

# Epidemiological, Clinical, Paraclinical and Prognostic Profile of Children Aged 0 to 5 Years with Cerebral Palsy in Medical Department of Niamey National Hospital (NNH)

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**How to cite this paper:** Daou, M., Sidibé, H., Brah, S., Adamou, K.A., Andia, D.K.A., Mamadou, Z., Konaté, M., Kaka, Y.A., Bako, D.I., Beydou, S., Seydou, Y., Adehossi, E. and Sanoussi, S. (2022) Epidemiological, Clinical, Paraclinical and Prognostic Profile of Children Aged 0 to 5 Years with Cerebral Palsy in Medical Department of Niamey National Hospital (NNH). *Open Journal of Internal Medicine*, 12, 69-83.

<https://doi.org/10.4236/ojim.2022.121009>

**Received:** December 28, 2021

**Accepted:** March 21, 2022

**Published:** March 24, 2022

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## Abstract

**Introduction:** Cerebral palsy is the most common motor disorder of childhood according to the Center for Disease Control and Prevention (CDC). It is much more common in premature infants. The majority of fixed organic lesions occur during the development of the central nervous system. **Objective:** To study the epidemiological, clinical, paraclinical and prognostic profile of children aged 0 to 5 years with Cerebral Palsy (CP) seen in consultation in the medical neurology department of the National Hospital of Niamey. **Methods:** This was a transverse descriptive study with prospective collection of patient records (0 - 5 years) over a 6-month period. **Results:** During our study, 100 children aged 0 to 5 years had to be consulted. The prevalence was 61%. 62.3% of patients were male. The mean age was 2.01 years. The majority of the patients were from the urban area (63.93%). Almost half of the mothers had no schooling. 4.92% of patients were from consanguineous marriages. Pregnancies were monitored with at least 4 prenatal visit in 86.98% of cases and 83.61% of children were born at term. In 75.41% of cases, patients were born by eutocic delivery. In 36.07% of cases, the patients were resuscitated at birth. Fetal distress was present in 29.5%. Seizures were present in 49.18% of the patients of which 50% were generalized. Tonicity was not good in 35.15% and among them 52.63% had hypertonia. 18.03% of patients had a motor deficit. For 34.43% of the patients the first nerve was affected, followed by the

third nerve with 13.11%. 65.57% of the patients had fixed retardation and for 78.69% of the patients the retardation was homogeneous. Neuromalaria, meningitis and hydrocephalus were the most incriminated causes of delay in 63.51%, 19.67% and 18.03% of cases respectively. Anticonvulsants and psychoanalptics were the most used in 49.18% and 32.79% of cases respectively. 32.79% of patients had received motor rehabilitation. All mothers had received psychotherapy. **Conclusion:** Cerebral palsy (CP) is a pathology that is gaining ground and whose management is multidisciplinary. However, preventive measures are quite effective to limit the risks of their occurrence.

## Keywords

Cerebral Palsy, Epilepsy, NNH, Niger

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## 1. Introduction

Based on an international consensus, a generally agreed upon definition of Cerebral Palsy (CP) is as follows:

CP describes a group of permanent disorders of movement and posture, causing activity limitation, that is attributed to nonprogressive disturbances that occurred in the developing fetal or immature brain. The motor disorders of CP are often accompanied by disturbances of sensation, perception, cognition, communication, and behavior, by epilepsy, and by secondary musculoskeletal problems [1].

CP is characterized by heterogeneity in risk factors, underlying specific etiology, clinical features, severity of functional limitations, associated and secondary conditions, treatment options, and evolution of the condition over the lifespan of the individual [2] [3] [4]. Shevell (2019) has explored the argument for a consideration to view CP as a spectrum disorder rather than a discrete unitary clinical condition [5].

The majority of fixed organic lesions (80% - 90%) occur during the development of the central nervous system during the ante- and perinatal periods and in 10% - 20% of cases after this period [6].

According to data from the majority of studies, the prevalence of CP is between 1.5 and 4 per 1000 worldwide [7]. Cerebral palsy is the most common motor disorder of childhood according to the Center for Disease Control and Prevention (CDC) [8]. It is much more common in premature infants and according to Nelson accounts for 50% of cases in premature infants [9].

In the United States, the average prevalence of CP in four cities (Alabama, Georgia, Missouri, and Wisconsin) was 3.3 per 1000 births in 2002 [10].

According to the Surveillance of Cerebral Palsy in Europe (SCPE) network, Cerebral Palsy affects approximately 2 children per 1000 births, or 1500 new cases each year in the general population of developed countries. It affects males 1.33 times more than females [7].

In France, the available data for prevalence show that the CP is between 1.15 and 1.25 per 1000. There is a common trend, that is to say a stability of the prevalence of the pathology for more than 30 years in all developed countries [11].

The cost of care for children with cerebral palsy is ten (10) times higher than for children without cerebral palsy or intellectual disability (\$16,721 per year versus \$1674 in 2005) [12].

In the Democratic Republic of Congo (D.R.C.), it is estimated that 2% of children are disabled, of which 1.2% are in the 0 - 4 year age group, which is the preferred period for CP (0 - 2 years) [13].

Cerebral palsy affects 6% - 8% of children with a birth weight of less than 1500 grams or born before 30 weeks of pregnancy [14] [15]. Cerebral palsy is more common the earlier the birth and/or the lower the birth weight of the child [16]. Among children born after 27 and 30 weeks of amenorrhea, with a birth weight varying between 500 and 1280 grams, 13% have a severe handicap, 25% have a moderate handicap and 70% will present school problems despite a normal intellectual quotient [15].

