The African Journal of Medicine and Medical Sciences

Volume 17
1988

BLACKWELL SCIENTIFIC PUBLICATIONS
Oxford London Edinburgh Boston Palo Alto Melbourne

Afr. J. Med. med. Sci. (1988) 17, 71–75.

Haemolytic anaemia associated with Nigerian barbecued meat (red suya)

C. K. O. WILLIAMS, B. O. OSOTIMEHIN*, G. B. OGUNMOLA‡ AND A. A. AWOTEDU†
*Departments of Haematology, *Chemical Pathology and †Medicine, College of Medicine, and ‡Department of Chemistry, University of Ibadan, Ibadan, Nigeria*

Summary

Five cases of haemolytic anaemia occurring in male Nigerians following the ingestion of spiced barbecued meat (suya) are described. Although suya is a popular food item in various parts of Nigeria, all five patients described in this report had consumed a special brand, called red suya, purchased from vendors at a popular road junction between the cities of Lagos and Ibadan. Ingestion of the culprit suya sample was followed within 24 h by malaise and fever, while passage of dark-coloured urine and jaundice followed 1–3 days thereafter. Glucose-6-phosphate dehydrogenase (G6PD) deficiency was demonstrated by a fluorescent screening test in all cases, while the enzyme phenotype was shown to be GdA^- in all four cases studied by starch-gel electrophoresis, thus suggesting that G6PD deficiency was a predisposing factor in the cases reported in this series. The haemolytic disease was self-limiting and full recovery followed in all cases. In view of the markedly circumscribed range from where the patients originated, the culprit agent responsible for the haemolytic disease is believed to be a recently introduced food additive that is probably accessible only to a limited number of suya vendors.

Résumé

Cinq cas d'anémie hémolytique chez des Nigérians après l'ingestion d'une viande rôtie et hautement épicée ('le suya') sont décrits. Bien que le 'suya' soit une denrée populaire presque partout au Nigéria, tous les cinq patients décrits dans ce rapport en avaient consommé un type particulier dénommé le 'suya rouge' qui se vend à un arrêt populaire sur l'autoroute qui va de Lagos à Ibadan. En 24 heures après l'ingestion d'un échantillon du 'suya rouge', la malaise et la fièvre sont apparues chez les patients, suivies entre un à trois jours après par le passage d'une urine d'une couleur foncée et la jaunisse. Une carence de Glucose-6-phosphate dehyrogenase (G6PD) chez tous les patients a été démontrée utilisant un écran fluorescent, tandis que le phénotype de la diastase s'est relevé être Gda^- chez quatre patients étudiés d'après l'électrophorèse de la gélatine de fécule. Ceci pourrait indiquer qu'une carence de G6PD est un facteur prédisposant dans les cas rapportés de cette série. La maladie hémolytique s'est limitée elle-même et il y avait une guérison complète dans tous les cas. Compte tenu de la région très réduite d'où sont issus tous les patients, il semblerait que l'agent responsable pour cette maladie hémolytique soit un additif alimentaire récemment introduit et auquel seulement quelque vendeurs du 'suya' ont accès.

Correspondence: Dr C. K. O. Williams, Department of Haematology, University College Hospital, Ibadan, Nigeria.

Introduction

Haemolytic anaemia occurring after ingestion of food has a classical recognition in the syndrome of favism [1], a clinical state that occurs following ingestion of fava beans (*Vicia fava*), and which has been associated with the deficiency state of glucose-6-phosphate dehydrogenase (G6PD) in individuals of Mediterranean, but not of African origin [2]. Recently, however, cases of haemolytic crisis occurring after the consumption of a specially spiced brand of Nigerian barbecued meat, locally

called red suya, have been observed in Southern Nigeria.

