RESEARCH PAPER

Paroxysmal Dyskinesia, an unusual but Particular Presentation of Congenital and Neonatal Hyperthyroidism: A Case Report with Literature Review

Georges Pius Kamsu Moyo[1], Suzanne SapNgo Um[2], Hubert Desire Mbassi Awa[3], Christelle Ngo Kana[4], Laura Kuate Makowa[5], Therese Audrey Mbang[6], Binda Virginie[7], Paul Olivier Koki Ndombo[8]

[1]Faculty of Medicine and Biomedical Sciences, the University of Yaounde I, Yaounde, Cameroon

ABSTRACT

Neonatal hyperthyroidism is a clinical entity almost always associated with maternal Grave's disease, which occurs in 0.2% of pregnant women. Paroxysmal myoclonic dyskinesiais an extremely rare presentation in neonatal hyperthyroidism, semiologically close to erratic movement disorders, of which very few exceptional cases have been described in adults with hyperthyroidism. In this paper we present a case of fetal and neonatal autoimmune hyperthyroidism in a 14-days old neonate infant with dyskinesia. This case was a typical in its clinical presentation marked with repeated persistent spontaneous and/or inducible myoclonus, but classical in its management as usual medication with methimazol and propanolol were used with favorable outcome.

Key words: Neonatal hyperthyroidism, Grave's Disease, Dyskinesia, Myoclonus.

BACKGROUND

Fetal and neonatal hyperthyroidism or thyrotoxicosis is predominated by auto-immune hyperthyroidism, also known as neonatal Grave's disease in some literatures [1]. It occurs in 1-5% neonates from women with active or past history of Grave's hyperthyroidism. Fetal and neonatal hyperthyroidism is associated with increased morbidity such as polymal formations and neuro developmental anomalies which may lead to considerable mortality if not diagnosed and properly treated [2]. Therefore, women with Grave's hyperthyroidism must be followed-up, their pregnancy duly planned and newborns automatically tested and managed. Neonatal Grave's disease occurs as a result of transplacental passage of Thyroid Stimulating Hormone (TSH) receptor-stimulating antibodies (TRab) to the fetus. This becomes effective as from the 17th to 20th weeks of gestation, with fetal TSH receptors becoming responsive [1, 3]. TRab produced in the mother, in addition with excess thyroid hormones from untreated or insufficiently balanced mothers cause fetal and neonatal hyperthyroidism [4]. The maternal transfer of thyroid auto antibodies may vary throughout the course of the pregnancy with marked increase in the beginning of the second trimester. "Neonatal hyperthyroidism of late onset" may occur as well in infants aged 2 to 3 months, due to the persistence of maternal antibodies remaining in the serum [5-7]. Maternal risk factors for fetal and neonatal autoimmune hyperthyroidism include: long-standing Grave's hyperthyroidism, late presentation to obstetric clinic, frequent infections, gestational hypertension, twin pregnancies, anemia, inconsistent use of medications, abnormal outcomes in prior pregnancies [8-11]. Uncontrolled maternal hyperthyroidism is associated with abortion, prematurity, low birth weight, intrauterine growth restriction, stillbirth, congenital malformations [11-13]. Observations from obstetrical ultrasonography studies have permitted to describe goiter,

Address for correspondence:
Georges Pius Kamsu Moyo, Faculty of Medicine and Biomedical Sciences, the University of Yaounde I, Yaounde, Cameroon.

