cover. The sinus bearing portion of the skin and subcutaneous tissue was widely excised through an elliptical excision and the cavity curetted and the wound packed with gauze soaked in honey. The honey dressing continued till the wound granulated and healed secondarily. Culture of pus grew no organisms but the histology of the excised skin revealed granulation tissue lined tracts in the skin. Five months after the operation she had a mild episode of similar symptoms which responded to a combination of oral ceftriazone, metronidazole and ketoprofen.

Pathology

Pilonidal sinus disease is common in Caucasians and rare in Africans. Its pathology is related to midline pits in the skin overlying the sacrococcygeal region which communicate with a granulation tissue lined cavity in the subcutaneous tissue containing loose hair which are thought to be central in the pathogenesis of the disease. It is considered to be acquired, though congenital sinuses are also possible. Repeated infection of the cavity through the tract is responsible for its chronicity and abscess formation. It is thought that repeated trauma to the natal cleft drives hair into the subcutaneous tissue and eventually a tract is formed which communicates with the cystic cavity. Tight clothing is suspected to contribute to driving hair into the subcutaneous cavity; and our index patient was observed to prefer tight trousers or skirt. The common organisms cultured from pilonidal abscess are bacteroides and anaerobic cocci in greater than 70%.

Clinical Presentation

Pilonidal sinus disease predominantly affects males with a peak incidence after adolescence till age 40. Obesity, being hairy, poor personal hygiene, sedentary occupation and a dark skin colour appear to promote the development of a sinus. The rarity of the disease in blacks is unexplained; under-reporting of cases may be contributory. The disease may be asymptomatic but typically presents as a painful midline swelling with discharge of pus. An abscess follows infection of the tract and present with afluctuant, warm and tender swelling over the sacrococcygeal region which is often recurrent and may extend into either or both buttocks. Fever and a painful swelling overlying the sacrum are the most common complaint when an abscess develops. The differential diagnoses for pilonidal sinus disease are as in Table 1.

Table 1: Differential Diagnoses of Pilonidal Sinus

S/no.	Diagnosis
1.	Anal fissure
2.	Hidradenitissuppurativa
3.	Pyodermagangrenosum
4.	Congenital lesions ex. Presacral sinus, inclusion dermoid and sacrococcygeal sinus

Management

The optimal surgical treatment for pilonidal sinus disease, judged by primary wound healing and recurrence, is unknown. Many procedures, which could be simple or complex, are described and practiced but none fully satisfies all the idealcriteria for treatment of the disease (See Table 2). Pilonodalabscess are managed by incision and drainage, broad spectrum antibiotics and analgesics leading to resolution of symptoms; extensive procedures are not recommended. Patients are advised against putting on tight fitting clothing or sitting on hard surfaces to prevent wound complications.

Table 2: Ideal Criteria for Surgery in Pilonidal Sinus Disease

S/no.	Criteria
1.	Easy to perform
2.	Can be done in theambulatory setting
3.	Low recurrence rate
4.	Minimal pain and wound care
5.	Early return to work
6.	Cost effective

Severe and recurrent cases require more extensive procedures. Excision of the sinus alone or with primary suturing is the most common modality of treatment for the sinus. In a randomized trial of excision alone, excision with primary suturing and excision with primary suturing and clindamycin , Zimmermann-Nielsen *et al.* noted that healing without revision surgery or excision of new sinuses occurred in most

Diagnosis of Malaria in Infants: Evaluation of Clinical Signs

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ABSTRACT

Malaria infection during infancy greatly mimics infections caused by other pathogens such as bacteria and viruses. This similarity in symptomatology and clinical presentation of malaria compared to other infections makes its diagnosisparticularly difficult. The objective of this study was to assess the usefulness or not of certain clinical signs in the diagnosis of malaria in febrile infants attending the Children's Emergency Room of the Lagos University Teaching Hospital and General Hospital, Surulere. Three hundred and twenty two consecutively presenting infants who were febrile either at presentation or had a history of fever in the last 72 hours prior to presentation with absence of obvious localizing causes for the fever were recruited into the study between August 2001- February 2002. Each patient had a full history and physical examination and blood samples were obtained for malaria parasite counts and specie identification. Out of 322 infants, one hundred and forty six infants were females and 176 were males giving a M: F ratio 1.2:1.Amongst the clinical signs elicited in the children, only anaemia (haematocrit<33%) showed a significant association with the presence of malaria parasitaemia (P = 0.00035). Calculation of the sensitivity, specificity, positive and negative predictive values for the clinical signs showed no sensitivity for their use in the diagnosis of malaria in infants. Clinical signs such as fever, splenomegaly and hepatomegaly were not found to be useful in this study as an aid in diagnosing malaria in infancy. Anaemia had a significant association with malaria parasitaemia but had a low overall sensitivity as a sign that could be used for diagnosing malaria in infants.

