

Heart attack, stroke, diabetes, and hypertension in West Indians, Asians, and whites in Birmingham, England

Hypertension is more common in black Americans than whites and is also common in Jamaica.^{1,2} In Jamaica heart attacks are infrequent, and mortality in blacks in Britain may also be lower.³ The prevalence and complications of hypertension among West Indians in Britain have not yet been systematically examined. After observing that heart attacks seemed unduly uncommon in this group we examined admissions to Dudley Road Hospital over five years and compared rates for heart attack, stroke, hypertension, and diabetes among the three main ethnic groups.

Number (%) of admissions for all causes (excluding obstetrics and gynaecology) compared with heart attack, stroke, diabetes, and hypertension among ethnic groups aged 30 to 59 years during 1974 to 1978 inclusive

Reason for admission	Place of birth				Total
	Europe and UK	West Indies	India, Pakistan, Bangladesh	Other/not known	
<i>Men</i>					
All admissions	11 157 (71.4)	1227 (7.9)	2289 (14.7)	951 (6.1)	15 624
Heart attack	513 (73.7)	30 (4.3)	109 (15.7)	44 (6.3)	696*
Stroke	322 (71.1)	47 (10.4)	51 (11.2)	33 (7.3)	435†
Diabetes	206 (50.0)	73 (17.7)	115 (27.9)†	18 (4.4)	412‡
Hypertension	294 (55.8)	121 (23.0)	80 (15.2)	32 (6.0)	527‡
<i>Women</i>					
All admissions	9235 (76.7)	1048 (8.7)	1156 (9.6)	604 (5.0)	12 043
Heart attack	103 (87.3)	6 (4.0)	10 (6.7)	3 (2.0)	149§
Stroke	234 (74.5)	43 (13.7)	21 (6.7)	16 (5.1)	314*
Diabetes	180 (50.1)	90 (25.1)	66 (18.4)*	23 (6.4)	359‡
Hypertension	199 (52.2)	135 (35.4)	35 (9.2)	12 (3.2)	381‡

* $p < 0.01$. † $p < 0.05$. ‡ $p < 0.001$. § $p < 0.02$. (χ^2 tests of significance)

Patients, methods, and results

Between 1974 and 1978 27 667 men and women aged 30 to 59 years were admitted and were coded by the Hospital Activity Analysis (HAA) system for diagnosis and place of birth. We noted the frequency of admissions of patients born in Europe ("whites"); those born in the West Indies; and those from India, Pakistan, and Bangladesh ("Asian") for all causes and compared these with the frequency of admissions for heart attack (ICD code 410-4109), stroke (ICD code 430-438), hypertension (ICD code 400-404), and diabetes (ICD code 250). The expected numbers of West Indian and Asian patients admitted with these diagnoses were calculated and compared with the actual numbers (table). The number of heart attacks among West Indians was about half what was expected while the number among Asians was as expected; strokes were much more common in West Indian women and slightly more common in West Indian men. Admissions for diabetes were more common in both Asians and West Indians, but hypertension was more common only in West Indians.

Comment

These results show important differences in admission rates between the three ethnic groups in Birmingham despite similar home backgrounds. Despite the higher incidence of hypertension among West Indians they had fewer heart attacks, although they had proportionally more strokes. The HAA coding system we used has in general proved free of errors for definitive diagnoses,⁴ although errors are found in the coding of non-specific diagnoses such as "viral infections" or "gastroenteritis." We have checked and confirmed its reliability in this study by a manual examination of heart attack admissions in 1976.

What is the explanation of these ethnic differences? Differences in other coronary risk factors remain to be examined, but in a survey of local factory employees smoking rates were equally high among West Indians and whites (J K Cruickshank and D G Beevers, unpublished observations). Both ethnic minorities may be subjected to greater "urban stress" than whites. If so, they clearly respond differently since Asians do not share the relative immunity to heart attack. Perhaps a clue to this apparent protection from heart attack enjoyed by West Indians comes from Jamaica itself. Ashcroft and Desai,⁵ in the only long-term follow-up study from the West Indies, found that

mortality was unrelated to blood pressure below 180 mm Hg systolic and 110 mm diastolic (phase IV). This exciting finding needs further prospective study among West Indians in Britain.

We are grateful to Mrs M Thomas and her staff in the department of medical records for their help.

Reprints from DGB, Dudley Road Hospital, Birmingham.

- Kleinbaum DG, Kupper LL, Cassel JC, *et al.* Multivariate analysis of risk of coronary heart disease in Evans County, Georgia. *Arch Intern Med* 1971;128:934-48.
- Ashcroft MT, Stuart KL. Acute myocardial infarction in the University Hospital, Jamaica, 1968-1970. *West Indian Med J* 1973;22:60.
- Adelstein M. Current vital statistics. *Br Med J* 1978;ii:983-7.
- George LM, Maddock K. Accuracy of hospital activity notifications for infectious diseases. *Br Med J* 1979;ii:1332-9.
- Ashcroft MT, Desai P. Blood pressure and mortality in a rural Jamaican community. *Lancet* 1978;ii:1167-70.