DOI: 10.33309/2638-7654.040102

tachycardia, advanced bone age, oligo or polyhydramnios, heart failure and hydrops in fetuses with hyperthyroidism [14-17]. Whereas, in neonates, classical findings include: exophthalmos, craniosynostosis, microcephaly, intrauterine growth retardation, exaggerated Moro reflex, increase in other reflexes, goiter, hyperactivity, irritability, sleep disorder, inability to gain weight or weight loss despite excessive appetite, diarrhea, vomiting, fever, sweating, tachypnea, arrhythmia, hypertension, hypertensive encephalopathy, heart failure, pulmonary hypertension, chilothorax, cholestasis, hepatosplenomegaly, lymphadenopathy, hip dysplasia, prolonged acrocyanosis, and sialadenitis. Radiography may reveal an enlarged thymus and advanced bone age. Increase in the levels of transaminases AST, ALT and direct bilirubin, hypoglycemia, thrombocytopenia, and hyperviscosity have been reported as well. Most importantly, serum thyroxin (T4) and TRab are elevated while TSH is lowered. As a transient disease in neonates, the clinical manifestation of congenital autoimmune hyperthyroidism may subside within 3-16 weeks when maternal antibodies disappear [5,18,19]. The mainstay of the treatment regimen for neonatal hyperthyroidism is the antithyroid drug methimazol between 0.5–1 mg/kg/day to which the beta-blocker propranolol at 2 mg/kg/day is associated in severe cases [3]. Propylthiouracl may only be prescribed as second intention treatment at 0.2-0.5 mg/kg/day divided into three doses. This is due to hepatotoxicity, liver failure and death, particularly reported in children under this medication [20]. Suppression-replacement therapy with methimazol and levothyroxine, just as the use of glucocorticoids and iopanoic acid are rarely required. Patient Follow-up is necessary until hyperthyroidism resolves few months later on and can be discontinued with rising TSH and disappearing TRab. The child will then be controlled every 6 to 12 months to monitor developmental milestones, neurological and cardiovascular states, with eventual complications as well [3, 20].

CASE PRESENTATION

This is the case report of a 14 days old female neonate who was brought to consultation presenting with repeated spontaneous intermittent myoclonus of the upper limbs, evolving from the first day of life, with onset immediately after delivery. They were predominant during sleep without alleviating factor. They lasted for about 10 to 15 minutes in an afebrile context and were accompanied with sweating. This led to a first consultation in a health center where the baby was treated with neonatal meningitis protocol: Ampicillin, Gentamycin, Cefotaxim for 10 days. The neonate was also given phenobarbital and clonazepam at adequate doses for 2days. The evolution was marked with the persistence of the abnormal movements and the baby was then referred to our setting for better management.

Concerning past history, the baby was delivered at 39 weeks of Gestation (GA) of an uneventful pregnancy with 8 antenatal consultations. Iron, folic acid, tetanus and malaria prophylaxis were properly administered. The work-ups during pregnancy were unremarkable. There was no pregnancy-related pathology, however the mother had complained of discomfort with regular fetal active movements and fetal tachycardia sometimes noted according to the gynecologist-obstetrician report. The neonate was delivered through a eutocic per vaginal nuchal cord delivery with birth weight at 2800g. There was no notion of fever nor instrumental delivery. The baby's breathing was spontaneous at birth, meconium and urine emission followed within 24 hours, BCG and polio vaccines were administered the same day. The mother was 29 years old, dentist by profession and this was her second child. She was being followed-up for Basedow's disease (Grave's hyperthyroidism) lasting for over 8 months prior to the pregnancy. She had been on antithyroid drug methimazole during 4 months before being operated upon with partial thyroidectomy indicated for goiter. She was then prescribed levothyroxine at 100mg daily for post-operative hypothyroidism. Methimazol was discontinued in the meantime and pregnancy was planned in accordance with the gynecologist-obstetrician.Systemic enquiry revealed irritability, agitation, sweating, sleeplessness and intermittent diarrhea in the neonate.

