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Médecine et maladies infectieuses

Médecine et maladies infectieuses 42 (2012) 585–590

Original article

Predictive value of the eosinophil counts in the biological diagnosis of lymphatic filariasis in French Polynesia

Valeur prédictive de l'éosinophilie dans le diagnostic biologique de la filariose lymphatique en Polynésie française

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Received 21 March 2012; received in revised form 20 July 2012; accepted 25 September 2012
Available online 29 October 2012

Abstract

Settings. – Lymphatic filariasis is common in many tropical and subtropical areas and is a major public health issue in south Pacific islands. In endemic areas, most infected individuals are asymptomatic but may harbor microfilariae or filarial antigens in their peripheral blood. Microscopy remains the reference diagnostic tool for the identification of microfilariae but is weakly sensitive. The diagnosis of *Wuchereria bancrofti* infection was dramatically altered by the development of filarial antigen tests, which are easy to perform but expensive for routine use. Lymphatic filariasis is responsible for acquired eosinophilia and blood eosinophil count is commonly used as a screening tool in endemic areas.

Method. – We retrospectively analyzed all the results of eosinophil counts, antigen and microfilariae detection performed in our laboratory over a 24-month period. We calculated the prevalence of antigenemia for various eosinophilic cut offs.

Results. – The prevalence of antigenemia was estimated at 25.78% with eosinophilia defined as a count eosinophilic PMN above 500 per mm^3.

Discussion. – Our prevention strategy against lymphatic filariasis is based on annual mass drug administration, vector control, and systematic treatment of antigenemic and microfilaremic patients. Antigenemic and microfilaremic detection cannot be routinely performed because of their cost. Current treatments used for lymphatic filariasis are safe and cheaper than antigenic detection. A possible additional strategy to decrease the prevalence of antigenemia would be the systematic treatment of patients with hypereosinophilia.

Keywords: Eosinophilia; Filariasis; Microfilariae; *Wuchereria bancrofti*

Résumé

Cadre. – La filariose lymphatique est fréquente dans les régions tropicales et subtropicales et constitue un problème majeur de santé publique dans les états insulaires du Pacifique sud. La plupart des sujets infectés sont asymptomatiques mais peuvent être porteurs de microfilaires ou d'antigènes filariens dans leur sang périphérique. L'identification des microfilaires en microcopie reste l'examen diagnostique de référence mais est peu sensible. Le diagnostic a été révolutionné par la mise au point des tests de dépistage antigénique qui sont faciles à réaliser mais dont l'utilisation en routine est limitée par le coût. La filariose lymphatique est responsable d'une hyperéosinophilie secondaire qui est un argument biologique d'orientation en zone d'endémie.

Méthode. – Nous avons analysé rétrospectivement toutes les numérations formules sanguines et toutes les recherches d'antigènes filariens et de microfilaires réalisées sur une période de deux ans dans notre laboratoire.

Résultats. – Au-delà d'une valeur seuil de 500 polynucléaires éosinophiles/mm^3, la prévalence de l'antigénémie a été évaluée à 25,78 %.

Discussion. – Notre stratégie de lutte contre la filariose lymphatique est basée sur les campagnes annuelles de distribution de masse de molécules antifilariennes, la lutte antivectorielle et le traitement des patients antigénémiques et microfilarémiques. La recherche d'une antigénémie et d'une microfilarémie ne peut être réalisée en routine en raison de leurs coûts. Les traitements antifilariens présentent très peu d'effets

Mots clés : Éosinophilie ; Filariose ; Microfilaires ; *Wuchereria bancrofti*

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http://dx.doi.org/10.1016/j.medmal.2012.09.006

secondaires, sont moins onéreux que la détection antigénique. Une possible stratégie complémentaire serait le traitement systématique des patients hyperéosinophiliques.

1. Introduction

Lymphatic filariasis (LF) is a parasitosis caused by three species of nematodes: *Wuchereria bancrofti* (the only species present in the South Pacific), *Brugia malayi*, and *Brugia timori* [1,2]. LF is transmitted by various mosquitoes: *Aedes*, *Anophele*, *Culex*, and *Mansonia.* It is endemic in several tropical and subtropical regions and is a public health issue in South Pacific islands, including French Polynesia (FP) [3].