Based on this observation, we measure its disabling impact on society, its lack of knowledge by providers and decision makers, justifying the paucity of epidemiological and clinical data, especially in Niger.

Hence our motivation to carry out this study in order to assess the situation of children with Cerebral Palsy from 0 to 5 years of age in the Internal Medicine and Neurology Department of the National Hospital of Niamey.

## 2. Patients and Methods

This was a descriptive cross-sectional study with prospective collection of the records of patients (0 to 5 years) who consulted for CP in the Internal Medicine and Neurology Department during the data collection period, which lasted 6 months (May 1 to October 31, 2020).

Patients aged 0 - 5 years with CP seen in the service were included in the study.

Patients with cerebral palsy associated with a genetic neurological pathology were not included.

The sample was exhaustive and included patients who met the above-mentioned inclusion criteria. The sample size is 61 Cerebral Palsy cases.

The data were collected by the administration of a questionnaire established for the circumstance.

The variables studied were socio-demographic parameters, history, clinical examination signs, causes and characteristics of BMI and means of treatment.

The assessment tool was the Blantyre Score.

A research authorization was obtained from the Faculty of Science and Health. Anonymity was respected during the analysis of the data. We removed the first and last names and all indications that could identify the patients. The use of the results of our work will be limited to the strict exploitation related to

the objectives of the study. Informed consent was signed by the participants of the survey.

### 3. Results

During our study, 100 children aged 0 to 5 years had to be consulted, 61 of them for CP; that is, a prevalence of 61%. In 62.3% of the cases (n = 38) the patients were male; the sex ratio (Male/Female) was 1.65. The mean age of the patients in our study was  $2.01 \pm 1.24$  years with extremes ranging from 3 months to 5 years. The 2nd child was the most affected with 18.03% of cases (n = 11). The majority of patients were from the urban area, *i.e.* 63.93% of cases (n = 39).

Mothers aged 25 to 29 years were more represented. The average age of the mothers was  $30.54 \pm 6.55$  years with extremes ranging from 20 to 46 years. In 80.33% of cases (n = 49), the mothers were housewives. Nearly half of the parturients did not attend school (40.98% of cases).

In 39.34% of cases (n = 24), the fathers were shopkeepers. In 40.98% of cases (n = 25), the fathers were not been to school (See **Table 1**).

**Table 1.** Socio-demographic and clinical characteristics of the patients.

Character	Variables	Number	%
Sex	M	38	62.3
	F	23	37.7
Age of patients (years)	≤2	44	72.13
	[2 - 4]	7	11.48
	[4 - 5]	10	6.25
Rank in siblings	1 - 3	27	44.36
	4 - 6	24	39.34
	7 - 9	10	16.39
Age of mothers	≤24	11	18.03
	25 - 34	33	54.10
	≥35	17	27.86
Educational level of mothers	Not in school	25	40.98
	Primary	24	39.34
	Secondary	8	13.11
	Higher	4	6.56
Clinical signs	Convulsive seizures	30	49.18
	Attention deficit disorders	18	29.51
	Language disorders	6	9.84
	Tone disorders	19	31.15
	Motor deficit	11	18.03
	Dysphagia	10	16.4

### *Personal History*

Pregnancies were followed up with at least 4 prenatal consultations (PNC) in 86.98% of cases (n = 53). All women had received iron and folic acid supplementation, deworming and intermittent preventive treatment (IPT) against malaria. In 11.48% of the cases (n = 7) the patients were born prematurely. The health center was the place of birth for 93.44% of patients (n = 57). In 13.11% of cases (n = 8), the delivery was dystocic. At the first minute only 1.64% of the patients (n = 1) had an Apgar score between 0 and 3, while those with a score between 4 and 6 at the first and fifth minute represented 13.11% (n = 8) and 4.92% (n = 3) respectively. For 31.15% of patients (n = 19), the birth weight was between 2500 and 3500 g. In 27.87% of cases (n = 17), the height at birth was less than 50 cm. Almost 1/4 or 24.59% of the patients (n = 15) had a normal head circumference. In 70.5% of cases, the patients had experienced respiratory distress. In 68.85% of cases (n = 42), patients had an up-to-date vaccination status. In our study, 65.57% of patients (n = 40) had already had neurological malaria.

### *Family history*

Unexplained miscarriage was the most common family history in 24.59% of cases (n = 15) followed by hypertension and sickle cell disease respectively with 3.19% of cases each (n = 8).

### *Clinical examination*

#### *General signs*

Consciousness was preserved in 98.36% of patients (n = 60). Mucous membranes were stained in 98.36% of patients (n = 60). Seizures were present in 49.18% of the patients (n = 30) of which 50% (n = 15) were generalized. The average weight was  $7.78 \pm 3.98$  kg with extremes ranging from 5 to 18. The average height was  $91.2 \pm 6.21$  cm with extremes ranging from 47 to 132 cm. In 36.07% of cases (n = 22), the height was between 70 and 79 cm. In 37.70% of patients (n = 23) the head circumference was between 45 and 49 cm. 14.75% of patients (n = 9) had severe acute malnutrition on admission. 24.59% of patients (n = 15) had microcrania.

