

# Immune Thrombocytopenic Purpura of Children in Black Africa: Two New Observations in Togo

Gbadoé AD<sup>1,2</sup>, Guédéhoussou T<sup>1\*</sup>, Dossou CF<sup>1</sup>, Guédénou KM<sup>1</sup> and Fiawoo M<sup>1</sup>

<sup>1</sup>Onco-Hematology Unit, CHU SO, 198 Hospital Street, Tokoin District B.P. 57 Lomé, Togo

<sup>2</sup>BeHospital, La Chapelle Street, Bè Pa de Souza, Lomé, Togo

## \*Corresponding author

Guédéhoussou Têtè, Senior Assistant Professor, CHU SO, Pediatrics Department, Lomé 198 Hospital Street, Tokoin District PO Box 57/7319, Lomé (Togo), Tel: (228) 22 21 25 01/90 05 17 99 99 53 34 17; E-mail: tetegued@hotmail.com./ tetegued@gmail.com

Submitted: 09 July 2018; Accepted: 16 July 2018; Published: 18 Aug 2018

## Abstract

**Introduction:** Immune thrombocytopenic purpura (ITP) is an auto immunedisorder that is very common in Europe and the USA but is poorly described in black Africa.

**Observations:** K.A., boy of 5 years 7 months was admitted in 2012 for post-varicella acute ITP with a hemorrhagic syndrome associated with severe isolated thrombocytopenia at 7,000 platelets / mm<sup>3</sup>.

S.D. is a boy of 3 years 8 months admitted in 2015 for ITP which history is marked by a delay in diagnosis despite several consultations. In both cases, complete remission was obtained under prednisone.

**Discussion:** Recently, the rarity of ITP has also been highlighted in the USA in the black race. The probable reasons given were genetic factors, a sub-diagnosis due to the discrimination of the blacks in the accessibility to healthcare, a relative discretion of the petechi as which can go unnoticed on black skin, but also the ignorance of the affection by the practitioners in Togo.

**Conclusion:** ITP is poorly known and poorly reported in Black Africa. The establishment of an ITP registry may increase its knowledge and impact.

**Keywords:** Immune Thrombocytopenic Purpura, Idiopathic thrombocytopenic Purpura, Black Africa, Child.

## Introduction

Immune thrombocytopenic purpura (ITP) is a primary hemostatic disorder characterized by isolated thrombocytopenia [1, 2]. This thrombocytopenia is due to peripheral destruction of platelets at the spleen resulting from an auto immune mediated autoantibody (AAB) process. It is also due to a lack of medullary production of immunological origin [1, 2]. ITP is a benign condition with favorable evolution most of the time. Cerebro-meningeal haemorrhage, its dreaded complication, rarely occurs (0.1-1% of cases), but is of extreme severity (mortality between 24 and 57%) [3-9]. In Europe and the USA, several cases have been described for several years with an estimated incidence of 1-12 cases per 100,000 children per year [10-12]. In 1998, we reported the rarity of this condition in black Africa by describing an isolated case in Togo [13]. Subsequently, Salawu et al in 2001 pointed out the same rarity in Nigeria, reporting 11 cases in 11 years [14]. Since that publication, no other case has been reported in black in Africa. This work describes two new cases in Togo and raises the question of the scarcity of cases in Black Africa.

## Observation

### Observation n° 1

K.A. was a boy of 5 years 7 months, born 3/4/07, admitted on 6/11/12 for hematuria and conjunctival haemorrhage. Two weeks prior to admission, K.A. presented with a simple evolution chickenpox. The occurrence of conjunctival haemorrhage and hematuria motivated a blood count that revealed severe thrombocytopenia at 7000 platelets / mm<sup>3</sup>, resulting in hospitalization in pediatric hematology for better management.

K. A. was born of a father and mother who are both teachers. He was the second of two siblings, with no known pathological antecedents.

He weighed 20 kg, measured 106 cm for a BMI of 19.66 kg / m<sup>2</sup>. He had a normal nutritional status, a preserved general state, moderate palmo-plantar pallor without jaundice, and an hemorrhagic syndrome made of:

- Multiple petechiae all over the body;
- Conjunctiva hemorrhage;
- Hemorrhagic blebs without active bleeding in the oral cavity;
- Minimal hematuria (traces of blood in the urine)

Buchanan's score was evaluated at 3.

