Relation between mouth and haematogenous infection in total joint replacements

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Abstract

Objective—To investigate the source of infections associated with orthopaedic prostheses.

Design—Analysis of four infections of prosthetic joints with case records; minimum inhibitory and minimum bactericidal concentrations and sodium dodecylsulphate polyacrylamide gel electrophoresis of the cell wall polypeptides of the *Streptococcus sanguis* isolates from the mouth and infected prostheses; examination of the patients' mouths for periodontal disease and caries.

Subjects—Four adults (three men) aged 58–83.

Results—For each patient the strain of *S sanguis* isolated from the mouth was indistinguishable from that isolated from the prosthesis. All patients had severe periodontal disease and caries.

Conclusions—The mouth was probably the source of bacterial infection in the prosthetic joints of these patients; the route of infection was possibly haematogenous. Incipient oral infection should be treated before joint replacement, and oral health should be maintained indefinitely.

Introduction

Deep infection of prosthetic joint replacements is a serious complication.^{1,2} Several circumstantial cases of secondary infection of prosthetic joints, presumed to originate in the mouth, have been reported.^{3–13} The mouth is a recognised source of bacteraemia, particularly if untreated dental caries or periodontal disease is present. To date, however, no conclusive evidence that the mouth is a definitive source of infection in total joint replacements has been reported. This paper describes four cases of deep infection—three in total knee replacements and one in a total hip replacement—caused by *Streptococcus sanguis* (an α -haemolytic streptococcus of the viridans group), a common constituent of the oral microflora.

Patients and methods

Table I shows details of the four patients.

We identified all the strains of *S sanguis* isolated from the infected prosthesis and mouth using the methods of Beighton and colleagues¹⁴ and determined their minimum inhibitory and minimum bactericidal concentrations against penicillin, erythromycin, tetra-

cycline, amoxycillin, and clindamycin according to the methods of Reeves and colleagues.¹⁵

ANALYSIS OF CELL WALL POLYPEPTIDES

We compared the cell wall polypeptides of the four pairs of *S sanguis* using sodium dodecylsulphate polyacrylamide gel electrophoresis.¹⁶ Briefly, the strains were grown in tryptone yeast extract (Lab M, London), supplemented with 2 g glucose, and incubated at 37°C aerobically for 18 hours. The bacterial growth was harvested by centrifugation and suspended in 10% trichloroacetic acid for one hour. The precipitate, collected by centrifugation, was solubilised in sodium dodecylsulphate. About 100 µg protein was subjected to discontinuous gel electrophoresis according to the methods of Laemmli,¹⁷ with a 5% stacking gel and a 8.75% resolving gel. All gels were fixed in 12.5% trichloroacetic acid and stained with 2% coomassie blue, before being destained in 10% acetic acid. We photographed the gels on Agfa, Dia Direct black and white transparency film (15 ASA; Agfa, London), thus avoiding any distortion by drying and bubble formation during scanning. We scanned photographs of the gels densitometrically (2202 laser densitometer linked to a 2202 plotting and recording integrator; LKB Pharmacia, Bromma, Sweden) and calculated the molecular weight and number of polypeptides resolved.

We analysed the polypeptide pattern further for qualitative differences by summary methods of measurement using the differences in each gel pattern. We arranged every polypeptide detected from each isolate in a contingency table, recording presence or absence. We compared the differences in the polypeptide pattern of the bands between the isolates from the oral cavity and those from the prosthesis. *S sanguis* ATCC 10556 was used as a control strain throughout the experiments.

Results

Table II shows the minimum inhibitory and minimum bactericidal concentrations of the isolates of *S sanguis* derived from the mouth and the prosthesis against five antibiotics. For each patient for each antibiotic the minimum inhibitory and minimum bactericidal concentrations of the isolates from the mouth were the same as for those from the prosthesis

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BMJ 1994;309:506-8

TABLE I—Details of four patients* with postoperative prosthetic infection caused by *Streptococcus sanguis*

Case No	Age (years)	Condition at presentation	Operation	Postoperative symptoms	Treatment	Condition of mouth	
						Infection	Decayed, missing, filled index†
1	83	Osteoarthritis	Total knee replacement	Pain and defect in skin over patella	Antibiotics; removal of prosthesis; arthrodesis with Küntscher nail	Gross inflammatory periodontal disease; periapical infection	34
2	58	Osteoarthritis	Total knee replacement	Pain four months postoperatively	Antibiotics; two stage revision	Gross inflammatory periodontal disease; periapical infection	28
3	64	12 years' history of osteoarthritis	Total knee replacement	Swelling and pain postoperatively	Antibiotics; arthrodesis with Küntscher nail; total knee replacement	Extensive pocketing of periodontal tissues; periapical infection	26
4	75	Two years' history of pain after total hip replacement	One stage revision after core biopsy	Uneventful	Long term antibiotic therapy	Extensive periodontal disease; periapical infection	30

*All patients were male except patient 1. †Index (0–100) relating to health of teeth—the higher the value the poorer the health.