According to the WHO, it could concern more than 120 millions individual in 83 countries and 1.3 billion could be infected [4]. In 1993, the Center for Disease Control considered LF as an eradicable disease worldwide [5]. In 1999, a program was implemented for its eradication in Pacific islands: the "Pacific Program to Eliminate Lymphatic Filariasis" (PacELF) [6] followed in 2000 by the WHO "global program to eliminate lymphatic filariasis" [7] consisting mainly in yearly campaigns of anti LF treatment mass distribution [8].

LF is responsible for lymphedema and elephantiasis in its chronic presentation; most infected individuals are asymptomatic but are reservoirs for disease propagation. It is not rare to discover fortuitously microfilariae on blood swabs performed for totally asymptomatic patients in endemic zones.

The reference diagnostic technique is microscopic identification of microfilariae [9,10]. This technique is time consuming, weakly sensitive, dependent on the volume of blood examined and on the periodicity of microfilariae circulation [11,12]. The blood test is not specific [13]. The diagnosis by molecular biology is not used in routine [14]. The biological diagnostic was revolutionized by tests detecting circulating filarial antigens [15] in patients carrying adult worms, whether symptomatic or not, with or without microfilariae. Their detection is more sensitive than screening and does not depend on the periodicity of microfilariae circulation; ELISA (Og4C3) or rapid immunochromatographic tests (ICT cards) [16–19] are available. ICT tests are the most frequently used tests in the South Pacific and used routinely in FP.

The polymorphonuclear eosinophil count (EC) in peripheral blood is less than 500/mm^3 [20]. The most frequent etiologies of hypereosinophilia are parasitoses followed by allergies, autoimmune, inflammatory, and neoplastic diseases [21]. LF is responsible for acquired hypereosinophilia which is a biological argument suggesting the diagnosis in endemic zones. Besides LF, the most frequent causes of eosinophilia in FP are immune disorders and allergies, most often asthma. There are few other causes of hypereosinophilia due to parasitoses, probably because of albendazole (ALB) mass distribution campaigns, which have an impact on helminthiases with intestinal tropism.

The main agents used for the management of LF are diethylcarbamazine (DEC), ALB, and ivermectin, used alone or in combinations [22].

Our laboratory, located in Papeete (Tahiti, FP), receives samples every day from the five archipelagos in FP. We present two assessments of activity focused on the specific biological diagnosis of LF (antigenemia and screening for microfilariae) as well as on indirect biological diagnosis (eosinophilia). The correlation between the specific and indirect biological data allows us to suggest complementary propositions for the management of infected patients so as to decrease the prevalence of LF in FP.

2. Material and methods

We made a retrospective study on all the samples received by our laboratory from January 2010 to December 2011. We analyzed all complete blood counts (CBC) performed and for each the number of EC/mm^3 as well as all exams screening for filarial antigens and screening for microfilariae by hemoconcentration. All blood tests had been prescribed by a physician.

2.1. Blood samples

All venous blood samples had been kept and transported at 8 to 10 °C. All blood tests were performed within 24 hours after sampling.

2.2. Complete blood counts

The CBC were performed with the automat Sysmex XT-2000*i*^{TM} (Roche). The determination of leukocyte subpopulations, including the EC, was systematically performed. Nine different threshold values were considered for EC: 200, 300, 400, 500, 600, 700 to 800, 900 and 1000/mm^3. A blood swab was performed for each EC greater than 700/mm^3, stained with May-Grünwald Giemsa, and examined by optical microscope to confirm hypereosinophilia, screen for a possible blood disease, as well as for microfilariae.

2.3. Determination of antigenemia

The detection of filarial antigens was performed with the immunochromatography BinaxNOW filariasis test (Inverness medical), according to recommendations of the technical file issued with the reagents. The test results were read 10 minutes after deposit of serum on a strip to prevent false positive results [23].

2.4. Screening for microfilariae

Screening for microfilariae by hemoconcentration was performed by filtering 1 mL of blood sampled in an EDTA tube and mixed with 5 mL of sodium bicarbonate dampener. The mixture was filtered on a nucleopore filter (Whatman) held by filter carrier. The filtered solution was then rinsed, deposited on a

slide, cool dried, and stained. The reading was performed with an optical microscope at a magnification of 10 × .

2.5. Correlation between eosinophilia and antigenemia

We compared the number of ICT tests performed and their result, for each EC threshold value when the CBC and screening for filarial antigens were available for the same patient. We calculated the prevalence of antigenemia (number of positive ICT results compared to the number of individuals for whom tests were performed for the threshold value considered) for each threshold value. We included all patients for whom antigenemia and CBC were performed on a same blood sample, as well as those for whom CBC was performed in the 15 days following antigenemia assessment. The patients, for whom screening for filarial antigens was prescribed, following the discovery of hypereosinophilia, were not included for the correlation between the two tests.