On physical exam, the general state was normal but the baby was hyperactive. The weight was 3200g and the other anthropometric parameters were all normal for age. The temperature and the breathing rate were normal, but the heart rate was accelerated at 175 cycles/minutes. No dysmorphism or deformity was noted; neither was there a cutaneous sign nor signs of dehydration. The chest was symmetrical; there was tachycardia on cardiac auscultation, with a normal rhythm and without murmur. The pulmonary and abdominal exams were without peculiarities. The neurological exam featured explosive intermittent myoclonus of the upper limbs with slight trembling; exaggerated Moro reflex prolonged with myoclonus, increased deep tendon reflexes. The diagnosis of neonatal hyperthyroidism was posed and differentials such as meningitis, encephalitis, epilepsy, electrolyte imbalance were evoked.

Para clinical exams such as serum electrolytes were tested and showed mild hypokalemia, electrocardiogram (EKG), electroencephalogram (EEG), Full Blood Count (FBC), C - reactive protein (CRP), Cerebrospinal Fluid (CSF) analysis and transfontanellar ultrasound were done with normal results. Thyroid Stimulating Hormone (TSH) was decreased at 0.0274µu/ml, TSH receptor antibodies (TRab) was positive at 4.1 IU/l and blood thyroxin (T4) level was high at 25mU/l. This enabled to confirm the diagnosis of neonatal hyperthyroidism

and to rule out the differential diagnoses. The treatment comprised propranolol at 1 mg/kg per day and Methimazol at 1 mg/kg per day as well, but was to be introduced 2 days after the initiation of Propanolol.

The neonate was seen 2 weeks later on and 6 weeks thereafter with no more myoclonus, nor sweating. There was regression of hyperactivity and improvement of sleep. However, constipation was reported. TSH control at 8 weeks after the start of treatment was 25.4 µu/ml, Propanolol and methimazol were discontinued; TSH and TRab controls were to be done the 12th week.

DISCUSSION

Most studies conducted on neonatal hyperthyroidism indicate the onset of clinical manifestations as early as within the 1st to 3rd day of life. This is particularly the case inneonates from mothers with Grave's disease who are not on antithyroid drug medication (ATDs) such as methimazol or propylthiouracil. Whereas, an average age at onset within 7 to 17 days is described in neonates with mothers under this medication [21]. This is due to transplacental passage of antithyroid drugs from mothers onto fetuses, which inhibits TRab's action on thyroid glands as long as they persist in the blood system. It is responsible for a "pseudo-protection" in the early neonatal period, and eventually a late onset of the symptomatology in some babies. In the case reported, the onset of symptoms was quite early, immediately after delivery, given that the mother was not on antithyroid drugs. She had rather undergone partial thyroidectomy indicated for goiter, developed after being on ATDs alone for close to five months before the pregnancy. In effect, partial thyroidectomy may reduce overall TRab secretion in severe Grave's disease, but sometimes with residual activity sufficient enough to induce congenital and neonatal hyperthyroidism [22, 23]. Although no deformities occurred in our patient, it may be important to note teratogenicity as an adverse effect of methimazol during the first trimester of pregnancy, which justifies its replacement with propylthioural during this period. The inverse is true in neonates, due to hepatic and renal toxicity with propylthiouracyl [24, 25]. However, neonates from mothers with Grave's disease taking any of ATDs or not are at risk of hypothyroidism. This might either be transient iatrogenic fetal hypothyroidism due ATDs or induced central hypothyroidism due to elevated thyroid hormones in case the mother is untreated [26]. On the other hand, these neonates are prone to manifest hyperthyroidism later on in the neonatal period as well [24, 25].Therefore, there is need for keen multidisciplinary monitoring by the gynecologist-obstetrician, ENT surgeon and the pediatric endocrinologist throughout the perinatal period and even beyond [25-48].

Although we could not find evidence for preconceptional TRA belevation as a predictive factor for fetal and neonatal autoimmune hyperthyroidism in our patient (as recommended in the literature), cessation of ATDs was a non-negligible risk factor [23,49, 50]. The notions of fetal tachycardia as from the 5th month of pregnancy and the recurrence of early active fetal movements as described by the gynecologist-obstetrician might have accounted for fetal or congenital hyperthyroidism, coinciding with transplacental passage of TRab, inducingthe so-called "fetal thyroid responsiveness" [1,3].