(Accepted 13 August 1980)

University Department of Medicine, Dudley Road Hospital, Birmingham, West Midlands Regional Health Authority, and the University of Birmingham

J K CRUICKSHANK, BSC, MRCP, research fellow (formerly lecturer in medicine, University of West Indies)
D G BEEVERS, MD, MRCP, senior lecturer
VERDELLE L OSBOURNE, AAMS, research assistant
R A HAYNES, AMR, regional HAA officer
J C R CORLETT, medical student
S SELBY, medical student

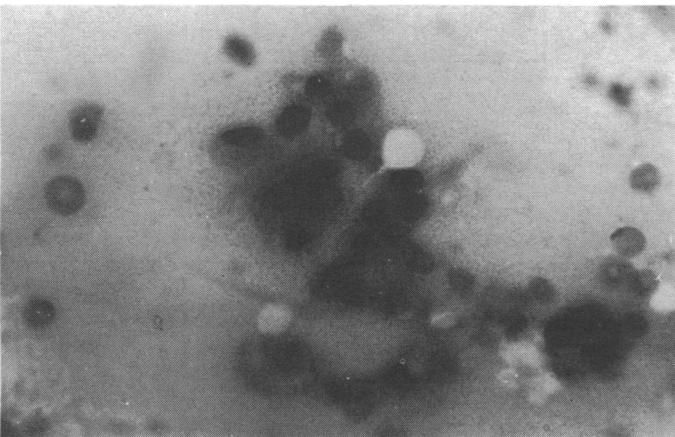
Bone-marrow necrosis and Q fever

Bone-marrow necrosis is rarely diagnosed during life. It has been reported in malignancy and sickle-cell disease.¹ Although it has been described in association with infection, this was based on the findings at necropsy.² We report a case of antemortem bone-marrow necrosis in association with serologically diagnosed Q fever.

Case report

A 42-year-old marine fitter was admitted to a nearby hospital with severe bone pain in the lumbosacral region. Three weeks before admission he had had transient right-sided pleuritic chest pain due to basal consolidation. On admission he had back pain, tenderness, and residual signs of right basal consolidation. His haemoglobin concentration was 12.6 g/dl and white blood cell count $15.7 \times 10^9/l$ ($15\ 700/mm^3$). He was given a week's course of ampicillin and was transfused because his haemoglobin concentration had dropped to 7.5 g/dl without evidence of bleeding or haemolysis. A repeat blood film was noted to be leucoerythroblastic, and he was transferred to our unit. He still had backache and was unwell, jaundiced, and feverish. Examination showed only generalised bone tenderness and signs of resolving pneumonia.

Results of biochemical investigations were normal except for plasma bilirubin concentration 41 $\mu\text{mol/l}$ (2.4 mg/100 ml); conjugated bilirubin 20 $\mu\text{mol/l}$ (1.2 mg/100 ml), serum alanine aminotransferase 279 U/l, serum γ -glutamyltranspeptidase 339 U/l, serum alkaline phosphatase 1329 U/l, and serum 5'-nucleotidase 31 U/l. Concentrations of α -fetoprotein and immunoglobulins G, A, and M were normal. Autoantibody screen and hepatitis B surface antigen were negative. An electrocardiogram was normal. Haemoglobin concentration was 10.3 g/dl, white blood cell count $6.4 \times 10^9/\text{l}$ (6400/mm³; leucoerythroblastic picture), platelet count $18 \times 10^9/\text{l}$ (18000/mm³), reticulocytes 3%, erythrocyte sedimentation rate 12 mm in first h, plasma viscosity 1.54 mPa s(cp), and leucocyte alkaline phosphatase score 174; serum vitamin B₁₂, folate, and iron concentrations, and serum total iron-binding capacity, were normal. Ham's test and direct Coombs test were negative; kaolin-cephalin clotting time was 37 s, prothrombin time British ratio 1.42, and serum fibrinogen concentration 2.8 g/l. Bone-marrow aspirate taken on three occasions from different sites (sternum and both posterior iliac crests) showed classical changes of bone-marrow necrosis (figure). Examination of core biopsy specimen confirmed this, and no malig-



Photomicrograph of bone-marrow aspirate obtained from posterior iliac crest, stained with May-Grunwald-Giemsa stain. Nuclei are intensely basophilic, recognisable features of all cell types are obscured, and cells are surrounded by amorphous acidophilic material. (Original magnification $\times 6000$.)

nant tissue or intracellular organisms (Giemsa and Macchiavello's staining) were seen in the samples.

The patient was treated empirically with steroids (without response) and required regular blood and platelet transfusions. He died three weeks later from a combined cerebral and gastrointestinal haemorrhage. Permission for necropsy was refused.

