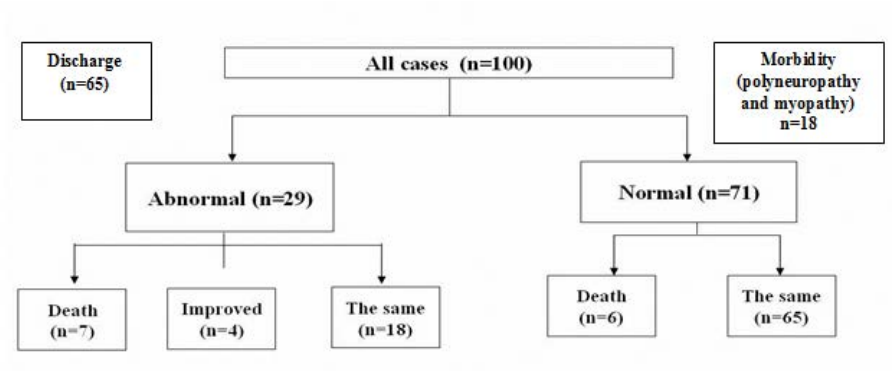

Figure (1): Outcome of our studied patients

DISCUSSION

Critical illness polyneuropathy (CIP) and myopathy (CIM) are complications of critical illness that present with muscle weakness and failure to wean from the mechanical ventilator. In addition to prolonging mechanical ventilation and hospitalization, CIP and CIM increase hospital mortality in patients who are critically ill and cause chronic disability in survivors of critical illness **(Nicola L et al., 2011)**.

In the present study 29 patients (29%) developed CIP/CIM mostly of the axonal polyneuropathy pattern (24%). The mechanism of the reduced motor and sensory nerve amplitudes in CIP is reported to be due to dysfunction of voltage-gated sodium channels, as abnormal membrane depolarization found in different tissues of critically ill patients may cause reduced membrane excitability **(Koch S et al., 2016)**. On the other hand, the pathophysiology of CIM is a complex process in critically ill patients that may be attributed to a constellation of microvascular changes, electrical phenomena, inflammatory and cellular metabolic changes as well as an imbalance between protein synthesis and breakdown **(Hermans G, Van den Berghe G, 2015)**.

Our study showed that the incidence CIP/CIM was strongly related to the cause of admission as following; 62.1% with sepsis, 31% with respiratory diseases and 6.9% with other causes, sepsis being the commonest risk factor. And this agree with **(Field-Ridley et al., 2016)** who founded that Patients with respiratory and infectious disease had a higher likelihood of developing CIP/CIM relative to patients admitted with other primary diagnostic categories. And our study agrees with **(Jasvinder Chawla et al., 2010)** who stated that: CIP and CIM are now the most common acquired neuromuscular condition in the ICU setting with a risk of development that may reach 50% in patients with sepsis.

Laboratory investigations in our study showed that CIP/CIM patients had number of significant abnormalities, including decreased platelet count, elevated liver enzymes, prolonged PT, acidosis, lower serum calcium and albumin levels as well as higher blood glucose level.

Thrombocytopaenia can be explained by several mechanisms encountered in ICU settings, such as haemodilution and increased platelet consumption which is very

common after tissue trauma, bleeding and disseminated intravascular coagulopathy. Increased platelet destruction related to immune dysfunction and reduced platelet production may also be confounding factor **(Greinacher A, Selleng K, 2010)**.

Hypocalcaemia was more pronounced among CIP/ CIM patients, increasing the burden of neuromuscular dysfunction in these children and reflecting the vital role of calcium ion for normal neuronal properties and muscular performance. **(Kress JP and Hall JB, 2014)** reported that there is an increase in diaphragmatic contractility immediately after repletion of calcium and suggesting that its deficiency impairs diaphragmatic contractile properties.

Hypoalbuminaemia was strongly associated with abnormal neurophysiological as low values of albumin may be related to inflammation and loss of muscle mass that may be attributable to activation and release of specific proteases **(Mitch WE, 2006)**.

High ALT and AST occurred along with prolonged PT in patients with CIP/CIM. This could be augmented by that observed in septic shock with hypoxic hepatitis where the altered enzymatic changes of hypoxic hepatitis combine with sharp increases in ALT, AST blood levels that occur 24 h after the start of septic shock with a dramatic drop in prothrombin level **(Nesseler N et al., 2012)**.

Regarding Serum creatine kinase (CK), there was no statisticaly significant difference between the two studied groups and therefore CK is not particularly helpful in the diagnosis of CIP/CIM and this in agreement to **(Hermans G et al., 2008)** who found that CK levels may be normal in people with CIM but do not have muscle necrosis or have scattered muscle necrosis. Furthermore, in those with CIM and muscle necrosis, the CK elevation is typically transient and may be missed on laboratory analysis.

In our study we founded that using of some drugs specially if used for more than 3 days such as sedative, N. M blockers and specifically steroids had a great association with increased incidence of critical illness acquired weakness and this agree with **(Charles et al., 2005)** who concluded that neuromuscular blocking agents, such as pancuronium bromide or the shorter acting vecuronium, and steroids, singly or in combination induced either a pure axonal motor

neuropathy or a primary myopathy.