#### *Physical signs*

Autonomous walking was present in 100% of the patients (n = 61), as well as grasping, Moro's reflexes and the 4 cardinal points. Attention disorders were found in 29.51% of patients (n = 18). In 68.85% of the cases (n = 42) the tonicities were good. Among the patients who had tonicities disorders, 52.63% (n = 10) had hypertonia. In our study, 18.03% of patients (n = 11) had a motor deficit. Muscle strength was rated at 5/5 for 60.66% of patients (n = 37). Sensitivity was preserved in all patients (n = 61). In 34.43% of the patients (n = 21) the 1st nerve was affected, followed by the 3rd with 13.11% (n = 8). Gastroesophageal reflux syndrome was found in 27.87% (n = 17) of patients.

#### *Paraclinical signs*

CT scan abnormalities were found in 80.34% of patients (n = 49). Diffuse point-waves and epileptic seizures were found in 50% of cases each (n = 10).

### *Etiologies*

Neuromalaria, meningitis and hydrocephalus were the most incriminating in 31.15% (n = 19), 19.67% (n = 12) and 18.03% (n = 11) of cases respectively.

### *Treatment*

Anticonvulsants and phytotherapy were the most used with 49.18% and 32.79% respectively. In our study 32.79% (n = 20) of the patients had received motor rehabilitation and the evolution was favorable for 45% of them. All mothers had received psychotherapy.

For 65.57% of the patients (n = 40) the delay was fixed. For 78.69% of patients (n = 48) the delay was homogeneous.

## **4. Discussion and Comments**

In our series, CP represented 61% of the pathology of the children followed in these services.

This percentage is higher than that found in the majority of publications. Our results are higher than those of Ramanatsoa [17] in Madagascar which is 22.29%, Motchie [18] in Cameroon (20%), Gandema [19] in Burkina (11%), Nottidge in Ibadan [20] in Nigeria which is 16.2%, Tiemoko [21] in Burkina Faso at 21.48% and Marlene [22] in France who found 23%. We can explain this discrepancy on the one hand, by the specialization of our department in neurological diseases and on the other hand, by the fact that the majority of published studies use incidence or prevalence in determining the frequency of BMI, whereas in our study, we reported the frequency of BMI in a population of children consulting for different pathologies.

The predominance of males in our patients is clear. It represented 62.3% of the population with a sex ratio of 1.65. This male predominance was also found in the work on BMI of Djibo [23], Ramanatsoa [17] and Ndiaye [24] at 59%. Motchie *et al.* [18] found 60.54% of patients to be male and a sex ratio of 1.3. Saleh *et al.* [25] found 69% of male patients in their series and a sex ratio of 1.45. The work of Karuma *et al.* [26] carried out in Dar-es-Salam (Tanzania) also found 56 male patients out of 100 patients with CP.

However, a study carried out in Yaoundé by Bediang [27] in 2007 showed a predominance of women (52.5%) and for Serdaroglu *et al.* [28], gender has no effect on the prevalence of BMI.

These results lead us to ask questions. Is there not a correlation between Cerebral Palsy and gender? Would male gender increase the susceptibility of children to develop permanent brain damage during brain growth? Or would female individuals be more resistant to pathologies that could cause permanent damage during brain growth and maturation?

Johnston MV *et al.* [29] in a study of adult animals and adult patients in Baltimore (USA) stated that cerebral palsy is more frequent in males than in females. Sex hormones (estrogens) are thought to protect against anoxic damage. The brain of the fetus and the newborn would also be influenced by

these hormones. He states in his article that other reports indicate that there is a neurobiological difference between the neurons of male and female subjects, leading to a differentiation of responses during the onset of brain damage.

The average age of the patients was 2.01 years (25 months) with extremes from 3 months to 5 years. This agrees with Ndiaye [24] who found an average age of 24 months. Saleh *et al.* [25] in their study on the clinical aspects of CP in Jordan found a mean age of 38.3 months.

CP usually occurs in a growing brain. This period corresponds to the first three years of life [30]. These results are consistent with our own (73.77% are older than 1 year) and with the data in the literature. This is also illustrated in the study of Djibo [23] in Mali and that of Motchie [18] *et al.* in Cameroon, which found that 71.43% of the patients were more than one year old at the time of the consultation. According to Cahuzac M. *et al.* [31] this age group would be the one of diagnostic accuracy. Very often, parents only came to consult their child after having noticed the inefficiency of self-medication.

We note that 40.98% of the mothers and fathers had no education. This can have a considerable negative impact on the management of the disease. These results are almost superposable to those of Djibo [23] and Tiemoko [21] in Mali.

The occupation of the parents shows that housewives were the most represented with 80.33% and men, traders represented 39.34%. Our results agree with those of Sidibe B [32] and Prazuck *et al.* [33].

This could be explained by the low schooling rate of the Nigerien population, especially among women.

We consider it important to mention the place of delivery because home deliveries are most often carried out in rudimentary conditions. Among the CP cases studied, 93.44% were born in health facilities compared to 6.56% at home, as also illustrated in the study by Ramanatsoa [17]. Here, despite the predominance of delivery in health facilities, home delivery should not be ignored.

In relation to the mode of delivery, a large majority of 75.41% of the CP cases resulted from normal vaginal delivery, as opposed to 13.11% from instrumental delivery and 11.48% by caesarean section. This paradoxical finding between the predominance of cerebral pain, the use of health facilities and eutocic delivery allows us to deduce for the study that the type of delivery is probably not the only factor predisposing to the occurrence of cerebral palsy due to cerebral pain. Thus, it is likely that most of the damage occurs during pregnancy and less than previously thought during delivery.