The rest of the somatic examination was normal with no spleenomegaly and lymphadenopathy. The hemogram showed the following result: hemoglobin level at 8.4 g / dl; red blood cells at 3.46. 106 / mm<sup>3</sup>; hematocrit at 30.8%; VGM at 89 fl; TCMH at 30 µg; white blood cells at 19,000 / mm<sup>3</sup> with 36% neutrophils, 63% lymphocytes, 1% monocyte; platelets with 7000 platelets / mm<sup>3</sup>.

Given this picture of hemorrhagic syndrome and isolated thrombocytopenia, the diagnosis of acute ITP was suspected and confirmed by a myelogram showing in the marrow with very many megakaryocytes present at all stages of maturation. The erythroblast line was slightly increased but there were no cytological abnormalities. The granular line was relatively uninhibited and without morphological abnormalities. The non-myeloid lineage was qualitatively without abnormalities. Intra-lineage balance was respected in all lineages. The ratio of erythroblasts to granular was disrupted in favor of erythroblasts.

The other para clinical assessments carried out gave the following results:

- Negative HIV serology, Negative hepatitis B serology,
- ECBU: absence of biological signs of urinary tract infection,
- Fund of eye without abnormalities apart from conjunctival haemorrhage,
- Assessment of normal hemostats is outside an elongated bleeding time.

As a treatment, K.A. benefited from a transfusion of two pockets of platelet concentrates; deformed with albendazole suspension for 3 days; oral and external corticosteroid therapy based on prednisone according to the Blanc et al [30] regimen: 4 mg / kg / day the first week; 2 mg / kg / day the 2nd week and 1 mg / kg / day the 3rd week, in the middle of meals.

The evolution was marked by a gradual normalization of platelets at the end of the first week, with a maximum of 800,000 platelets / mm<sup>3</sup> obtained at the end of corticosteroid therapy. Control one month later showed a platelet count of 400,000 / mm<sup>3</sup>.

In December 2015, K.A. was doing well, with a normal platelet count.

### Observation n° 2

S.D. was a boy of 3 years 8 months, born on 15/09/11, admitted on 15/05/15 for petechiae on the body and gingivorrhagia. Its history went back to 2 years marked by several episodes of petechiae and several platelet counts ranging between 15 000 and 25 000 / mm<sup>3</sup> without the diagnosis being evoked and he had an adequate treatment administered. He was born to literate parents, engineer father and hairdresser mother. He was the third of three siblings with no known pathological antecedents.

S.D. weighed 15 kg, measured 99 cm for a BMI at 15.30 kg / m<sup>2</sup>. His nutritional status was therefore normal. On admission, S.D. had scalp pustulosis and hemorrhagic syndrome consisting of petechiae all over the body, middle grade epistaxis, intermittent gingivorrhage, and minimal hematuria (traces of blood in the urine). Buchanan's score was evaluated at 3.

The remainder of the somatic examination was normal without splenomegaly and without peripheral adenopathies.

The assessment done on admission noted the following results:

- **Hemogram:**  
Hb level at 12 g / dl; hematocrit at 38.8%; VGM at 84.6%; TCMH at 26.1 µg; Red blood cells at 4.59.106 / mm<sup>3</sup>; white blood cells at 11,600 GB / mm<sup>3</sup> with 57% neutrophils, 38% lymphocytes, 5% monocytes; thrombocytopenia at 23,000 platelets / mm<sup>3</sup>.
- **Plasmodium falciparum negative RDT; Hemoglobin AS; HIV negative serology.**
- Faced with this hemorrhagic syndrome and this isolated thrombocytopenia evolving for 2 years, a chronic ITP was evoked.

Eight days after admission and antibiotic treatment against pustulosis, prednisone corticosteroid therapy according to the pattern of Blanchette et al, was initiated after 3 days of albendazole worming: 4 mg / kg / day the 1st week; 2 mg / kg / day the 2nd week and 1 mg / kg / day the 3rd week in the middle of meals [15].

The evolution was marked by a progressive increase in platelet count of 206 250 / mm<sup>3</sup> at the end of corticosteroid therapy. Three months later, the platelet count was 414,000 platelets / mm<sup>3</sup>.

Six months later, S.D. was fine, with a normal platelet count.

### Discussion

Frequency of the ITP

In Europe and the USA, there are thousands of cases. The annual incidence of ITP in children is estimated at between 1 and 12 cases / 100,000 children per year [10-12].

In Asia, the incidence is not well known, but many cases have been published in India and Pakistan. Ali et al estimated the incidence of ITP at 4.8% in the hematology-oncology unit of the Lahore Children's Hospital in Pakistan between 2003 and 2004 [16].