Our study revealed that mortality rate was higher among patients with CIP/CIM versus those with normal neurophysiological studies: 7 patients (24.1%) and 6 patients (8.5%) respectively. This reflects higher PRISM score in patients with CIP/CIM comparing to patients with normal studies (26.76±5.77) and (17.31±5.61) respectively. This agree with **(Latronico N et al., 2014)**: who founded that mortality was higher among patients with abnormal neurophysiological studies than those with normal neurophysiological studies died, (21.3%) (7.4%) respectively.

Among patients CIP/CIM only 4 cases (13.7%) improved on follow up at discharge as their affection was mild and physiotherapy started early with correction of nutritional status and electrolytes disturbances while **(Latronico N et al., 2014)** found that 10 patients out of 28 patients (35%) who developed CIPNM improved at discharge.

The remaining 18patients (64%) showed persistence of abnormal neurophysiological studies at discharge from PICU so they referred to a chronic care or rehabilitation facility for physiotherapy. further studied will be needed for long term follow up of patients who developed CIP/CIM after their discharge.

CONCLUSION

CIP/CIM were detected in 29% of our critically ill patients, 25 patients with neuropathy and 4 with myopathy. Many risk factors are involved in development of CIP/CIM such as sepsis being the major risk factor, mechanical ventilation, hypoalbuminaemia, hyperglycemia and acidosis and hypocalcemia. Critical illness polyneuropathy and myopathy was associated with long PICU stay, delays weaning from MV, as well as increased ICU mortality rate.

RECOMMENDATIONS

1. Nerve conduction studies (NCS) and Electromyography (EMG) should be done to patients with prolonged ICU stay or on mechanical ventilation with difficult weaning from it or patients with repeated intubation.
2. Early management of severe sepsis and septic shock being the commonest risk factor for incidence of critical illness polyneuropathy and myopathy.
3. Correction of hypoalbuminemia and electrolytes disturbances if

present with optimizing nutritional status of the patients. Strict blood glucose control and avoidance of hyperglycemia in the ICU.

4. Minimizing the use of drugs which carried risk for development of CIPNM such as steroids, sedatives and N.M. blockers whenever possible.
5. Future studies will be needed for following up of the patients with critical illness polyneuropathy and myopathy after discharge from PICU.

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دراسه فسيولوجيه عصبيه للاعتلال العصبي والعضلى للأمراض الحرجه فى الأطفال المحجوزين بوحدة الرعايه المركزه للأطفال

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الهدف من البحث: تهدف هذه الدراسه الى اكتشاف الأعتلال العصبى والعضلى فى الحالات الحرجه للاطفال المحجوزين بوحده الرعايه المركزه من خلال الفحص الأكلينيكى وفحوصات الدم والفحوصات الفسيولوجيه العصبيه والتى تشمل رسم الأعصاب ورسم العضلات.

الوسائل والادوات: هذه دراسة مقطعية استكشافيه تم إجراؤها خلال الفترة من يوليو 2019 إلى ديسمبر 2020 و شملت 100 طفل محجوزين بوحدة الرعايه المركزه للاطفال بمستشفى سيد جلال الجامعى والذين تتراوح اعمارهم من شهرين الى 15 عاما حيث تم إخضاعهم جميعا لأخذ التاريخ الطبي كاملا، والفحص السريري الشامل، و عمل الفحوصات المخبرية اللازمه، بالاضافه الى عمل رسم عصبى وعضلى.

نتائج البحث: كان عدد الذكور54 (54٪) بينما كان عدد الإناث46 (46٪). سجلت الحالات ذات الاختبارات الفسيولوجيه العصبيه الغير طبيعيه نسبه 29% من الحالات.

حدوث اعتلال المحور العصبى فى أربع وعشرين حاله بنسبه 24% واعتلال خطى فى حاله واحده بنسبه 1% واعتلال عضلى فى اربع حالات. وأثبت الدراسه ان التسمم الدموى مثل اعلى نسبه حدوث فى الاعتلال العصبى والعضلى بنسبه 62.1% يليه امراض الجهاز التنفسي بنسبه 31% ثم الامراض الاخرى بنسبه 6.9% من الحالات ومثل وضع الحالات على اجهزه التنفس الصناعى عامل خطوره كبيره فى حدوث الاعتلال العصبى والعضلى بنسبه 79.3% من اجمالى الحالات .هناك علاقه ذات دلائل احصائيه بين طول فتره البقاء فى المستشفى، ارتفاع السكر ارتفاع حامضيه الدم، نقص فى مستوى الالبومين، ارتفاع وظائف الكبد واخيرا نسبه الوفيات وحدوث الاعتلال العصبى والعضلى فى الاطفال المصابين بالامراض الحرجه والمحجوزين بوحدة الرعايه المركزه.

الاستنتاج: الدراسات الفسيولوجيه العصبيه تعتبر من الدراسات السريريه الهامه لمرضى الحالات الحرجه فى العنايه المركزه للتنبؤ بحدوث الاعتلال العصبى والعضلى. التدخل المبكر لعلاج تسمم الدم وكذلك تصحيح عوامل الخطوره التى تزيد نسبه حدوث الاعتلال العصبى والعضلى. التحكم بمستوى السكر فى الدم وتجنب ارتفاع مستوياته عن المعدل الطبيعى والتحرك المبكر والمنظم للمرضى الموضوعين على جهاز تنفس صناعى وذلك لتعجيل فصل المرضى من عليه ومن ثم يتطلب الامر المزيد من الدراسات حول مختلف االمراض والربط بين شدتها وطرق عالجها ومضاعافاتها وبين اختبارات التشخيصيه الكهربي.