2.6. Data processing and statistical methods

We determined the sensitivity, specificity, positive and negative predictive value of EC compared to the result of antigenemia for each threshold value. The true positives were defined as patients with hypereosinophilia and positive antigenemia; the false positives were defined as patients with hypereosinophilia and negative antigenemia; the true negatives were defined as the patients with a normal eosinophilia and a negative antigenemia, the false negatives were defined as the patients with a normal eosinophilia and a positive antigenemia. The results of sensitivity (y axis) and of value 1-specificity (x axis) were compared on a Receiver Operating Characteristic (ROC) curve. The rate of prevalence between global antigenemia determined in this study and our previous study in 2008 [24] were compared with a Chi^2 test. The CI was set at 95%.

3. Results

We performed 37,066 CBC during the study period. The average EC was 365/mm^3 and the median 265/mm^3, with values ranging from 0 to 24,161/mm^3; 7503 of these patients or 20.24% (CI 19.84%–20.66%) presented with hypereosinophilia. We performed 1184 tests screening for filarial antigens (158 were positive, 10 indeterminate, and 1016 negative) as well as 222 screening tests for microfilariae by hemofiltration (25 positive and 197 negative).

Both results of antigenemia and of CBC (Table 1) were available for 1061 patients. The results of antigenemia compared to EC threshold values as well as the prevalence of positive antigenemia for each threshold value are listed in Table 1. The prevalence of antigenemia was 25.78% (CI 21.21%–30.35%) for an EC greater than 500/mm^3 and for an EC less than 500 EC/mm^3 it was 7.06% (CI 5.18%–8.96%). Among the 141 patients presenting with positive antigenemia, 50 or 35.46% (CI 27.54%–43.39%) had a normal EC. The global prevalence of antigenemia for these 1061 patients was 13.29% (CI 11.25–15.34).

Table 1
Antigenemia detection results and antigenemia prevalence for the various eosinophil cut offs.
Résultats de la recherche des antigènes filariens et prévalence de l'antigénémie en fonction du nombre de polynucléaires éosinophiles.

EC	Ag positive	Ag negative	Total	Prev Ag positive (%)
> 1000	40	77	117	34.19
> 900	46	102	148	31.08
> 800	57	133	190	30.00
> 700	69	170	239	28.87
> 600	82	222	304	26.97
> 500	91	262	353	25.78
> 400	106	360	466	22.75
> 300	120	486	606	19.80
> 200	128	668	796	16.08
Total	141	920	1061	13.29

EC: polymorphonuclear eosinophil count; Ag: antigen; Prev: prevalence.

Table 2
Results of microfilarial detection for positive and negative antigenemic patients.
Résultats des recherches de microfilaires par hémoconcentration en fonction des résultats de l'antigénémie.

	Ag positive	Ag negative	Total
Positive hemoconcentration	16	0	16
Negative hemoconcentratio	18	127	145
Total	34	127	161

Both results of antigenemia and of hemofiltration were available for 161 patients (Table 2).

Controls on smears for CBC allowed detecting two patients with microfilaremia out of the 3945 smears performed. For the first patient, antigenemia had been prescribed and was positive; the EC was 3720/mm^3. For the second, antigenemia had been prescribed and was negative, the EC was 1990/mm^3. This was the only demonstrated case of false negative in antigenemia observed over a period of two years.

The results of sensitivity, specificity, positive and negative predictive values of eosinophilia compared to antigenemia are listed in Table 3. The results of sensitivity and of values (1 − specificity) were traced on a ROC curve (Fig. 1). The shape

Table 3
Sensitivity, specificity, positive and negative predictive values of eosinophil count compared to antigenemia.
Résultats des sensibilités, spécificités, valeurs prédictives positives et négatives de l'éosinophilie par rapport à l'antigénémie.

EC	SS (%)	SP (%)	PNV (%)	PPV (%)
1000	0.28	0.92	0.89	0.34
900	0.33	0.89	0.90	0.31
800	0.40	0.86	0.90	0.30
700	0.49	0.82	0.91	0.29
600	0.58	0.76	0.92	0.27
500	0.65	0.72	0.93	0.26
400	0.75	0.61	0.94	0.23
300	0.85	0.47	0.95	0.20
200	0.91	0.27	0.95	0.16

EC: polymorphonuclear eosinophil count; SS: sensitivity; SP: specificity; PPV: Predictive Positive Value; PNV: Predictive Negative Value.