Despite the fact that classical clinical signs and symptoms of neonatal hyperthyroidism such as irritability, restlessness, sleep disorders, sweating, trembling of limb extremities, diarrhea and tachycardia seemed obvious in our patient, the diagnosis was delayed. This was partly because of the non-systematization of neonatal TSH sampling in our setting, contrary to normal recommendations with Guthrie test. In effect the early sampling of TSH, coupled with history of maternal Grave's disease would have made the diagnosis easier and enabled a prompt management [3]. The second reason for the diagnosis delay was the atypical presentation with paroxysmal myoclonic dyskinesia, for which a neurological disease absolutely had to be ruled out, as well as neonatal sepsis with meningitis or meningo-encephalitis [51].

Paroxysmal myoclonic dyskinesiais defined as a movement disorder characterized by sudden repetitive jerks of considerable amplitudes, which predominated in the upper limbs in our patient. They were spontaneous and/or inducible by any funny stress including hunger, high temperature and sweating, Moro reflex just to name some. This presentation was atypical in the sense that the most commonly reported movement anomaly in neonatal hyperthyroidism is tremor [5, 19, and 25]. Even though our patient equally manifested tremors of limb extremities, they were however almost shadowed by myoclonus. Very few authors have also described paroxysmal kinesigenic dyskinesia with chorea and dystonia in adult patients manifesting hyperthyroidism [52, 53]. In reality, the pathophysiology of movement disorders in hyperthyroidism in general and Grave's disease in particular is still unclear, but the most admitted hypotheses suggest a direct effect of excess thyroid hormones rather than autoimmune dysfunction [52-60].

In facts, hyperthyroidismcan be responsible for reversible metabolic disturbances of the basal ganglia circuits. In patients exhibiting paroxysmal kinesigenic dyskinesia, decreased striatal glucose metabolism has been observed in most patients with symptomatic chorea [52-60].This is probably due to the increased rate of metabolism in hyperthyroidism, which causes excessive demand for glucose use and relative systemic shortages of the substrate. Contrasting with normal blood glucose or even hyperthyroidism-induced hyperglycemia. Nevertheless, hyperthyroidism may as well be responsible for beta-adrenergic stimulation which is incriminated as a mechanism leading to tremor and probably chorea. This other

hypothesis is further comforted by the fact that both tremor and chorea respond well to beta-blockers [52-60].It may be important to emphasize that the magnitude of neurological signs in Grave's disease seems to depend upon the rising concentration of thyroid hormones rather than thyroid autoantibodies.

The management of our patient consisted of Methimazol and propranolol which is the actual recommended therapy. The outcome was favorable with marked regression of dyskinesia and other hyperthyroidism signs and symptoms. Hormonal inversion was noted after 8 completed weeks of treatment, with rising TSH and constipation as a symptom of iatrogenic hypothyroidism. This was suggestive of significant drop or disappearance of TRab as described in the literature. This permitted us to discontinue methimazol and propranolol. Growth was normal, developmental milestones and neurological exams were normal as well, showing no major neonatal complication [52-60].

CONCLUSION

Neonatal hyperthyroidism is most often a complication of maternal Grave's disease, due to transplacental transmission of thyroid auto antibodies which are specific stimulators of TSH-receptors. In addition to classical presentations, exceptional cases may occur with fetal and neonatal neurological manifestations such as increased active fetal movements or paroxysmal myoclonic dyskinesia after birth. However neurological signs as complex as they may be, just as other signs of hyperthyroidism all subside with adequate management comprising ATDs and beta-blockers. Nevertheless, because of major congenital and postnatal complications including malformations, abortion and developmental anomalies, pregnancies in mothers with Grave's disease should be planed and regularly followed-up. Neonates should be automatically screened, treated and monitored up to 3 years of age for a favorable outcome.