Children with a birth weight between 2500 - 4000 grams represent 32.79% of CP. These results differ from the data in the literature. This is explained by the fact that in most of our health facilities, low birth weight babies have a high morbidity. As a result, most of them die in the first days of life.

Cerebral palsy affects 6 to 8% of children with a birth weight of less than 1500 grams. Cerebral palsy is more frequent the lower the birth weight of the child [34]. This is explained by the increasing frequency of per partum pathologies that lead to in utero growth retardation and low birth weight.

In premature infants, the lesions responsible for CP are characteristically located or predominate in the white matter of the cerebral hemispheres; this is a chain of toxic phenomena for the brain cells, leading to the death or poor growth of neurons. This results in periventricular leukomalacia (PVML) due to lesions of the white matter around the cerebral ventricles.

Prematurity represents 11.48%. According to Metayer M. *et al.*, the prematurity factor concerns 30% to 40% of the CP population [35]. Keogh *et al.* [36], Li S. *et al.* [37], Livinec [38] and Tiemoko [21] found respectively 25%, 25%, 32% and 34.5% of prematurity.

Our results only confirm that a prematurely terminated pregnancy remains the first cause of hospital morbidity in tropical settings [39]. However, in our study this first place of prematurity in the causes of CP seems relative to us, given the high frequency of its relationship with socio-economic conditions, medical decision (maternal or fetal rescue), compliance with treatment and negligence that prevail in developing countries such as Burkina Faso.

In our sample, the anamnesis found that fetal suffering, whether pre, per or immediately post partum, occupied 14.75%, far from that of Djibo [23] at 31% and Nguefack *et al.* [40] in Cameroon at 70%. In a previous survey in Senegal, Ndiaye *et al.* [24] found a clear predominance of abnormalities of pregnancy and delivery, 44.08%. Fetal suffering is a major problem in the neonatal period because of its frequency, severity and consequences on the psychomotor development of the child. Good monitoring of the fetus, assisted extraction and good management in the delivery room could decrease the frequency of BMI related to fetal distress. Despite these resuscitation measures, the study by Diarra [41] showed that 15.3% of acute fetal distress developed neurological complications. El Idriss [42] in Morocco found 24% of perinatal asphyxia.

According to a study of neonatal morbidity and mortality in the pediatric intensive care unit at Gabriel Touré Hospital, 37 cases, *i.e.* 25.3% of children developed neurological sequelae after fetal distress [12].

The infectious cause was dominated by bacterial meningitis, severe malaria and neurological form in the course of the study with respectively 18.03% and 65.57%. According to Cahuzac M, the different germs incriminated were listeriosis, toxoplasmosis and rubella [34]. El Idriss [42] had 14.6% neonatal infection; Nguefack *et al.* [40] had 6% meningitis and 13.7% neuromalaria; Tiomoko [21] found 22.41% neonatal infection. The sequelae of these infections are mostly due to delays in treatment in specialized services and also because Niger continues to have meningitis epidemics due to its location in the “Lapeyssonie belt”. It was found in 4.92% of patients. Our results are lower than those of Youssef [34] and El Idriss [42] in Morocco who obtained 11% and 14% respectively.

The literature mentions other causes such as embryopathy, drug intoxication, trauma, organic and metabolic encephalopathies.

The diagnosis of epileptic seizures in CP is difficult because of the non-epileptic non-motor manifestations related to CP: tonic spasms, identification of non-epileptic myoclonus. The use of Electroencephalogram (EEG) is important, but the



difficulty of performing this examination makes its interest limited [43] [44]. The specificity of EEG in case of epilepsy associated with CP is 86% [45]. Seizures affected 49.18% of the CP children in our study. These results are comparable to data in the literature where the incidence of epilepsy reaches 30% to 71% [46]. El Idriss [42] had in his series 40% epileptic seizures, Youssef [34], 39.45% and Nguefack [40], 57.5%. According to Sheila J [46], the most reported type of epilepsy in the different CP entities seems to be generalized seizures with a prevalence of 50%.

Axial hypotonia in 47.37% and functional impotence in 18.03% of cases was found in our series. The series by Ndiaye [24] in Senegal found a frequency of axial hypotonia of 48.04%. This result was similar to that of Gérard P. and Traore who incriminated prematurity in this syndrome [47] [48].

Gastrointestinal problems and malnutrition are very common in children with CP [49] [50]. Campanozzi *et al.* [51] showed that malabsorption syndrome was present in 44.4% of children with CP. This rate was lower in our series: 4.92%.

Lewis *et al.* [52] proposed that Gastroesophageal reflux disease (GERD) is not a consequence but participates in malnutrition. Despite treatment with a proton pump inhibitor for 6 months, 55.5% of children with CP continue to have GERD [51] [53] [54].

According to Campanozzi [51], 67% of children with CP had constipation, and 43% had constipation associated with GERD. In our study, constipation was present in 1.64% of children and GERD in 27.87%.

Wesley [55] noted that chronic constipation was due to persistent excessive intra abdominal pressure which also gives GERD. Children with CP achieve urinary and fecal sphincter control at a later age than the normal population [56] [57]

Brain damage in the child with CP may be accompanied by several visual disorders. These disorders fall into three categories: Sensory disorders which correspond to a decrease in visual acuity and a decrease in the visual field, oculomotor disorders which affect the nerves and the ocular muscles and which are represented essentially by strabismus and finally, neuro-visual disorders which correspond to a bad interpretation of the environment by the brain [58].