TABLE II—Minimum inhibitory and minimum bactericidal concentrations ($\mu\text{g/ml}$) of *Streptococcus sanguis* isolated from mouth and prosthesis, against five antibiotics

Case No	Source of <i>S. sanguis</i>	Penicillin		Erythromycin		Tetracycline		Amoxycillin		Clindamycin	
		Inhibitory	Bactericidal	Inhibitory	Bactericidal	Inhibitory	Bactericidal	Inhibitory	Bactericidal	Inhibitory	Bactericidal
1	Mouth	0.03	0.5	0.12	1.0	0.12	4	0.06	0.12	0.1	1.0
	Prosthesis	0.03	0.5	0.12	1.0	0.12	4	0.06	0.12	0.1	1.0
2	Mouth	0.03	0.5	0.06	1.0	0.03	0.12	0.12	1.0	0.3	1.2
	Prosthesis	0.03	0.5	0.06	1.0	0.03	0.12	0.12	1.0	0.3	1.2
3	Mouth	0.5	1.0	0.12	1.0	0.5	1.2	0.3	1.0	0.12	1.0
	Prosthesis	0.5	1.0	0.12	1.0	0.5	1.2	0.3	1.0	0.12	1.0
4	Mouth	0.03	0.12	0.03	0.12	0.5	1.0	0.06	0.5	0.12	1.2
	Prosthesis	0.03	0.12	0.03	0.12	0.5	1.0	0.06	0.5	0.12	1.2
Control	<i>S. sanguis</i> ATCC 10556	0.03	0.6	0.12	0.5	0.03	0.12	0.03	0.5	0.1	1.0

and the minimum inhibitory concentrations differed from the minimum bactericidal concentrations.

The number of polypeptides detected from the cell wall of the isolates ranged from 26 to 31. In cases 2 and 4 the number of polypeptides in the isolates from the mouth was the same as the number in the isolates from the prosthesis; in cases 1 and 3 the numbers differed by one. None of the isolates completely matched the polypeptide pattern of the control strain: there were between 3 and 6 different bands. The figure shows the differences between the mouth and the prosthesis in the number and distribution of polypeptides from the isolates of *S. sanguis*.

Discussion

We tried to determine whether the strains of *S. sanguis* isolated from the mouth and the infected prosthesis in four patients were identical. As no definitive method of comparing strains of *S. sanguis* has yet been described we compared the isolates by their antibiograms and cell wall polypeptides. The results of these analyses are consistent with the strain of *S. sanguis* isolated from the mouth being the same as that from the infected prosthesis. Although an exogenous source of *S. sanguis* cannot be unequivocally excluded, a bloodborne dissemination of this oral commensal to the prosthesis is the most likely source of the infections in these four patients.

The mouth has been previously implicated as a source of infection in orthopaedic implants.^{3,13} The proportion of hip replacements that develop haematogenous infection has been estimated at 0.3%.^{18,19} The proportion of hip and knee replacements that are infected from the mouth must be much smaller. The early infections of cases 1 to 3 are the only instances in which *S. sanguis* has been isolated from prostheses at the Wirral hospital in the past four years: an incidence of <0.25%. Despite the unexpected presence of *S. sanguis* in orthopaedic prostheses a neglected mouth can be treated and can be eliminated as a source of such infection. Some studies have suggested a preoperative dental check up before a joint replacement.⁷ We strongly recommend that before orthopaedic pros-

Lane	Source of <i>S. sanguis</i>	No of polypeptides	Difference
1	Mouth	28	1
2	Prosthesis	27	
3	Mouth	31	0
4	Prosthesis	31	
5	Mouth	26	1
6	Prosthesis	27	
7	Mouth	30	0
8	Prosthesis	30	
9	<i>S. sanguis</i> ATCC 10556	26	
10	Molecular weights		

Profiles from sodium dodecylsulphate polyacrylamide gel electrophoresis of cell wall polypeptides of *Streptococcus sanguis* isolates from mouth and prosthesis

theses are implanted incipient oral sepsis should be treated and that patients with implanted prostheses should maintain oral health indefinitely.

We thank Mrs Wendy McKeown for the primary microbiological investigations and Mr N P J Geary for allowing us to include the fourth case in our study.

Clinical implications

- The source of secondary infection of prosthetic joints has been attributed to the mouth
- No definitive evidence for this association, however, has been reported
- This study confirms that oral sepsis can be a source of bloodborne infection of prosthetic joints
- Incipient oral sepsis should be treated before a patient has a joint replacement, and oral health should be maintained indefinitely

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(Accepted 15 June 1994)

Relation between increase in length of hip axis in older women between 1950s and 1990s and increase in age specific rates of hip fracture

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Abstract

Objective—To determine whether length of hip axis in elderly women has increased over the past 40 years and, if so, whether the increase may have contributed to the increase in the age adjusted rate of hip fractures during those years.