Keywords: Malaria, Infants, Clinical signs

INTRODUCTION

Malaria in infancy, particularly early infancy, had hitherto been regarded as an uncommon entity.[1] The difficulty in the diagnosis of clinical malaria in this age group lies in the similarity in clinical presentation of other disease entities such as sepsis, which are thought to be more common than malaria in infancy.[2,3] Common symptoms and signs associated with malaria infection include fever, vomiting, diarrhea, cough, pallor, splenomegaly, hepatomegaly and jaundice[2] which can also mimic various other disease conditions. This difficulty in distinguishing clinical malaria in infants acts as the impetus for this study. Therefore this study seeks to evaluate the usefulness of certain clinical signs in the diagnosis of malaria in febrile infants attending the Children's Emergency Room of the Lagos University Teaching Hospital and General Hospital, Surulere.

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MATERIALS AND METHODS

This study was carried out in Lagos, a city located in the South Western part of the country on the Atlantic coast. It is a region characterized by low altitude, high rainfall and high humidity and has two seasons; the rainy season which lasts from May to October, and the dry season which extends from November to April. Malaria transmission occurs throughout the year with peaks during the rainy season. Lagos is holoendemic for malaria.
Two sites were chosen for this study; the Lagos University Teaching Hospital and General Hospital, Surulere. These two centers are located in Mushin and Surulere Local Government areas of Lagos State respectively. These two sites were chosen because they represent the spectrum of population distribution of high and medium density areas of Lagos respectively. Population density has an indirect effect on the prevalence of malaria. Areas with high population density have inherent difficulties in the maintenance of good environmental sanitation thereby creating numerous breeding sites for mosquitoes and indirectly increasing the prevalence of the disease. The two Local Government Areas are contiguous and the two hospitals are readily accessible to the inhabitants of Mushin and Surulere. They are the two largest Government

health facilities in the area and therefore draw the largest clientele of low and medium socioeconomic groups.

The study involved consecutively presenting infants aged one day old to twelve months of age, attending the Children's Emergency Room of the Lagos University Teaching Hospital and the Paediatric Outpatient Unit of General Hospital, Surulere and recruited over a period of six months (from August 2001 February 2002). Informed consent was obtained from the parent/guardian of every patient. The study was approved by the hospitals' ethical committees with informed consent from the patients' care givers. Inclusion criteria: (a) all infants who are febrile at presentation or infants with a history of fever up to seventy two hours prior to presentation with fever being defined as an axillary temperature >37.5^{O}C[4] (b) absence any other obvious cause for the fever (localizing signs)likeabscesses, ear infections. Exclusion criteria: all febrile infants with localizing signs.

The definition of malaria for the purpose of this study was based on the following criteria:- (a) history of fever up to 72 hours prior to presentation or fever at presentation and (b) the presence of malaria parasitaemia in the infant. A detailed history and thorough physical examination was carried out for every infant enrolled in the study. Axillary temperature was measured with a mercury thermometer using four minutes of stabilization time. A diagnosis of malaria was considered for every febrile infant in the study pending confirmation of malaria parasitaemia on blood film.

The usefulness of some clinical signs such as fever, anaemia, hepatomegaly, splenomegaly, hepato-splenomegaly as aids in the diagnosis of malaria in infants were tested using sensitivity, specificity, positive predictive value and negative predictive value. One ml of venous blood was obtained from the patients for examination for malaria parasites and for haemoglobin estimation.