We found frequent ocular disorders at 12.7% manifested by strabismus (13.11%), a percentage different from that of Traore Y and Wallace [48] [59].

Karen [60] reported a prevalence of ophthalmologic disorders in children followed for BMI of 33%. Dufresne *et al.* [58] found that the most frequent ophthalmologic abnormalities in children followed for CP were strabismus (36.4%) followed by myopia (12.9%). In the study by El Idriss [42], the ophthalmological examination was abnormal in 206 cases (34%) and the predominant disorder was strabismus (78%). These ocular disorders reinforce the handicap of these children and the despair of their parents.

In our series, CT scan abnormalities represented 80.34% of cases and were dominated by cortical and subcortical lesions with 47.55%. Our result is inferior

to those of N'guefack [40] and Youssef [34] who found CT scan abnormalities in 90.5% and 82.3% of cases respectively. However, it is higher than that of El Idriss [42] with 77%. Karen [60] reported in a series of 122 children with BMI that the CT scan was abnormal in 75%. This rate is comparable to the study of Spinileo [61] and Li S. [37]. In spastic hemiplegia, Okumara *et al.* [62] and Steinlin *et al.* [63] reported that brain imaging was abnormal in 92% and 91% respectively. The predominant lesion was periventricular leukomalacia with a percentage of 42% [62].

Youssef [34] found cortical atrophy in 38.11% and hydrocephalus in 9.23% and El Idriss [42] found hydrocephalus in 6%.

The management of CMI must be early and multidisciplinary and the detection of a handicap in children at risk must be done during systematic monitoring during the first years of life [63]. This regular follow-up allows for real support of the child and his or her family by providing parental guidance from the start. BMI can only be absorbed in the complexity of its medical, psychological, family and educational components. Each one has its role to play at a given time, and a close correlation of actions is always necessary.

The management of motor control is essentially based on rehabilitation which has several objectives:

Learning motor skills will allow to get closer to the pre-programmed normal motor skills. There are attitudes and motor behaviors that are already regulated, which do not require learning. The role of the physiotherapist is to learn what does not come automatically to the child with CP. The more the automatic motor skills are impaired, the less natural compensation there will be. The role of therapeutic workers is to preserve the locomotor system as long as possible. Thus, the challenge is to maintain the acquired motor level or level of function [64].

In our series, 32.79% of our patients had followed motor rehabilitation sessions. This re-education is stressful for the parents, especially as they have to wait for hours because the re-education service is overloaded, especially as several sessions are needed.

The medical treatment reserved for CP is a treatment especially for complications. Indeed, botulinum toxin injections aim to decrease the degree of spasticity. The administration of botulinum toxin is done by intramuscular injection; it acts in a transient and localized manner on spasticity but does not act on muscle retraction and viscosity. It is indicated in postoperative care and as an aid to orthopaedic devices [65].

The administration regime of botulinum toxin depends on the age of the child and the number of muscles targeted. Injections are administered using conventional neurophysiological localization techniques [66]. Botulinum toxin has been shown to be effective in the treatment of equine feet in 50% to 61% of cases [67].

The use of anticonvulsants, vasodilatory phytotherapy and psychoanaleptics is also very frequent in order to improve the quality of life of patients. In our series

they were used respectively in 49.18% and 32.79% and 13.11%. None of our patients benefited from botulinum toxin injections.

These products are expensive and their availability causes problems, especially botulinum toxin. Therefore, drug treatment is not effective for these patients.

The majority of parents of children with CP experience a difficult and negative social and financial ordeal. Their mental, physical and psychological state is affected by the presence of a disabled child in the family [68].

In our series, 65.75% of patients had a fixed course and 78.69% had a homogeneous course. These data are consistent with the literature and are characteristic of children followed for Cerebral Palsy.

Most of these children survive into adulthood. Significant sucking and swallowing disorders that may require a gastrostomy reduce life expectancy.

Limitation of the study is the reluctance of mothers to cooperate, especially those who were not educated and difficult access to certain examinations, especially imaging, due to constant breakdowns of the equipment, despite the fact that they are free.

## 5. Conclusion

Cerebral palsy (CP) is the permanent, nonprogressive motor consequence of lesions that have reached the developing brain during conception through the fifth year of life. These brain lesions do not worsen, but as the child grows, the consequences become more noticeable and deformities may appear. It can be detected by the absence or persistence of archaic reflexes and by the disturbance or significant delay in psychomotor development. The symptomatology depends on the location and extent of the brain lesions. The treatment is multidisciplinary, long and involves a significant financial cost.

## Conflicts of Interest

The authors declare no competing interest.

## References

- [1] Rosenbaum, P., Paneth, N., Leviton, A., et al. (2007) A Report: The Definition and Classification of Cerebral Palsy April 2006. *Developmental Medicine and Child Neurology*, **109**, 8-14.
- [2] Patel, D.R., Greydanus, D.E., Calles, J.L., et al. (2010) Developmental Disabilities across the Lifespan. *Disease-a-Month*, **56**, 305-397. <https://doi.org/10.1016/j.disamonth.2010.02.001>
- [3] Stavsky, M., Mor, O., Mastrolia, S.A., et al. (2017) Cerebral Palsy—Trends in Epidemiology and Recent Development in Prenatal Mechanisms of Disease, Treatment, and Prevention. *Frontiers in Pediatrics*, **5**, Article No. 21. <https://doi.org/10.3389/fped.2017.00021>
- [4] Novak, I., Morgan, C., Adde, L., et al. (2017) Early, Accurate Diagnosis and Early Intervention in Cerebral Palsy: Advances in Diagnosis and Treatment. *JAMA Pediatrics*, **171**, 897-907. <https://doi.org/10.1001/jamapediatrics.2017.1689>
- [5] Shevell, M. (2018) Cerebral Palsy to Cerebral Palsy Spectrum Disorder: Time for a Name Change? *Neurology*, **92**, 233-235.