In Africa in the Maghreb, the incidence is not well known, but several articles have been published on the issue. In Tunisia S faihi L et al recruited 140 cases in 15 years (1995 to 2009) at the Hedi Chaker Teaching Hospital of Sfax in pediatric and hematology departments [17]. In Egypt, Elalfy et al recruited 1840 patients in 10 years (1997 to 2007) in 5 units of pediatric hematology.

In black Africa, publications are rare. In Nigeria, Salawu et al reported 11 cases hospitalized in 11 years at Ife Teaching Hospital between 1990 and 2001 [14]. In Togo, in 20 years (1995-2015), these are the 2nd and 3rd cases that we describe in this work [13, 18].

Is ITP really rare in black? And why would it be?

The hypothesis of the rarity of the ITP in the black race was reinforced by the work of Terrell et al in the USA in 2005 [19]. These authors found the difference in incidence between black and white populations by opening the ITP registry in Oklahoma [19]. They then undertook a review of the literature and found only 6 articles mentioning the disease among black Americans [20-25]. In these 6 articles, the incidence of ITP was significantly lower among blacks compared to Caucasians, taking into account the proportion of these two people in the general population.

In 1998, the hypotheses that we put forward to explain this rarity of the ITP among the blacks were the possibility of a genetic determinant or simply an under diagnosis of the cases due to the fact that petechiae could pass unnoticed for a long time on black skin. Salawu et al also mentioned these two hypotheses [14]. For Terrell et al, the third explanation of this scarcity of cases in black is a sub diagnosis due to the discrimination between black and white in accessibility to healthcare in United States [19]. The patient's history of our 2nd observation clearly showed a lack of awareness of the condition by practitioners who saw the child for 2 years without mentioning the diagnosis nor administering an adequate treatment.

In conclusion, we believe that ITP is probably not as rare as it is believed in Black Africa, but rather under-diagnosed because of its usual benignity, the difficulty of seeing petechiae on black skin, and the ignorance of the affection by practitioners.

### Treatment

Classically, in case of moderate thrombocytopenia greater than 30 000 / mm<sup>3</sup>, the therapeutically attitude is the therapeutic abstention with clinical and biological monitoring when the disease is asymptomatic [2, 26]. When treatment is decided for, in developed countries, the first-line treatment remains polyvalent immunoglobulin because of the short duration of treatment (one to four days according to the patterns) [2]. Our choice of corticosteroids as a first-line treatment is a reasoned choice due to its lower cost. Immunoglobulin IV, although sometimes available in black Africa, is too expensive. Other therapeutic alternatives are expensive and are not available: thrombopoietin receptor agonists (Romiplostim), Rituximab.

### Conclusion

ITP is a condition for which diagnosis and management remain within the reach of practitioners in Black Africa. And yet, cases are rarely reported. The rarity of this condition, recently demonstrated in the United States where black and white populations live together, is due to the discrimination of blacks to access to healthcare, less visible petechiae on black skin, and perhaps to genetic determinants. The ignorance of the affection by the practitioners would also explain the scarcity of the cases in Togo and probably in several countries of Black Africa. The creation of a national, or even African, register of the ITP would, among other things, help to increase its knowledge and its impact.

### References

1. Rodeghiero F, Stasi R, Gernsheimer T, Michel M, Provan D, et al. (2009) Standardization of terminology, definitions and outcome criteria in immune thrombocytopenic purpura of adults and children: report from an international working group. *Blood* 113: 2386-2393.
2. Haute Autorité de Santé (2009) Protocole National de Diagnostic et de Soins (PNDS) pour Purpura thrombopénique immunologique de l'enfant et de l'adulte. Saint-Denis la Plaine: Haute Autorité de Santé, Consultable sur : <http://www.has-sante.fr>.
3. Neunert CE, Buchanan GR, Imbach P, Bolton-Maggs PHB, Bennett CM, et al. (2013) Bleedings manifestations and management of children with persistent and chronic Immune thrombocytopenia: data from the Intercontinental Cooperative ITP Study Group (ICIS). *Blood* 121: 4457-4462.
4. Psaila B, Petrovic A, Page LK, Menell J, Schonholz M, et al. (2009) Intracranial hemorrhage (ICH) in children with Immune