The first observation of the association of haemolysis and red suya was made in the last week of 1980 when two brothers presented simultaneously to the University College Hospital (UCH, Ibadan, Nigeria) with evidence of haemolytic anaemia. On investigation, it was established that the two had shared a snack of red suya during a trip to Lagos shortly before their illness. On recovery, one of the patients, not believing the postulated association between the ingestion of red suya and the occurrence of haemolytic anaemia, rechallenged himself with a sample of red suya purchased from the original vendor. He developed the syndrome of haemolytic anaemia in the same fashion as previously. Since the recognition of the association, several cases of haemolytic anaemia diagnosed in our hospital have been attributed to consumption of red suya. However, we have had the opportunity of studying only three of these cases, which are included along with the two original ones in this report.

Patients and methods

The diagnosis of haemolytic anaemia was established on the basis of a full blood count (FBC), including a reticulocyte count done in the usual way [3], as well as determination of serum bilirubin, including the conjungated and unconjugated fractions. The status of erythrocyte G6PD was routinely screened for by a fluorescent 'spot test' [4] and subsequently confirmed by starch-gel electrophoresis [5].

Case reports

Patient 1 was a 34-year-old male Nigerian physician and medical scientist who was in good health until a few days prior to admission to the hospital with a 3-day history of malaise, fever and passing of dark-coloured urine. His 28-year-old brother, patient 2, simultaneously presented with similar symptoms. Both of them denied a previous illness of the kind, while admitting to having shared some red suya bought from a vendor at the Shagamu Junction of the Ibadan–Lagos Expressway a few days prior to the onset of their illness. Both admitted to having previously eaten suya purchased elsewhere even from the other side of the Expressway, without similar sequelae. Patient 1 admitted to taking a curative dose of amodiaquine when he became febrile prior to the onset of haemolytic episode, but his brother (patient 2) did not. On examination, neither the spleen nor the liver was palpably enlarged in either of the patients. They were both jaundiced and had moderate conjunctival pallor.

Patient 3 was a 19-year-old male Nigerian bank clerk who stopped at the Shagamu Junction and purchased red suya, which he shared with a female companion. About 24 h thereafter, the patient became ill with fatigue, malaise and fever, and observed the passage of dark-coloured urine. On examination, there was marked conjunctival pallor and moderate scleral jaundice. Neither the liver nor the spleen was palpably enlarged. The female companion did not show any evidence of haemolytic anaemia.

Patient 4 was a 35-year-old male Nigerian poultry farmer who, within 24 h of consuming some suya purchased at the Shagamu Junction, developed fever, malaise and observed darkening of the colour of his urine. He admitted to having eaten suya in the past without problems. On examination, there was conjunctival pallor but no scleral jaundice. Neither the liver nor the spleen was palpable.

Patient 5 was a 30-year-old male Nigerian marketing executive, who purchased and consumed an undetermined quantity of red suya at the Shagamu Junction. Within 24 h, the patient felt 'unwell' and treated himself with chloroquine sulphate. Three days after ingestion of red suya, he observed the passage of dark-coloured urine and was observed to be jaundiced. Further clinical examination did not reveal other remarkable findings.

Results

The five patients who were seen at the University College Hospital, Ibadan between December 1980 and December 1983 were all middle-class Nigerian men between the ages of 18 and 35 (median: 32) years. In all five cases, the culprit suya sample had been purchased at the Shagamu Junction of the Expressway, Lagos–Ibadan route, in two cases (patients 3 and 4)

and Ibadan–Lagos route in three cases (patients 1, 2 and 5). Overt evidence of haemolysis was preceded in all cases by non-specific signs of ill-health within 1–3 days of ingestion of red suya, and then by the passage of dark-red urine, variously likened to Coca-Cola or blackcurrant juice. Scleral jaundice occurred in four patients within 1–3 days. Apart from signs of anaemia and icterus, there were no remarkable physical findings.

The laboratory findings are outlined in Table 1. These were consistent with a haemolytic disease in all four in whom the relevant data were obtained. There was G6PD deficiency in all five patients and the phenotype of the enzyme was shown to be GdA^- in all four in whom starch-gel electrophoretic analysis was carried out. Of the two patients who had laboratory assessment of renal functions performed, one (patient 4) showed evidence of acute renal failure on initial presentation. This subsequently improved with conservative medical treatment. Although all five patients were severely ill at the time of admission to the hospital, the haemolytic disease was self-limiting and there was complete haematological recovery within 3–4 weeks in every case.