- <https://doi.org/10.1212/WNL.0000000000006747>
- [6] Tangara, G. and Keita, S. (2001) Profil démographique et économique du Mali 1960-2000. Programme majeur population développement, Bamako.
- [7] Johnson, A. (2002) Prevalence and Characteristics of Children with Cerebral Palsy in Europe. *Developmental Medicine and Child Neurology*, **44**, 633-640.
- [8] Capute, A.J. and Accardo, P.J. (2008) Capute & Accardo's Neurodevelopmental Disabilities in Infancy and Childhood. 3rd Edition, Brookes Publishing, Baltimore.
- [9] Nelson, K.B. (2002) The Epidemiology of Cerebral Palsy in Term Infants. *Mental Retardation and Developmental Disabilities Research Reviews*, **8**, 146-150.  
<https://doi.org/10.1002/mrdd.10037>
- [10] Yeargin-Allsopp, M., Braun, K.V.N., Doernberg, N.S., et al. (2008) Prevalence of Cerebral Palsy in 8-Year-Old Children in Three Areas of the United States in 2002: A Multisite Collaboration. *Pediatrics*, **121**, 547-554.
- [11] Rumeau, R.C., Mazaubrun, C. and Verrier, A. (1994) Prévalence des handicaps: Evolution dans trois générations d'enfants 1972, 1976, 1981. Editions INSERM, Paris, 177 p.
- [12] Coulibaly, S. (2003) Fièvres prolongées chez l'enfant: Etude clinique, étiologique et évolution dans le service de pédiatrie de l'hôpital Gabriel Touré. Thèse de Médecine, Université de Bamako, Bamako, 82 p.
- [13] Mossige, A. (2003) Etude pilote de risque et de la vulnérabilité en république démocratique du Congo. Rapport Préliminaire, 2ème Version, Kinshasa.
- [14] INSERM (2004) Déficiences ou handicap d'origine périnatale dépistage et prise en charge.
- [15] Association des paralysés de France (1996) Déficiences motrices et handicaps, aspects sociaux, psychologiques, médicaux, techniques et législatifs, troubles associés. APF/Editions Vuibert, Paris, 505 p.
- [16] Philippe, D., Chocard, A.S., Malka, J. and Ninus, A. (2011) Infirmité Motrice Cérébrale. In: Duverger, P., et al., Eds., *Psychopathologie en service de pédiatrie*, Elsevier Masson SAS, Amsterdam, 264-266.  
<https://doi.org/10.1016/B978-2-294-70689-9.00036-3>
- [17] Ramanatsoa, Y. (2003) Les infirmités motrices cérébrales consécutives à l'accouchement dystocique. Médecine Humaine, Antananarivo.
- [18] Motchie, F., Mbonda, E. and Camara, M. (1992) Infirmités motrices cérébrales: Aspects étiologiques, cliniques et thérapeutiques. Thèse de Médecine, Université de Yaoundé I, Centre Universitaire des Sciences de la Santé, Yaoundé.
- [19] Gandema, S. (2012) Profil épidémiologique du handicap physique au Burkina Faso. *Médecine d'Afrique Noire*, N°5911, 542-547.
- [20] Nottidge, V.A. and Okogbo, M.E. (1991) Cerebral Palsy in Ibadan, Nigeria. *Developmental Medicine and Child Neurology*, **33**, 241-245.  
<https://doi.org/10.1111/j.1469-8749.1991.tb05113.x>
- [21] Tiemoko, S. (2014) Profil épidémiologique et clinique des infirmités motrices cérébrales au Centre hospitalier universitaire Sourô Sanou de Bobo-Dioulasso. Université Polytechnique de Bobo Dioulasso, Bobo-Dioulasso, 130 p.  
<http://www.beep.ird.fr/collect/upb/index/assoc/INSSA-2013-SAN-PRO/INSSA-2013-SAN-PRO.pdf>
- [22] Marlène, G. (2015) Prévention et handicap moteur. Université Paris Diderot, Paris.
- [23] Djibo, A. (1988) Etude épidémie-clinique de l'infirmité motrice cérébrale (IMC) chez les enfants de 0-14 ans dans les services de rééducation de Bamako à propos