ACKNOWLEDGEMENT

All collaborators to this project.

CONFLICT OF INTEREST

The authors declare that they have no competing interest.

REFERENCES

1. Cooper DS, Laurberg P. Hyperthyroidism in pregnancy. Lancet Diabetes Endocrinol. 2013;1:238–49.
2. Zimmerman D. Fetal and neonatal hyperthyroidism. Thyroid. 1999;9:727–33.
3. Alexander EK, Pearce EN, Brent GA, Brown RS, Chen H, Dosiou C, Grobman WA,Laurberg P, Lazarus JH, Mandel SJ, Peeters RP, Sullivan S. 2017 guidelines of theAmerican Thyroid Association for the diagnosis and Management of ThyroidDisease during Pregnancy and the postpartum. Thyroid. 2017;27:315–89.
4. Levy-Shraga Y, Tamir-Hostovsky L, Boyko V, Lerner-Geva L,Pinhas-Hamiel O. Follow-up of newborns of mothers withGraves' disease. Thyroid 2014; 24: 1032-9.
5. Segni M, Leonardi E, Mazzoncini B, Pucarelli I, Pasquino AM. Special features of Graves' disease in early childhood.Thyroid 1999; 9: 871-7.
6. Smith C, Thomsett M, Choong C, Rodda C, McIntyre HD,Cotterill AM. Congenital thyrotoxicosis in premature infants.Clin Endocrinol (Oxf) 2001; 54: 371-6.
7. Zakarija M, McKenzie JM. Pregnancy-associated changesin the thyroid-stimulating antibody of Graves' diseaseand the relationship to neonatal hyperthyroidism. J ClinEndocrinol Metab 1983; 57: 1036-40.
8. Davis LE, Lucas MJ, Hankins GD, Roark ML, Cunningham FG. Thyrotoxicosiscomplicating pregnancy. Am J Obstet Gynecol. 1989;160:63–70.
9. Mestman JH. Hyperthyroidism in pregnancy. Best Pract Res Clin EndocrinolMetab. 2004;18:267–88.
10. Laurberg P, Bournaud C, Karmisholt J, Orgiazzi J. Management of Graves'hyperthyroidism in pregnancy: focus on both maternal and foetal thyroidfunction, and caution against surgical thyroidectomy in pregnancy. Eur JEndocrinol. 2009;160:1–8.
11. Millar LK, Wing DA, Leung AS, Koonings PP, Montoro MN, Mestman JH. Lowbirth weight and preeclampsia in pregnancies complicated by hyperthyroidism.Obstet Gynecol. 1994;84:946–9.
12. Hamburger JI. Diagnosis and management of Graves' disease in pregnancy.Thyroid. 1992;2:219–24.
13. Abeillon-du Payrat J, Chikh K, Bossard N, Bretones P, Gaucherand P, Claris O,Charrie A, Raverot V, Orgiazzi J, Borson-Chazot F, Bournaud C. Predictivevalue of maternal second-generation thyroid-binding inhibitory immunoglobulinassay for neonatal autoimmune hyperthyroidism. Eur J Endocrinol.2014;171:451–60.
14. Polak M, Van Vliet G. Therapeutic approach of fetal thyroid disorders. HormRes Paediatr. 2010;74:1–5.
15. King JR, Lachica R, Lee RH, Montoro M, Mestman J. Diagnosis andManagement of Hyperthyroidism in pregnancy: a review. Obstet GynecolSurv. 2016;71:675–85.
16. Gruner C, Kollert A, Wildt L, Dorr HG, Beinder E, Lang N. Intrauterinetreatment of fetal goitrous hypothyroidism controlled by determination ofthyroid-stimulating hormone in fetal serum. A case report and review of theliterature. Fetal Diagn Ther. 2001;16:47–51.
17. American College of O, Gynecologists. Practice Bulletin No. 148: Thyroiddisease in pregnancy. Obstet Gynecol. 2015;125:996–1005.
18. Laurberg P, Wallin G, Tallstedt L, Abraham-Nordling M,Lundell G, Tørring O. TSH-receptor autoimmunity inGraves' disease after therapy with anti-thyroid, surgery,or radioiodine: a 5-year prospective randomized study.Eur J Endocrinol 2008; 158: 69-75.