Discussion

We have described a syndrome of haemolytic anaemia in five male G6PD-deficient Southern Nigerians following the ingestion of red suya, a specially spiced brand of barbecued meat popularly consumed in the northern parts of the country, but which has recently become more popular among middle-class Nigerians of the South. The present series includes two patients in whom the association of red suya with haemolytic anaemia was first established, one of whom (patient 1) having recovered from the first bout of haemolysis, rechallenged himself voluntarily with red suya from the original vendor and recorded the reoccurrence of the syndrome (Fig. 1). All five patients claimed to have eaten suya without similar problems in the past. It, therefore, appears likely that the agent responsible for haemolysis in these cases was a recently introduced food additive that was available only to a limited number of suya vendors. Since the recognition of the syndrome of 'red suya-associated haemolytic anaemia' (RESUYAHA), we are aware that a number of other patients, including women, have been suspected on clinical grounds as suffering from the syndrome. However, such cases have not been adequately investigated and have, therefore, not been included in this series. It is, however, conceivable that RESUYAHA may not be as rare as this report might suggest. A recent report described 10 cases of RESUYAHA at another Nigerian Medical Centre [6].

In view of the fact that RESUYAHA is food-associated, one wonders whether it is caused by

Table 1. Laboratory profile on admission of patients with 'red suya'-associated haemolytic anaemia (RESUYAHA)

Patients	PCV	Ratio(%)	Serum bilirubin (mmol/l)		Asp-T* (iu/l)	Ala-T† (iu/l)	SU‡ (mmol/l)	SCR§ (mmol/l)	AP¶ (iu/l)	G6PD Phenotype
			Total	Direct						
1	29	10	188.1	12.0	55	33	n.d.	n.d.	50	A^-
2	25	8	78.7	10.3	44	18	n.d.	n.d.	50	A^-
3	23	n.d.**	126.5	18.8	19	14	n.d.	n.d.	n.d.	A^-
4	23	n.d.	n.d.	n.d.	n.d.	n.d.	37	2.8	n.d.	'Deficient'††
5	27	n.d.	63.3	10.3	25	37	5	0.2	n.d.	A^-

*Asp-T: Aspartate transaminase (serum).
†Ala-T: Alanine transminase (serum).
‡SU: Serum urea.
§SCR: Serum creatinine.
¶AP: Alkaline phosphatase (serum).
**nd.: Not done.
††'Spot' test only.

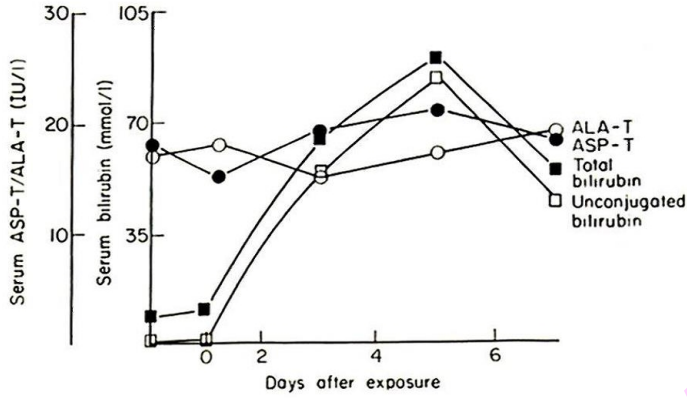

Fig. 1. Biochemical profile in patient 1 following voluntary exposure to red suya.