Design—Retrospective assessment of anteroposterior x ray films of the pelvis.

Setting—Radiology department of a rheumatology hospital, New Zealand.

Patients—Two cohorts of women aged >60 (mean 70) who were x rayed on the same apparatus in either the 1950s or the 1990s.

Main outcome—Length of hip axis (distance from the medial aspect of the pelvis to the lateral aspect of the femur along the axis of the femoral neck), length of femoral neck (length of hip axis excluding the femoral head and more medial structures), and width of femoral neck (see figure).

Results—Both the mean length of the hip axis and the mean length of the femoral neck were significantly greater in the women whose x ray films were taken in the 1990s than in those in the 1950s (124.0 mm (SE 1) v 130.5 (1), $P=0.0002$; 79.4 (1) v 84.9 (1), $P<0.0001$, respectively). The width of the femoral neck did not change, and the lengths expressed as ratios to width were greater in the more recent x ray films, indicating that these findings are not due to an unrecognised change in radiographic technique.

Conclusions—An increase in the length of the hip axis in elderly women in New Zealand during the past 40 years has occurred which is large enough to account for the increase in the age adjusted rate of hip fractures during those years.

Introduction

Hip fractures occur in about 15% of postmenopausal white women, and morbidity and mortality from these fractures is higher than from all other osteoporotic fractures. The rates of hip fracture have increased substantially in many countries in the past 50 years. The increases are accounted for only partly by the aging of the population, and age adjusted rate of fracture in women increased twofold in Britain during the 1950s to the 1980s,^{1,4} in Scandinavia during 1940-80,^{5,7} and in Hong Kong during 1966-91.⁸ A similar rise has occurred in New Zealand, where the annual number of hip fractures in women aged over 65 increased from 317 in 1950 to 2153 in 1987, while the population of women in this age group increased only twofold.⁹ The reason for the increases in the age adjusted rate of hip fracture is unknown.

Recently, Faulkner *et al* reported that the geometry of the proximal femur is an independent risk factor for hip fracture.¹⁰ They found that the longer the hip axis (the distance from the medial aspect of the pelvis to the

lateral aspect of the femur along the axis of the femoral neck (figure)) the more likely the risk of fracture. This may be due to the length of the hip axis being related to the femoral moment arm—the longer the arm the less force is needed to produce a fracture. The length of the hip axis is also related to height,¹⁰ which has increased substantially in the past 50 years due to improved nutrition. The length of the hip axis, therefore, may also have increased and, if so, may be a factor in the rising age specific rate of hip fracture. To assess this possibility we determined whether changes in the length of the hip axis have occurred in the population at risk of fractures during the period that the rate of fractures has increased.

Methods

We studied anteroposterior x ray films of the pelvis taken in white women aged over 60. We assessed only films that showed the left hip correctly positioned with a clear outline of the greater trochanter and without major deformity or disease. We found 52 films from 1950-62 (median 1956) that met these criteria. We initially searched for similar films taken during 1990-93 but extended our search back to 1986 to find 52 films for comparison. The median date of these films was 1991.

The patients whose films we studied were being investigated for osteoarthritis (36), rheumatoid arthritis (18), back or leg pain (35), or generalised arthralgias (13). All the x ray films that we studied were from the same rheumatology unit in New Zealand, which had used the same radiography equipment and retained all its films since 1950. A film to tube distance of 36 inches (914.4 mm) was used in the 1950s, but this was increased to 40 inches (1016 mm) before the films from 1986-93 were taken. The increase in this distance decreased the size of the image in the later films by about 2%.

The width of the femoral neck and the length of the hip axis were measured from the x ray films with precision callipers (figure). The line of the hip axis was defined as passing through the mid-point of the femoral neck and the point on the femoral head (identified with a compass) furthest from the neck's mid-point. This definition gave an objective and reproducible method for defining the axis. Because the length of the hip axis is potentially influenced both by abnormalities in the space around the hip joint and by deformity of the femoral head, a new dimension, the length of the femoral neck, was also measured. This was defined as the distance along the line of the hip axis from the lateral aspect of the femur to the line joining the superior and inferior extremes of the hip joint (defined as the points of intersection of the margins of the femoral head and the margins of the lateral pelvis (see figure)). The standard deviations for duplicate

Hip joint showing length of hip axis (AC), length of femoral neck (BC), and width of femoral neck (DE). DE was the shortest line across the femoral neck parallel to the broken line. Placing of the lines is described in text

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BMJ 1994;309:508-9