19. Goldstein I, Lockwood C, Belanger K, Hobbins J. Ultrasonographicassessment ofgestational age with the distal femoraland proximal tibial ossification centers in the third trimester.Am J Obstet Gynecol 1988; 158: 127-30.
20. Rivkees SA, Mattison DR. Propylthiouracil (PTU) Hepatoxicity in children andrecommendations for discontinuation of use. Int J Pediatr Endocrinol.2009;2009:132041.
21. Papendieck P, Chiesa A, Prieto L, Gruñeiro-Papendieck L.Thyroid disorders of neonates born to mothers with Graves'disease. J Pediatr Endocrinol Metab 2009; 22: 547-53.
22. Kautbally S, Alexopoulou O, Daumerie C, Jamar F, Mourad M, Maiter D.Greater efficacy of Total thyroidectomy versus radioiodine therapy onhyperthyroidism and thyroid-stimulating immunoglobulin levels in patientswith Graves' disease previously treated with Antithyroid drugs. Eur Thyroid J.2012;1:122–8.
23. Laurberg P, Wallin G, Tallstedt L, Abraham-Nordling M, Lundell G, Torring O.TSH-receptor autoimmunity in Graves' disease after therapy with anti-thyroiddrugs, surgery, or radioiodine: a 5-year prospective randomized study. Eur JEndocrinol. 2008;158:69–75.
24. Smith CM, Gavranich J, Cotterill A, Rodda CP. Congenitalneonatal thyrotoxicosis and previous maternal radioiodinetherapy. BMJ 2000; 320: 1260-1.
25. Laurberg P, Wallin G, Tallstedt L, Abraham-Nordling M,Lundell G, Tørring O. TSH-receptor autoimmunity inGraves' disease after therapy with anti-thyroid, surgery,or radioiodine: a 5-year prospective randomized study.Eur J Endocrinol 2008; 158: 69-75.
26. Chiabi A,Kago DA, Moyo GPK, Obadeyi B. "Relevance and Applicability of the Apgar Score in Current Clinical Practice". EC Paediatrics 2019;8(11):01-07.
27. Moyo GPK, Tetsiguia JRM. Discussing the "First Cry" as an Initial Assessment for Neonates. Am J of Pediatr. 2020;6(2): 129-132. doi: 10.11648/j.ajp.20200602.22
28. Moyo GPK, Sobguemezing D, Adjifack HT. Neonatal Emergencies in Full-term Infants: A Seasonal Description in a Pediatric Referral Hospital of Yaoundé, Cameroon. Am J Pediatr. 2020;6(2):87-90. doi: 10.11648/j.ajp.20200602.13
29. Moyo GPK, Um SSN, Awa HDM, et al. The pathophysiology of neonatal jaundice in urosepsis is complex with mixed bilirubin!!! *J Pediatr Neonatal Care.* 2022;12(2):68–70. DOI: 10.15406/jpnc.2022.12.00458
30. Moyo GPK, Nguedjam M, Miaffo L. Necrotizing Enterocolitis Complicating Sepsis in a Late Preterm Cameroonian Infant. *Am J Pediatr*. 2020;6(2):83-86. doi: 10.11648/j.ajp.20200602.12
31. Moyo GPK, Sap Ngo Um S, Awa HDM, Mbang TA, Virginie B, Makowa LK et al. An Atypical Case of Congenital and Neonatal Grave's Disease. Annal Cas Rep Rev: ACRR-314. 2022;2. doi: 10.39127/2575-8253/IAPN:1000114
32. Ngwanou DH, Ngantchet E, Moyo GPK.Prune-Belly syndrome, a rare case presentation in neonatology: about one case in Yaounde, Cameroon.Pan Afr Med J. 2020;36:120.doi: 10.11604/pamj.2020.36.102.24062