Examination of the peripheral blood smear was used in this study because it remains the current universal "Gold Standard" for the diagnosis of malaria.[5] It is a very reliable method as a parasite count as low as one per microlitre can be detected in a thorough (at least ten minutes) examination of the thick film, [6] hence it's use in this study. Parasite density was also estimated for each patient.

RESULTS

Table 1 shows the clinical characteristics of the infants including their mean ages, mean weights, sex distribution and clinical parameters on presentation. One hundred and forty six infants were females and 176 were males (M: F ratio 1.2:1).

Table 1: Clinical Characteristics of Study Infants by Age Group in Months

Characteristics n = no of patients	= 1month (n=22)	1.1-2 (n=23)	2.1-4 (n=40)	4.1-6 (n=52)	6.1-8 (n=66)	8.1-10 (n=52)	10.1-12 (n=67)	P-Value	P-Value
✍ Age	0.6(0.27)	1.67(0.31)	2.96(0.13)	5.38(0.58)	7.49(0.47)	9.45(0.5)	11.6(0.49)	0.00*	0.00(sig)
✍ Weight	3.62(0.74)	4.53(1.48)	6.16(1.09)	6.76(1.11)	7.57(0.45)	7.96(1.03)	8.59(0.98)	0.00*	0.00(sig)
Sex: Female (%)	9(40.9)	14(60.8)	18(45)	24(46)	27(41)	34(65)	20(30)	0.01*	0.01(sig)
Male (%)	13(59.1)	9(39.1)	22(55)	28(54)	39(59)	18(35)	47(70)	0.01*	0.01(sig)

*P = 0.05 statistically significant

Pilonidal Sinus Disease: A Case Report

***Udo I, Umeh K, Akpanudo E**

ABSTRACT

Pilonidal sinus disease affects the posterior anal or sacral region and when infected may present with an abscess. It is rare in Africans and recurrence after surgery is common. The aim of this study is to report the first case of pilonidal abscess managed by drainage and excision of the sinus tractin our surgical unit. This is a case report with review of clinical presentation, pathology and treatment of the disease in a single patient. Result shows successful outcome with drainage of abscess, laying open/excision of sinus tract and oral broad spectrum antibiotics. In conclusion, drainage of the abscess with laying open/excision of the sinus tract is a simple but effective treatment for pilonidal abscess.

Keywords: Pilonidal abscess, sinus tract excision, broad spectrum antibiotics.

INTRODUCTION

Pilonidal sinus disease with its complications is very rare in our practice. The pathology affects the posterior aspect of the anal and sacro-coccygeal region. It is frequent in Caucasians males and uncommon in Africans and Asians. Approximately 20% of the patients present with an abscess and the rest with a chronic discharging sinus. The presenting features in the index case were classical of pilonidal abscess and the management challenging because it was our first time of diagnosing and managing the disease.

Case Report

A 22-year old female presented at the out-patient clinic with a one week history of a painful swelling in her natal cleft which prevented her from sitting up or lying on her back. The pain did not respond to simplenon-prescription analgesics. The swelling had increased in size markedly three days to presentation and this coincided with the period of maximum pain. She had continuous high grade fever associated with chills. She volunteered a history of similar symptoms for which she had undergone incision and drainage twice in the last three months. These produced temporary relief with symptoms recurring within weeks.

She was in severe painful distress with a temperature of 38.5^{o}C. There was a fluctuant swelling overlying the sacrum and coccyx extending into her buttocks, hyperaemic overlying skin, warm to touch and tender with induration extending into her buttocks (see Fig 1).

Figure 1: Pilonidal abscess overlying the coccyx in an Adult Female. Notice the previous incision scar and midline pits (arrows).

Two sinuses were found in the midline of the swelling with their openings blocked by black thick viscid substance. A vertical scar was overlying the swelling to the right of the midline. Rectal examination was unremarkable.

She underwent an open drainage of the abscess under general anaesthesia with antibiotic

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Table 2 shows the clinical signs in malaria parasite positive and negative infants. Only anaemia (haematocrit <33%) showed a significant association with the presence of malaria parasitaemia (P = 0.00035). The other clinical parameters like fever, splenomegaly, and hepatomegaly did not show any significant association with the presence of malaria parasites.