- thrombocytopenia (ITP): study of 40 cases. *Blood* 114: 4777-4783.
5. Lyori H, Bessho F, Ookawa H, Konishi S, Shirahata A, et al. (2000) Intracranial hemorrhage in children with immune thrombocytopenic purpura. Japanese study group on childhood ITP. *Ann Hematol* 79: 691-695.
6. Tourneux P, Jouvencel P, Mischeau M, Jouvencel AC, Chateil JF, et al. (2005) Purpura thrombopénique à Parvovirus B19 compliqué d'hémorragie cérébro-méningée. *Archpediatr* 12: 281-283.
7. Heitink-Pollé KMJ, Pot DJ, Engelkes M, Bruin MCA (2011) Intracranial hemorrhage after high-dose Methylprednisolone in a child with acute immune thrombocytopenic purpura. *Ann Hematol* 90: 1361-1363.
8. George JN, Woolf SH, Raskob GE, Washer JS, Aledort LM, et al. (1996) Idiopathic thrombocytopenic purpura: A practice guideline developed by explicit methods for the American Society of Hematology. *Blood* 88: 3-40.
9. Neunert C, Lim W, Crowther M, Cohen A, Solberg LJR, et al. (2011) The American Society of Hematology 2011 evidence-based practice guideline for immune thrombocytopenia. *Blood* 117: 4190-4207.
10. Mattia DD, Principe D, Vicchio GCD, Jankovic M, Arrighini A, et al. (2000) Acute childhood idiopathic thrombocytopenic purpura: AIEOP consensus guidelines for diagnosis and treatment. *Haematologica* 85: 420-424.
11. Lilleyman JS. (1999) Management of childhood idiopathic thrombocytopenic purpura. *British journal of haematology* 105: 871-875.
12. George JN, El-Harake MA, Aster RH (1995) Thrombocytopenia due to enhanced platelet destruction by immer BS, Kipps TJ (eds): *Williams Hematology*. New York, NY, McGraw-Hill, 1315.
13. Gbadoe AD, Messie K, Atakouma DY, Vovor A, Tatagan-Agbi K, et al. (1998) Le purpura thrombopénique idiopathique de l'enfant en Afrique Noire : à propos d'un cas au Togo. *Cahier de santé* 8: 337-341.
14. Salawu L, Durosini MA (2001) Immune Thrombocytopenic purpura: 11-year experience in Ile-Ife, Nigeria. *African J Med Sci* 30: 99-103.
15. Blanchette V, Imbach P, Andrew M (1994) Randomized trial of intravenous Immunoglobulin G, intravenous anti-D and oral prednisone in Childhood acute immune thrombocytopenic purpura. *Lancet* 344: 703-706.
16. Ali AS (2005) Idiopathic thrombocytopenic Purpura- Initial illness subsequent behavior. *Pakistan Pediatric Journal* 29: 93-99.
17. Sfaihi L, Kassar O, Medhaffar M, Kamoun T, Hadji S, et al. (2014) Thrombocytopenie immune primaire de l'enfant : Etude régionale dans le sud tunisien. *La Tunisie Médicale* 92 : 219-223.
18. Blot I, Nicolas E, Kessie K, Gnamey K, Assimadi K, et al. (1982) Les syndromes hémorragiques cutanéomuqueux de l'enfant. A propos de deux observations. *Revue de médecine biologique de l'enfant* 3: 17-21.
19. Terrell DR, George JN, Veseley SK, Johnson KK (2005) Is immune thrombocytopenic purpura less common among black Americans? *Blood* 105: 1368-1369.
20. Block GE, Evans R, Zajtcuk R (1966) Splenectomy for idiopathic thrombocytopenic purpura. *Arch Surg* 92: 484-489.
21. Wilde RC, Ellis LD, Cooper WM (1967) Splenectomy for

- 
- chronic idiopathic thrombocytopenic purpura. Arch Surg 95: 344-350.
22. Akwari OE, Itani KMF, Coleman RE, Rosse WF (1987) Splenectomy for primary and recurrent immune thrombocytopenic purpura. Ann Surg 206: 529-541.
  23. Guthrie TH, Brannan DP, Prisant LM (1988) Idiopathic thrombocytopenic purpura in the older adult patient. Amer J Med Sci 296: 17-21.
  24. George JN, Raskob GE, Vesely SK (2003) Initial management of immune thrombocytopenic purpura in adults: a randomized controlled trial comparing intermittent anti-D with routine care. Amer J Hematol 74: 161-169.
  25. Aledort L, Hayward CPM, Chen M-G, Nichol J, Bussel J, et al. (2004) Prospective screening of 205 patients with ITP, including diagnosis, serological markers, and the relationship between platelet counts, endogenous thrombopoietin, and circulating antithrombopoietin antibodies. Amer J Hematol 76: 205-213.
  26. Rehman A (2009) Immune thrombocytopenia in children with reference to low- income countries. Eastern Mediterranean Health Journal 15: 729-737.

**Copyright:** ©2018 Guédéhoussou Têtê, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.