a local plant-based food additive and, therefore, may represent Africa's equivalent of favism, or more likely the chemically induced 'primaquine-sensitive haemolytic anaemia'. Although patients 1 and 5 admitted to taking curative doses of the anti-malarial drugs amodiaquine and chloroquine, respectively, prior to the onset of haemolysis, it is not clear whether this influenced the degree of the haemolysis. The clinical features of RESUYAHA are, however, similar to those of G6PD-deficiency haemolytic disorders [7], be they drug-induced or secondary to ingestion of fava beans. Orange RN (monosodium salt of 1-phenylazo-2-naphthol-6-sulphonic acid) present in the red-colouring powder used as red suya garnish has been postulated by Akinyanju and Odusote [8] as the culprit agent in the haemolytic event of RESUYAHA. The suggestion that a metabolite of this chemical, i.e. 1-amino-2-naphthol-6-sulphonic acid (ANSA) is probably responsible for the haemolytic red cell destruction [9] would tend to explain the variability in the clinical manifestations of RESUYAHA. Thus, its occurrence, as well as the onset of haemolysis, would be subject to several factors including the quantity of the culprit agent consumed as well as host metabolic and disposal factors.

The syndrome of red suya-associated haemolytic anaemia does not appear to constitute a significant community health problem, despite the high prevalence of G6PD deficiency in the Nigerian population [2]. The description in this report of only five cases from an observation period of 3 years should not be taken to reflect the prevalence of the haemolytic disease, especially since several cases of brisk haemolytic disease that were observed in the hospital during the observation period were assumed to be RESUYAHA based on history only. Such cases were not further investigated. The transient nature of the disease and its complete reversability through conservative management probably contributed to this situation. Furthermore, the localized nature of site of exposure, coupled with the fact that only members of the very small middle- and upper-class of the Nigerian society were, for socio-economic reasons, at risk of exposure to the culprit agent, probably contributed to the limited number of the observed cases. Another factor that might have contributed to the low number of cases observed may be that individuals at risk were more likely to seek help in private hospitals rather than in the public General or Teaching Hospitals.

Cases of RESUYAHA appear to have sharply declined in recent months, probably as a consequence of the dramatic reports of the illness in the news media, and perhaps because of some less publicized actions of health authorities against suspected culprit vendors of Shagamu. Nonetheless, it would appear that the case of RESUYAHA should illustrate the community health problems of indiscriminate disposition into developed countries of potentially hazardous food additives like Orange RN, which reportedly has been banned from the markets of some developed countries [8].

References

1. Kattamis CA, Kyriazakou M, Chaidas S. Favism: Clinical and biochemical data. J Med Genet 1969;6:34–41.
2. Bienzle U. Glucose-6-phosphate dehydrogenase deficiency. Part I: Tropical Africa. In: Luzzatto L, ed. Haematology in Tropical Areas. London: W.B. Saunders, 1982:785–99.
3. Dacie JV, Lewis SM. Practical Haematology, 5th edn. Edinburgh: Churchill Livingstone, 1975.
4. Beutler E, Mitchell M. Special modifications of the fluroescent screening method for glucose-6-phosphate dehydrogenase deficiency. Blood 1968;32:816–8.
5. Luzzatto L, Afolayan A. Enzymic properties of different types of human erythrocyte glucose-6-phosphate-dehydrogenase, with characterization of two new genetic variants. J Clin Invest 1968;47:1833–42.
6. Akinyanju OO, Odusote KA. Red Suya Syndrome (Letter). Lancet 1983;i:935.
7. Beutler E. Glucose-6-phosphate dehydrogenase deficiency. In: Williams WJ, Beutler E, Erslev AJ, Rundles WR, eds. Haematology. New York: McGraw-Hill Book Co., 1977:466–78.
8. Akinyanju OO, Odusote KA. Cause of red suya syndrome is food dye orange–RN (Letter). Lancet 1983;ii:1314.
9. Wuertzen G, Larsen J Chr, Tarding F. Formation of hemiglobin (Fe) *in vivo* and *in vitro* after administration of a food additive, Orange RN. Scand J Clin Lab Invest 1982;29 (Suppl 126):13–20.

(*Accepted* 5 *March* 1987)