33. Tague DAT, Evelyn Mah, Félicitee Nguefack, Moyo GPK, Tcheyanou LLK, Faustin Mouafo. Beckwith-Wiedemann Syndrome: A Case Report at the Gynaeco-Obstetric and Pediatric Hospital in Yaounde, Cameroon. Am J Pediatr 2020;6(4):433-436. doi: 10.11648/j.ajp.20200604.17
34. Hermann ND. Moyo GPK. Neonatal Determinants of Inadequate Breastfeeding: A Survey among a Group of Neonate Infants in Yaounde, Cameroon. Open Access Library Journal (2020); 7: e6541. DOI: 10.4236/oalib.1106541
35. Hermann ND, Moyo GPK, Ejake L, Félicitée N, Evelyn M, Linda M. Determinants of Breastfeeding Initiation Among Newly Delivered Women in Yaounde, Cameroon: a Cross-Sectional Survey Health Sci Dis 2020; 21:20-24. Doi: https://www.hsd-fmsb.org/index.php/hsd/article/view/2265
36. Moyo GPK, Dany Hermann ND. Clinical Characteristics of a Group of Cameroonian Neonates with Delayed Breastfeeding Initiation. Am J Pediatr 2020;6(3):292-295. doi: 10.11648/j.ajp.20200603.28
37. Moyo GPK, Mendomo RM, Batibonack C, Thérese Mbang AT. Neonatal Determinants of Mothers' Affective Involvement in Newly Delivered Cameroonian Women. Journal of Family Medicine and Health Care 2020;6(2):125-128. doi: 10.11648/j.ajp.20200602.21
38. Moyo GPK. Perinatality and Childbirth as a Factor of Decompensation of Mental Illness: The Case of Depressive States in Newly Delivered Cameroonian Women ABEB 2020: 4(4). ID.000592. DOI: 10.33552/ABEB.2020.04.000592
39. Moyo GPK, Hermann ND. The Psycho-Sociocultural Considerations of Breastfeeding in a Group of Cameroonian Women with Inadequate Practices.J Psychiatry Psychiatric Disord 2020; 4 (3): 130-138.doi: 10.26502/jppd.2572-519X0100
40. Moyo GPK, Djoda N. Relationship Between the Baby Blues and Postpartum Depression: A Study Among Cameroonian Women. American Journal of Psychiatry and Neuroscience 2020; 8(1): 26-29. Doi: 10.11648/j.ajpn.20200801.16
41. Moyo GPK, Ngwanou DH, Sap SNU, Nguefack F, Mah EM. The Pattern of Breastfeeding among a Group of Neonates in Yaoundé, Cameroon.International Journal of Progressive Sciences and Technologies 2020;22(1):61-66. DOI: 10.52155
42. Nguefack F, Ngwanou DH, Kamsu Moyo GP, Ejake L, Mah, EM, Wafeu G, Fodoung Wamba DS, Chiabi A. Reasons for Delayed Breastfeeding Initiation among Newly Delivered Women in Two First-Category Hospitals in Yaoundé, Cameroon. Open J Pediatr 2020; 10:474-485. https://doi.org/10.4236/ojped.2020.103048
43. Moyo GPK, Hermann ND (2020) Caesarean Delivery as a Predictor of Inadequate Breastfeeding among a Group of Neonates in Yaoundé, Cameroon. J Perina Clin Pediatr: 2: 105. DOI: 10.29011/JPCP-105.100005
44. Moyo GPK. Epidemio-clinical Profile of the Baby Blues in Cameroonian Women. Journal of Family Medicine and Health Care. 2020;6(1):20-23. doi: 10.11648/j.jfmhc.20200601.14
45. Moyo GPK, Djoda N. The Emotional Impact of Mode of Delivery in Cameroonian Mothers: Comparing Vaginal