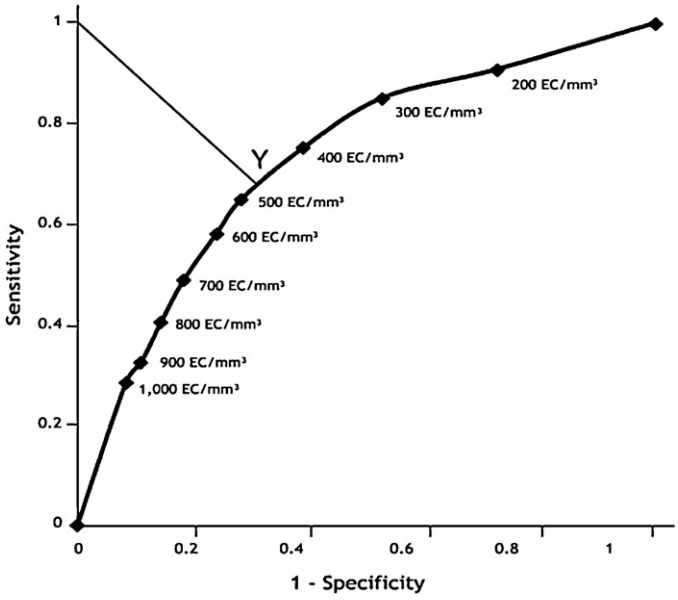

Fig. 1. Sensitivity and specificity, Receiver Operating Characteristic (ROC) curve.

Fig. 1. Sensibilité et spécificité, courbe ROC.

of the curve confirmed the association between antigenemia and hypereosinophilia but did not allow determining an optimal threshold value for the determination of a positive antigenemia from the EC. The point of the curve the farthest away from the line of equity (Youden index) was close to the threshold value of 500 EC/mm^3.

For each threshold value of EC, we calculated the probable number of antigenemic patients (AgPro) by multiplying the number of CBC performed by the prevalence of antigenemia for the given threshold value and we compared the result to the number of antigenemic patients actually detected (AgDet) to this threshold value (Table 4). For example, for a threshold value greater than 500 EC/mm^3 the prevalence of antigenemia was 25.78, the probable number of antigenemic patients was thus 1934 (CI 1591–2277); we detected only 91 antigenemic patients among all the submitted cases.

Table 4
Comparison between the number of probable positive antigenemic patients and detected positive antigenic patients.
Comparaison entre le nombre de patients probablement antigénémiques et le nombre de patients effectivement détectés.

EC	AgPro	AgDet	AgP – AgD
> 1000	608	40	568
> 900	700	46	654
> 800	891	57	834
> 700	1139	69	1070
> 600	1451	82	1369
> 500	1934	91	1843
> 400	2474	106	2368
> 300	3189	120	3069
> 200	3824	128	3696

EC: polymorphonuclear eosinophil count; Ag: antigen; AgPro: antigen positive probable; AgDet: antigen positive detected.

4. Discussion

The prevalence of LF is important in FP, screening for filarial antigens and for microfilariae by hemofiltration are not performed routinely for each blood test because of the cost of antigenemia and the time needed to perform the tests for hemofiltration. Furthermore, screening for filarial antigens is not included in the list of reimbursed biological examinations. On the other hand, CBC are commonly prescribed examinations and counting the leukocyte sub-populations is performed systematically with each CBC.

Besides the yearly campaigns of antifilarial drugs mass distribution, our prevention strategy against LF is based on treating antigenemic and/or microfilaremic patients, combined with vector eradication. This strategy can be applied only if screening has been performed but we noted in our or laboratory that in most cases only major hypereosinophilia (> 1500 EC/mm^3) is followed by complementary biological examinations. Mild hypereosinophilia is not investigated and screening for asymptomatic filariasis is not performed. Screening for filarial antigens for every hypereosinophilic cannot be performed routinely because of the prevalence of hypereosinophilia in our population (20.24%).

Calculating the relationship between hypereosinophilia and antigenemia allowed us calculating the prevalence of antigenemia according to EC, in other words, the probability for a patient to be carrying adult *W. bancrofti* worms according to his EC. The prevalence of antigenemia was estimated at between 21.21% and 30.35% for our population for a threshold value of 500 EC mm^3/mL. The relationship between LF and hypereosinophilia was known but the relationship between hypereosinophilia and antigenemia had never been demonstrated, and the prevalence of antigenemia according to the EC had never been calculated.