Table 2: A Comparison of Clinical Signs in Malaria Parasite Positive and Negative Infants

CLINICAL SIGNS	MP POSITIVE (n = 178)	MP NEGATIVE (n = 144)	χ^2	P-VA LUE
Fever(at presentation)	102 (57.3%)	72(50.0%)	0.94	0.33
Anaemia (haematocrit<33%)	77 (43.3%)	34(23.6%)	12.75	0.01*
Hepatomegaly	29 (16.3%)	24 (16.7%)	0.0	0.95
Splenomegaly	8 (4.5%)	2 (1.4%)	1.62**	0.1
Hepatosplenomegaly	4(2.2%)	1(0.7%)	0.45**	0.25

*P = 0.05 = statistically significant** Fisher exact results

The sensitivity, specificity, positive predictive value and negative predictive value of the above clinical signs were calculated to investigate their value in the diagnosis of malaria in infancy as shown in table 3. None of the clinical signs showed a high overall sensitivity in the diagnosis of malaria though splenomegaly and hepatomegaly showed high specificities.

Table 3: Sensitivity, Specificity and Predictive Values of Clinical Signs in Diagnosisof Malaria in Infancy by Age Group (In Months)

Variable	Measures	Age 0-=1	>1-=2	>2-=4	>4-=6	>6-=8	>8-=10	>10-=12	All ages
Fever	Sensitivity	63.6	26.7	45.0	51.8	65.8	72.4	57.1	56.7
	Specificity	36.3	75.0	65.0	48.0	36.0	47.8	50.0	49.3
	PPV*	50.0	66.7	56.2	51.8	62.8	63.6	55.5	58.0
	NPV*	50.0	35.3	54.0	48.0	39.1	57.9	51.6	47.9
Anaemia	Sensitivity	18.2	53.3	50.0	40.7	41.5	44.8	45.7	43.2
	Specificity	63.6	100	65.0	60.0	76.0	60.8	90.6	76.4
	PPV	33.3	100	58.8	52.3	73.9	59.1	84.2	69.4
	NPV	43.8	53.3	56.5	45.5	44.2	46.7	60.4	52.1
Hepato-Megaly	Sensitivity	0.0	6.7	15.0	25.9	17.1	17.2	17.1	16.3
	Specificity	100	100	80.0	84.0	76.0	47.8	81.2	83.3
	PPV	0.0	100	42.8	63.6	53.8	29.4	50.0	54.7
	NPV	50.0	36.3	48.5	51.2	35.8	31.4	47.2	44.6
Spleno-Megaly	Sensitivity	0.0	0.0	0.0	7.4	2.4	13.8	2.8	4.5
	Specificity	100	100	100	100	96.0	100	96.8	98.6
	PPV	0.0	0.0	0.0	100	50.0	100	50.0	80.0
	NPV	100	34.8	50.0	50.0	37.5	47.9	47.7	45.5
*H + S	Sensitivity	0.0	0.0	0.0	3.7	4.8	6.9	0.0	2.2
	Specificity	100	100	100	100	96.0	100	100	99.3
	PPV	0.0	0.0	0.0	100	66.6	100	0.0	80.0
	NPV	100	34.8	50.0	49.0	38.1	46.0	47.8	45.1

*PPV: Positive predictive value *H + S (Hepatomegaly + Splenomegaly)
*NPV: Negative predictive value

DISCUSSION

As earlier noted, the clinical presentation of malaria mimics many other disease conditions as seen in the infant. The value of clinical signs such as fever, anaemia, hepatomegaly and splenomegaly in the diagnosis of malaria in infancy were investigated in this study. Fever, hepatomegaly and splenomegaly did not demonstrate any significant association with the presence of malaria parasitaemia and this is as noted in other studies.[7,8,9,10] Only anaemia showed a significant association with the presence of parasitaemia and this finding is corroborated by some other authors.[7,8,11,12] The development of anaemia in malaria parasite positive patients has been adduced to several factors, one of the most important being direct destruction of the red blood cells by the malaria parasites.[13]