Delivery and Caesarean Section. American Journal of Psychiatry and Neuroscience. 2020;8(1): 22-25. doi: 10.11648/j.ajpn.20200801.15

46. Foumane P, Olen JPK, Fouedjio JH, GPK Moyo, Nsahlai C,Mboudou E. Int J Reprod Contracept Obstet Gynecol. 2016 Dec;5(12):4424-4427.DOI: http://dx.doi.org/10.18203/2320-1770.ijrcog20164357
47. Moyo GPK, Djomkam IFK. Factors Associated with Stunting in School Children of an Urban Community in Cameroon. Am J Pediatr. 2020;6(2):121-124. doi: 10.11648/j.ajp.20200602.20.
48. Moyo GPK, Djomkam IFK. Epidemio-clinical Profile of Stunting in School Children of an Urban Community in Cameroon. Am J Pediatr 2020;6(2): 94-97. doi: 10.11648/j.ajp.20200602.15
49. Mestman JH. Hyperthyroidism in pregnancy. Curr Opin Endocrinol Diabetes Obes. 2012;19:394–401.
50. Hesarghatta Shyamasunder A, Abraham P. Measuring TSH receptor antibodyto influence treatment choices in Graves' disease. Clin Endocrinol. 2017;86:652–7.
51. Georges Pius K M, Aurore Albane E, Marie-Paul B, et al. Neonatal Sepsis: Highlights and Controversies. J Pediatr Neonatal. 2022; 4(1): 1-5. Doi: 10.33425/2689-1085.1035
52. Delhasse S, Debove I,Arnold-Kunz G, Ghika J, Chabwine JN. Erratic movement disordersdisclosing Graves' disease andparalleling thyroid functionbut not autoantibody levels.J Int Me Res 2019; 47(3): 1378–1386.
53. Puri V, Chaudhry N. Paroxysmal kinesigenic dyskinesia manifestation ofhyperthyroidism.Neurol India 2004;52(1).
54. Seeherunvong T, Diamantopoulos S, Berkovitz G. A. Nine-year-old girl withthyrotoxicosis, ataxia, and chorea. BrainDev 2007; 29: 660–661.
55. Yu J-H and Weng Y-M. Acute chorea as apresentation of Grave disease: case reportand review. Am J Emerg Med 2009; 27:369.e361–369.e363.
56. Nagaoka T, Matsushita S, Nagai Y, Kobayashi K. A woman who trembled, then had chorea. Lancet 1998; 351: 1326.
57. Loh L, Hum A, Teoh H, Lim E. Grave'sdisease associated with spasmodic truncalflexion. Parkinsonism Relat Disord 2005;11: 117–119.
58. Moyo GPK, Sap Ngo Um S, Awa HDM, Mbang TA, Virginie B, et al. An Atypical Case of Congenital and Neonatal Grave's Disease. Annal Cas Rep Rev 2022: ACRR-314.doi: 10.39127/2575-8253/IAPN:1000114
59. Sudo K and Tashiro K. Hyperthyroidismassociated chorea. Lancet 1998; 352: 239.
60. Kung AW. Neuromuscular complications of thyrotoxicosis. Clin Endocrinol (Oxf) 2007; 67: 645–650.

How to cite this article: Moyo G P K, Um S S, Awa H B M, Kana C N, Makowa L K, Mbang T A, Virginie B, Ndombo P O K. Paroxysmal Dyskinesia, an unusual but Particular Presentation of Congenital and Neonatal Hyperthyroidism: A Case Report with Literature Review. Clin Res Pediatr 2022;4(1):06-11.
DOI: 10.33309/2638-7654.040102