- de203 cas. Université de Bamako, Bamako.
- [24] Ndiaye, M., Thiama, B., Ndao, A.K., Sene-Diouf, F., Diop, A.G., et al. (2002) Cerebral Palsy in Dakar. *Dakar Medical*, **47**, 77-80.
- [25] Al Ajlouni, S.F., Aqrabawi, M., Oweis, N. and Daoud, A.S. (2006) Clinical Spectrum of Cerebral Palsy in Jordanian Children: An Analysis of 200 Cases. *Journal of Pediatric Neurology*, **4**, 251-255. <https://doi.org/10.1055/s-0035-1557333>
- [26] Karumuna, J.M. and Mgone, C.S. (1990) Cerebral Palsy in DAR-ES-SALAM. *Central African Journal of Medicine*, **36**, 8-10.
- [27] Bediang, G.W. (2007) Aspects Cliniques, Etiologiques et Scanographiques des Infirmités Motrices Cérébrales de l'enfant à Yaoundé.
- [28] Serdaroqlu, A., Consu, A., Özkan, S. and Tezcan, S. (2006) Prevalence of Cerebral Palsy in Turkish Children between the Ages of 2 and 16 Years. *Developmental Medicine and Child Neurology*, **48**, 413-416. <https://doi.org/10.1111/j.1469-8749.2006.tb01288.x>
- [29] Johnston, M.V. and Hagberg, H. (2007) Sex and the Pathogenesis of Cerebral Palsy. *Developmental Medicine and Child Neurology*, **49**, 74-78. <https://doi.org/10.1017/S0012162207000199.x>
- [30] Georges, T. (2011) Problèmes orthopédiques de l'IMC. Elsevier Masson SAS, Paris.
- [31] Cahuzac, M., Claverie, P. and Nichil, J. (1980) L'enfant infirme moteur d'origine cérébrale. Masson, Paris, 27-70.
- [32] Sidibé, B. (1986) Contribution à l'étude des accouchements prématurés en milieu obstétrical Bamakois à propos de 140 cas. Université de Bamako, Bamako, 116 p.
- [33] Prazuck, T., Tall, F., Roisin, A.J., et al. (1993) Risk Factors for Preterm Delivery in Burkina Faso (West Africa). *International Journal of Epidemiology*, **22**, 489-494. <https://doi.org/10.1093/ije/22.3.489>
- [34] Youssef, E. (2014) Epidémiologie de l'infirmité Motrice cérébrale. Université Cadi Ayyad, Maroc.
- [35] Metayer, M. (2003) Kinésithérapie pédiatrique, Rééducation cérébromotrice du jeune enfant. Education thérapeutique. 2ème Edition, Masson, Paris.
- [36] Keogh, J.M. and Badawi, N. (2006) The Origins of Cerebral Palsy. *Current Opinion in Neurology*, **19**, 129-134. <https://doi.org/10.1097/01.wco.0000218227.35560.0d>
- [37] Li, S., Hong, S.X., Wang, T.M., et al. (2003) Premature, Low Birth Weight, Small for Gestational Age and Childhood Cerebral Palsy. *Chinese Journal of Pediatrics*, **41**, 344-347.
- [38] Livinec, F., Ancel, P.Y., Marret, S., et al. (2005) Prenatal Risk Factors for Cerebral Palsy in Very Preterm Singletons and Twins. *Obstetrics & Gynecology*, **105**, 1341-1345. <https://doi.org/10.1097/01.AOG.0000161375.55172.3f>
- [39] Lespargot, A., Tardieu, C. and Bret, M.D. (1989) La chirurgie tendineuse des fléchisseurs du genou est-elle justifiée chez l'IMC. *Rev Chir Orthop*, **75**, 532-536.
- [40] Nguéack, S. (2015) Aspects Cliniques et Étiologiques des Infirmités Motrices Cérébrales chez l'Enfant À Yaoundé. *Health Sciences and Disease*, **16**, 5.
- [41] Diarra, K. (2008) Souffrance fœtale aigue dans le service de Gyneco Obstetrique du CHU gabriel touré de Bamako. Thèse 2008, 130 p.
- [42] El Idrissa, T.E.A. (2014) Profil épidémiologique, clinique, paraclinique et évolutif de l'infirmité motrice cérébrale au niveau du service de pédiatrie A du CHU Mohammed VI Marrakech. Thèse 2014, 78 p.
- [43] Perlstein, M., Gibbs, E.L. and Gibbs, F.A. (1953) The Electroencephalogram in In-