The overall sensitivity of clinical signs in this study for the diagnosis of malaria in infancy was not high and this is in keeping with the demonstrated lack of association between malaria parasitaemia and the clinical signs noted above. Thus only hepato-splenomegaly showed a relatively high degree of specificity ranging from 83.3%-99.3% for hepatomegaly and splenomegaly respectively. This high degree of specificity noted implies that hepatomegaly and splenomegaly are non-specific as signs in distinguishing malaria from other infections. There are other disease conditions that can give rise to enlargement of the liver and spleen in infancy such as sepsis, cytomegalovirus infection, toxoplasmosis. In consequence, elements of clinical algorithms which incorporate fever =38^{o}C and splenomegaly,[14,15] as significant predictors of *P. Falciparum* parasitaemia were not found to be useful in this study. They have been found to have increasing significance only in children above one year of age and in those with malaria parasitaemia>10,000 parasites/µl[7]Generally, parasite counts/densities in infants have been found to be low with 41-49% of parasite positive infants having a parasite density of less than 1000/l.[16]
In this study, the highest parasite count was 5,600 parasites/µl and therefore further corroborates the fact that the outlined parameters are not useful in malaria diagnosis when the parasite count is less than 10,000 parasites/µl.

In summary, the use of clinical algorithms which incorporate certain clinical signs such as fever, splenomegaly and hepatomegaly have not been found to be useful as an aid in diagnosing malaria in infancy. In this study, even though anaemia was found to have a significant association with the presence of malaria parasitaemia, it was not found to have a high overall sensitivity as a clinical sign which could be used as an aid in diagnosing malaria in infancy. However, a high index of suspicion for malaria should be exercised in any ill infant who presents with anaemia in the presence of non-specific symptoms and signs. Such an infant should be given the benefit of the doubt and treated for malaria while being investigated to exclude other possible diagnoses.

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often, no treatment is necessary.[13] It is however paramount to determine the underlying etiology and treat accordingly. Correction of blood sugar, treatment of underlying infection and management of the structural lesion remain the mainstay of therapy.[13,20] Patients with hemiballismus secondary to acute ischaemic stroke will require evaluation and treatment.[21] In patients with hyperosmolar hyperglycaemic state, correction of blood sugar and longtime diabetic control is optimal for good recovery.[20]

Medical treatment in hemiballismus has a dual role. Control of these violent movements helps in the prevention and reduction of injuries in the severe cases. Similarly in pronged cases of ballismus, the use of medications is of paramount importance to prevent exhaustion. In 1976, Klawans et al reported 11 patients who developed hemiballismus and had a marked response to neuroleptic therapy.[6] A similar observation that Levodopa worsened chorea led to the hypothesis that the dopamine system was involved in ballism. Early therapy with dopamine receptor antagonists was noted to be successful in these patients.[21] Neuroleptics therefore have been the mainstay of drug therapy for hemiballismus in the past three decades. Of all the neuroleptics, Haloperidol has been the most successful.[12] Other drugs like Chlorpromazine, Primozide and Diazepam have been tried. More recently, newer atypical neuroleptics (Olanzapine[22] and Clozapine[23]) and Dopamine depleting agents (Tetrabenazine[24]) have been reported to be of benefit. Drug- induced Parkinsonism is a noted complication of the dopamine antagonist or depleting agents .[21]

All our patients had similar management which was mainly supportive. This included rehydration with normal saline, correction of blood sugar for those with hyperglycaemia and Haloperidol 5 mg daily. Some of the patients had to be sedated with Diazepam in the Normal Saline infusion. Supportive care to avoid injuries was also given including providing a safe environment. The movements reduced and patients were discharged home well on antihypertensive drugs, hypoglycaemic agents and Statins as applicable. All the patients were placed on Aspirin for secondary prevention of stroke. Only two of the patients came for follow up. Both are well with no residual abnormal movements.

Prognosis

The natural history of hemiballismus varies depending on the etiology.[5] Most patients who develop acute hemiballismus however, have a favorable prognosis irrespective of the cause.[6] In a 2010 observational study of 15 patients with hemiballismus, 8 patients(53%) had a rapid resolution of the movements without treatment and over a period of 17 months, 11patients (73%) had full resolution.[25] Most of these eleven patients had full resolution of movements within two months.[25] In patients developing hemiballismus secondary to hyperosmolar hyperglycaemic state, the clinical course is favourable with the normalization of blood sugar.[5] In our series, all the patients had a remarkable reduction of the movements before discharge. The only two persons who continued follow up had full resolution within one month.