Our results confirm the weak sensitivity of screening for microfilariae by hemoconcentration compared to antigenemia since 18 patients had a positive antigenemia and negative microfilaremia. They prove that performing a simple blood swab to look for microfilariae in case of hypereosinophilia is weakly contributive (only two cases detected out of 3945 swabs performed) and may be a misleading diagnostic factor for the prescriber.

The strategy implemented by the WHO for the eradication of filariasis, consisting in campaigns of antifilarial drugs mass distribution is based on the effectiveness of a dose of DEC (6 mg/kg) for the clearance of microfilariae. Adding ALB (400 mg) potentializes the microfilaricidic effect. The South Pacific regions where LF is endemic follow WHO recommendations and implement these distribution campaigns targeting all their population. In 2007, only three of the 11 Pacific territories endemic for LF saw the prevalence of antigenemia decrease below 1% (Tonga, Niue, and Vanuatu) after five distribution campaigns [25].

Between 1993 and 1999, FP made 14 semesterly campaigns of mass distribution with DEC alone and, since 2000, nine yearly campaigns of mass distribution with bi-therapy combining DEC and ALB (interruption in 2008 and 2009). In 2008, we studied the prevalence of filarial antigenemia to measure the impact of the PacELF program on our population. The serums were collected on a randomized sample of patients more than two

years of age. Antigenemia was determined by ICT cards. The study included 1180 individuals; the global prevalence of antigenemia was 11.3%. The global prevalence calculated on our non-randomized sample was not significantly different (13.29%) compared to our previous study. We drew the conclusion that the relationship between eosinophilia and the results of antigenemia calculated on our patients could be extrapolated to all the Polynesian population. Furthermore, these results confirm that despite campaigns of mass distribution, the prevalence of LF remained much higher than the target value of less than 1% set by the WHO; so other prevention strategies should be developed.

A possible explanation for the still high prevalence of the disease is a low coverage of the population during distribution campaigns. The observance of drug intake in the global population was 85.8% in 2006, 77.9% in 2007, and 75.8% in 2008. A new strategy of "direct controlled intake" was adopted in 2010 to improve the observance. Another explanation explication is related to the vector *A. polynesiensis*. This vector presents with "limitation", meaning that it is more likely to transmit filariasis when the prevalence of filariasis decreases in the population [26].

During the two-year study period we detected 141 antigenemic patients, who were treated. We also detected 7503 hypereosinophilic patients among whom 1934 (1591 to 2277) were probably antigenemic but were not treated. These non-treated patients were probably carriers of adult worms so on one part susceptible to develop a chronic presentation of the disease but also potentially capable of transmitting the disease. Thus, according to our results, around one hypereosinophilic out of four is a potential reservoir for microfilariae.

These patients are not treated because screening for filarial antigens is not performed and they can thus escape the mass distribution campaign, the population coverage of which is not exhaustive.

According to the WHO recommendations, ALB and DEC are well-tolerated agents, especially when used alone and in single doses. Authors of recent studies have reported that their administration in bi-therapy does not increase their toxicity. There are few adverse effects, eventually requiring the administration of antalgics or antipyretics. Thus, there should be no risk in treating hypereosinophilic patients systematically with monodoses (monotherapy or bi-therapy), respecting usual contraindications (including pregnant women and children less than two years of age). A possible strategy would be to treat hypereosinophilic patients systematically. One of the limiting factors for this strategy could be the cost of treatments but we can currently benefit from reduced cost for DEC and ALB by the manufacturer, and with the WHO program. The systematic treatment of all the hypereosinophilic patients would cost less than performing antigenemia systematically for hypereosinophilic patients, followed by treatment of positive patients.

The safety of the minute treatment recommended by the WHO for the treatment of antigenemic patients, and its low cost compared to screening for filarial antigens, allow us to offer a treatment strategy, outside of masse distribution campaigns, based on the systematic treatment of hypereosinophilic patients rather than treatment based on the sole previous and random screening of antigenemic patients.

The possible treatment strategy based on the result of EC added to results of antigenemia or of microfilaremia are valid for our population and for the prevalence of filarial antigenemia in FP. This strategy would be even more interesting in a region with a higher prevalence. Furthermore, this strategy could not be considered in a region where other major causes of hypereosinophilia would be identified as well as in regions with a very low filarial prevalence.

Disclosure of interest

The authors have not supplied their declaration of conflict of interest.

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