- fantile Cerebral Palsy. *American Journal of Physical Medicine & Rehabilitation*, **34**, 477-496.
- [44] Sussova, J., Seidl, Z. and Faber, J. (1990) Hemiparetic Forms of Cerebral Palsy in Relation to Epilepsy and Mental Retardation. *Developmental Medicine & Child Neurology*, **32**, 792-795.
- [45] Panteliadis, C., Jacobi, G., Covanis, A., et al. (2002) Epilepsy in Children with Congenital Hemiplegia: Correlation between Clinical, EEG and Neuroimaging. *Epileptic Disorders*, **4**, 251-255.
- [46] Wallace, S.J. (2001) Epilepsy in Cerebral Palsy. *Developmental Medicine & Child Neurology*, **43**, 713-717.
- [47] Bobath, B. and Bobath, K. (1985) Développement de la motricité des enfants IMC. Masson, Paris.
- [48] Youssouf, T. (2009) Aspects épidémiologiques, cliniques, thérapeutiques et évolutifs de la diplégie spastique. Université Cadi Ayyad Faculté de Médecine et de Pharmacie Marrakech, Marrakech.
- [49] Dahl, M., Thommessen, M., Rasmussen, M. and Selberg, T. (1996) Feeding and Nutritional Characteristics in Children with Moderate or Severe Cerebral Palsy. *Acta Paediatrica*, **85**, 697-701. <https://doi.org/10.1111/j.1651-2227.1996.tb14129.x>
- [50] Odding, E., Roebroek, M.E. and Stam, H.J. (2006) Cerebral Palsy in Childhood *Disability and Rehabilitation*, **28**, 183-191. <https://doi.org/10.1080/09638280500158422>
- [51] Campanozzi, A., Winter, S. and Meberg, R. (2007) Impact of Malnutrition on Gastrointestinal Disorders and Gross Motor Abilities in Children with Cerebral Palsy. *Brain & Development*, **29**, 25-29. <https://doi.org/10.1016/j.braindev.2006.05.008>
- [52] Lewis, D., Khoshoo, V. and Pencharz, P.B. (1994) Impact of Nutritional Rehabilitation on Gastroesophageal Reflux in Neurologically Impaired Children. *Journal of Pediatric Surgery*, **29**, 167-169. [https://doi.org/10.1016/0022-3468\(94\)90312-3](https://doi.org/10.1016/0022-3468(94)90312-3)
- [53] Grunow, J.E., Al-Hafidh, A. and Tunell, W.P. (1989) Gastroesophageal Reflux Following Percutaneous Endoscopic Gastrostomy in Children. *Journal of Pediatric Surgery*, **24**, 42-44. [https://doi.org/10.1016/S0022-3468\(89\)80298-2](https://doi.org/10.1016/S0022-3468(89)80298-2)
- [54] Sulaeman, E., Udall, J.N., Brown, R.F., et al. (1998) Gastroesophageal Reflux and Nissen Fundoplication following Percutaneous Endoscopic Gastrostomy in Children. *Journal of Pediatric Gastroenterology and Nutrition*, **26**, 269-273. <https://doi.org/10.1097/00005176-199803000-00006>
- [55] Wesley, J.R., Coran, A.G. and Sarahan, T.M. (1981) The Need for Evaluation of Gastroesophageal Reflux in Brain-Damaged Children Referred for Feeding Gastrostomy. *Journal of Pediatric Surgery*, **16**, 866-871. [https://doi.org/10.1016/S0022-3468\(81\)80836-6](https://doi.org/10.1016/S0022-3468(81)80836-6)
- [56] Canadian Paediatric Society (2000) Toilet Learning: Anticipatory Guidance with Child-Oriented Approach. *Paediatrics & Child Health*, **5**, 333-335. <https://doi.org/10.1093/pch/5.6.333>
- [57] Satila, H., Kotamaki, A. and Koivikko, M. (2006) Low- and High-Dose Botulinum Toxin A Treatment: A Retrospective Analysis. *Pediatric Neurology*, **34**, 285-290. <https://doi.org/10.1016/j.pediatrneurol.2005.08.031>
- [58] Dufresne, D. and Dagenais, L. (2014) Spectrum of Visual Disorders in a Population-Based Cerebral Palsy Cohort. *Pediatric Neurology*, **50**, 324-328. <https://doi.org/10.1016/j.pediatrneurol.2013.11.022>

- [59] Wallace, S.J., Bottos, M. and Stanly, P. (2001) Epilepsy in Cerebral Palsy. *Developmental Medicine & Child Neurology*, **7**, 713-717.
- [60] Kwong, K.L., Wong, Y.C., Fong, C.M., et al. (2004) Magnetic Resonance Imaging in 122 Children with Spastic Cerebral Palsy. *Pediatric Neurology*, **31**, 172-176. <https://doi.org/10.1016/j.pediatrneurol.2004.02.005>
- [61] Spiniloa, A., Capuzzo, E., Orcesi, S., et al. (1997) Antenatal and Delivery Risk Factors Simultaneously Associated with Neonatal Death and Cerebral Palsy in Preterm Infants. *Early Human Development*, **48**, 81-91.
- [62] Okumara, A., Kato, T., Kuno, K., et al. (1997) MRI Findings in Patients with Spastic Cerebral Palsy. II: Correlation with Type of Cerebral Palsy. *Developmental Medicine & Child Neurology*, **39**, 369-372. <https://doi.org/10.1111/j.1469-8749.1997.tb07448.x>
- [63] Steinlin, M., Good, M., Martin, E., et al. (1993) Congenital Hemiplégia: Morphology of Cerebral Lesions and Pathogenetic Aspects from MRI. *Neuropediatrics*, **24**, 224-229. <https://doi.org/10.1055/s-2008-1071545>
- [64] Filipiti, P., Caldas, C. and Delpierre, Y. (2006) Spasticity Management and Progress in Ambulatory Cerebral Palsy. *Archives of Pediatrics*, **13**, 614-615.
- [65] Richards, C.L. and Malouin, F. (2013) Cerebral Palsy: Definition, Assessment and Rehabilitation. *Handbook of Clinical Neurology*, **111**, 183-195. <https://doi.org/10.1016/B978-0-444-52891-9.00018-X>
- [66] Brandenburg, J.E. (2013) Use of Rimabotulinum Toxin for Focal Hypertonicity Management in Children with Cerebral Palsy with Nonresponse to Onabotulinum Toxin. *American Journal of Physical Medicine & Rehabilitation*, **92**, 898-904. <https://doi.org/10.1097/PHM.0b013e31829231fa>
- [67] Graham, H.K., Aoki, K.R., Autti-Ramo, I., et al. (2000) Recommendations for the Use of Botulinum Toxin Type A in the Management of Cerebral Palsy. *Gait & Posture*, **11**, 67-79. [https://doi.org/10.1016/S0966-6362\(99\)00054-5](https://doi.org/10.1016/S0966-6362(99)00054-5)
- [68] Barber, L., Hastings-Ison, T. and Baker, R. (2013) The Effects of Botulinum Toxin Injection Frequency on Calf Muscle Growth in Young Children with Spastic Cerebral Palsy: A 12-Month Prospective Study. *Journal of Children's Orthopaedics*, **7**, 425-433.