CONCLUSION

Hemiballismus may not be as rare as previously thought. There is need for a high index of suspicion. The sudden surge may also be a reflection of the paradigm shift from communicable diseases to non- communicable diseases seen in Africa. Possible association of environmental and genetic factors is of considerable importance. The prognosis is good.

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patients were from the Ibibio ethnic group in the South South, Nigeria residing in Akwa Ibom state. Considering the rarity of this disorder, this surge seen in persons of a particular ethnic group residing in the same environment raises the question of possible environmental or genetic predilection.

The uncontrollable flinging movements started suddenly in all our patients. They all presented within the first week of the abnormal movements. There were no other complaints. No history of fever, seizures, loss of consciousness or slurred speech, blurred vision or headaches. No associated weakness of one side of the body, deviation of the mouth to one side or speech deficits. There was no history of weight loss or parasthesias.

All the patients had risk factors for stroke (nonmodifiable like older age, black race, male sex or postmenopausal women) and at least one modifiable risk factor for stroke. These were hypertension, Diabetes mellitus, obesity and dyslipidaemias. These remain the commonest risk factors for stroke in the Nigerian practice.[7] Some had more than one modifiable risk factor. Five (83.33%) out of the six patients were persons with known diagnosis of hypertension. The admitting blood pressure however was markedly elevated in all the six patients. Two had a previous diagnosis of Diabetes Mellitus with associated obesity. These two presented with severe hyperglycaemia and one was managed for hyperosmolar hyperglycaemic state. Brain imaging was not available in the hospital as at the time these patients presented. This was one great challenge in this observation. The hospital however purchased a CT scan machine shortly. Other investigations done were mainly base line investigations and those to identify the risk factors.

Localization/Pathophysiology

As far back as 1927 when it was first described, it was also recognized that patients developing acute onset hemiballismus/ hemichorea often had damage to the contralateral basal ganglia or thalamus typically acute ischaemic or haemorrhagic stroke. The corpus luysii e.g. subthalamic nucleus (STN) was identified early as the vital structure involved in the pathophysiology of hemiballismus[8]. Martin proposed naming the condition the 'syndrome of the body of Luys.[3] Further studies noted that lesions outside the sub thalamic nucleus can also cause hemiballismus. Some of these were found in experimental animal studies with rhesus monkeys.[9] More recently, surgical literature has provided additional insight to the localization of hemiballismus based on experience with stereotactic operations (thalamotomy and pallidotomy) for Parkinson disease in the 1960s.[10] Other structures implicated in hemiballismus are the lateral thalamic nucleus and rarely the globuspallidus.[11]

Etiology

Vascular and structural lesions within the contralateral STN and the basal ganglia remain the commonest causes of hemiballismus[1]. Stroke is by far the most common etiology[1] even though hemiballismus is seen in only 0.45% of stroke patients. Two studies noted 11 of the 21 patients with stroke in one and 18 of 25 in another.[11,12] In our series, all the patients had risk factors for stroke. Another commonly recognized etiology noted is hyperosmolar hyperglycaemic state. This is found mostly in South East Asia but there has been a report of six cases elsewhere in Nigeria.[12] Two of our patients had severe hyperglycaemia. Other more rare causes from case reports include encephalitis, vasculitis, central nervous system (CNS) Lupus, cerebral toxoplasmosis with AIDS, neurocystercosis, mass lesions (neoplasms and cysts) and multiple sclerosis. These are mostly from case reports.[12,14-16] Even more rarely, drugs have been implicated.[17,18] Traumatic brain injury as a cause of hemiballismus has been recently reported.[19]

Diagnosis

Diagnosis is characteristically clinical since the abnormal movements are usually dramatic. Characteristic CT scan and MRI findings are seen in almost all cases. Contralateral basal ganglia hyperdensities are common on head CT scan signifying haemorrhage.[19,20] Brain MRI shows T1 hyperintensities and T2 hypointensity in the contralateral basal ganglia.[20] We made a clinical diagnosis in all our patients based on the history and characteristic violent flinging movements. The hospital had not acquired a CT scan as at the period of this study. This absence of recent modalities of brain imaging was a major challenge in this report.

Management

Hemiballismus resolves with time, and

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