A search for malignancy—which included plain radiography of chest, abdomen, and skeleton; excretion urography; barium meal and enema; ultrasound scanning of hepatic and renal areas; and isotope scanning of liver and spleen—gave negative results. Cultures of blood, sputum, urine, faeces, and throat swab, and tuberculosis cultures of sputum and bone marrow were negative. The result of a Heaf test was grade one. Serological tests for viral antibodies showed a rise in Q fever phase II titre from less than 1/10 to 1/640, while titre to phase I had risen to only 1/20. Mumps V titre had also risen to 1/640 while mumps S titre remained unchanged at less than 1/10. Other viral titres were negative. Serological reagents were obtained from the Division of Microbiological Reagents and Quality Control, Colindale.

Comment

The diagnosis of Q fever was based on a significant rise in titre to phase II antigen, which is considered diagnostic of recent infection.³ A false-positive result was excluded by testing against normal yolk sac and chlamydia B antigen. The clinical findings of pneumonia with prostration and hepatitis also support the diagnosis. The source of infection was not identified. Although we are reasonably sure from clinical evidence that malignancy was not present, this was not confirmed by necropsy.

The only bone-marrow lesion recorded in Q fever has been granuloma.⁴ We believe that the association between Q fever and bone-marrow necrosis has never been described. Antemortem diagnosis of bone-marrow necrosis in the absence of sickle-cell disease carries serious connotations of underlying malignancy, with a median survival of 22 days.⁵

Although our patient did not benefit from the diagnosis of Q fever, as the results were obtained after death, the treatment is simple and

potentially curative. Q fever should be considered in undiagnosed cases of bone-marrow necrosis.

We thank Dr G B White for performing the serological tests and for helpful discussion, and Dr A H Rowson and Dr G P Sechiari for referring the patient.

- 1 Leysen MHJ, Verwilghen RL. Diagnosis of bone marrow necrosis. *Clinical and Laboratory Haematology* 1979;1:197-202.
- 2 Brown CH. Bone marrow necrosis: a study of seventy cases. *Johns Hopkins Med J* 1972;131:189-203.
- 3 Maudell GL, Douglas GR, Bennett JE. *Principles and practice of infectious diseases*. New York: John Wiley and Sons, 1979.
- 4 Delsol G, Pellegrin M, Familiades J, Auvergnat JC. Bone marrow lesions in Q fever. *Blood* 1978;52:637-8.
- 5 Kiraly JF, Wheby MS. Bone marrow necrosis. *Am J Med* 1976;60:361-8.

(Accepted 5 August 1980)

University Department of Haematology, Royal Liverpool Hospital, Liverpool L69 3BX

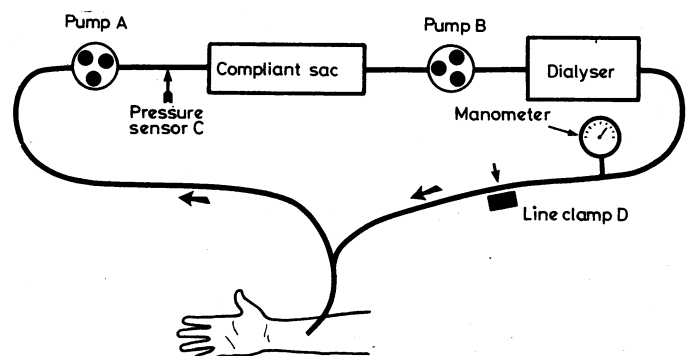
M BRADA, BSC, MRCP, registrar in haematology
A J BELLINGHAM, FRCP, professor of haematology

New system for single-needle dialysis

In regular haemodialysis two needles are usually inserted into forearm veins through which blood flow has been increased by an arteriovenous fistula. One needle supplies blood to the dialyser, the other returns it to the patient. A system in which blood is alternately taken from and returned to the patient through the same needle is theoretically preferable, because the number of needle insertions would be halved, patients would prefer it, dialysis would be possible when suitable needling sites are limited, and fistula life would possibly be longer. Low blood flow rates and recirculation from afferent to efferent dialyser blood lines, however, cause comparatively low dialyser clearances. Fluctuating high venous pressures necessitate the use of a compliant dialyser and predispose to excessive ultrafiltration. Systems using continuous flow via a single needle with a double lumen are less acceptable because the large needles required are difficult to insert and recirculation may be high if blood flow through the fistula is low. For these reasons single-needle dialysis has not gained wide acceptance. One of us (A H) has developed a simple system in an attempt to overcome these problems, and we have compared its performance with that of conventional two-needle dialysis.

Method and results

The system is illustrated in the figure. There are two blood pumps separated by a 50-ml compliant sac. Pump A extracts blood from the fistula and fills the sac. When the sac is full the pressure sensor C simultaneously switches pump A off and pump B on and opens the line clamp D, allowing blood to be pumped through the dialyser and back to the patient. When the sac is empty the sensor C reapplies the line clamp D and switches



The single-needle dialysis system. Dialysis monitor not shown.