progressive conditions can demand extensive treatment to curb infection and restore function.

The wide variation in rates of tooth loss and retention among states and by selected characteristics suggests that many older adults have not benefited fully from improvements in the prevention and control of oral diseases. Differences in tooth retention by education, income, and race/ethnicity reflect disparities in unmet dental needs (e.g., untreated caries and advanced periodontal diseases) among persons with limited education and income and among non-Hispanic blacks.[2] The findings in this report indicate that Hispanics and non-Hispanic whites reported similar rates of tooth retention; however, other surveys have indicated that Mexican-Americans had higher rates of untreated caries than non-Hispanic whites.[2] These associations might reflect differences in health literacy and behaviors, attitudes toward oral health and dental care, and access to and use of dental services and types of treatment received.[1,2,7] The lower prevalence of tooth retention among smokers might be related directly to the adverse effects of cigarette smoking,[7] which accounts for approximately half of all cases of periodontal disease in the United States.[8]

Self-ratings of health have been associated with functional ability.[9] These associations suggest that older persons who report poorer general health are at increased risk for limited dexterity, mobility, and tolerance of stress; such factors can compromise abilities to maintain oral hygiene, visit a dental office, or tolerate treatment. The results of this study suggest that many aging adults who are in poorer general health have retained most of their natural teeth. These persons likely will need caregiver assistance and innovative strategies to maintain daily self-care, obtain regular oral assessments, and receive primary and secondary prevention services.

The findings in this report are subject to at least four limitations. First, the sample is drawn from the noninstitutionalized population and excludes persons residing elsewhere (e.g., nursing homes or long-term–care facilities). Second, persons without residential telephone service (e.g., persons with lower incomes or households using cellular phones) are excluded. Third, results are self-reported data and have not been validated; however, a strong agreement between self-reported and clinically assessed number of teeth has been documented.[10] Finally, measures of oral health status are limited to tooth loss.

In the United States, older adults usually pay for dental services themselves without the benefit of insurance.[2] Medicare does not cover routine services, and Medicaid provides only limited coverage in certain states; the majority of elderly persons lose their dental insurance when they retire.[2] Community water fluoridation remains the most effective and cost-effective method for caries prevention[3]; current dental recommendations provide additional guidance on best practices in fluoride use, such as brushing teeth twice daily with fluoride toothpaste.[3] Expansion of community-based programs could help increasing numbers of dentate older adults manage their oral health needs. Programs that have focused historically on younger populations might promote oral health in older adults by (1) increasing public and professional awareness of common oral conditions, risk factors, and healthy behaviors; (2) expanding partnerships, especially with organizations focused on aging issues; (3) monitoring oral health status of older adults; (4) ensuring access to clinical services; and (5) increasing support for prevention research and involvement of oral health professionals in tobacco-control activities.[3,4,7]

REFERENCES

1. Burt BA, Eklund SA. Dentistry, dental practice, and the community, 5th ed. Philadelphia, Pennsylvania: WB Saunders Co., 1999.
2. U.S. Department of Health and Human Services. Oral Health in America: a Report of the Surgeon General. Rockville, Maryland: U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Dental and Craniofacial Research, 2000.
3. CDC. Recommendations for using fluoride to prevent and control dental caries in the United States. MMWR 2001;50(No. RR-14).
4. Truman BI, Gooch BF, Sulemana I, et al. Reviews of evidence on interventions to prevent dental caries, oral and pharyngeal cancers, and sports-related craniofacial injuries. Am J Prev Med 2002;23(suppl 1):35-43.
5. Eklund SA. Changing treatment patterns. J Am Dent Assoc 1999;130:1707-12.
6. U.S. Department of Health and Human Services. Healthy People 2010, 2nd ed. With Understanding and Improving Health and Objectives for Improving Health (2 vols.). Washington, DC: U.S. Department of Health and Human Services, 2000.
7. CDC. Total tooth loss among persons aged ≥65 years—selected states, 1995-1997. MMWR 1999;48:206-10.
8. Tomar SL, Asma S. Smoking-attributable periodontitis in the US: findings from NHANES III. J Periodontol 2000;71:743-51.
9. Idler EL, Kasl SV. Self-ratings of health—do they also predict change in functional ability? J Gerontol B Psychol Sci Soc Sci 1995;50:S344–S353.
10. Douglass CW, Berlin J, Tennstedt S. The validity of self-reported oral health status in the elderly. J Public Health Dent 1991;51:220-2.

Clostridial Endophthalmitis After Cornea Transplantation—Florida, 2003

MMWR. 2003;52:1176-1179

1 figure omitted

ENDOPHTHALMITIS IS A SEVERE CONDITION caused by inflammation of the ocular cavity that often is associated with infection of the internal structures of the eye. The source of infection can include bacteria disseminated through the bloodstream and contamination of the cornea at the time of ocular surgery or trauma. Complications include rapid, irreversible vision loss that can progress quickly to panophthalmitis, requiring surgical removal of the eye.[1] *Clostridium perfringens*, an anaerobic gram-positive bacillus found in soil and bowel flora, is an infrequent cause of endophthalmitis. Although the majority of cases are caused by penetrating injury with soil-contaminated foreign bodies, *C. perfringens* endophthalmitis has been reported in patients after cataract surgery.[2,3] This report describes two cases of *C. perfringens* endophthalmitis that occurred within 24 hours after transplant of contaminated corneas. These cases demonstrate the potential

for transmission of *Clostridium* infection from donor to recipient. Clinicians should be aware of potential infection risks associated with transplantation of corneal tissues and report any infections to the appropriate eye bank.

In February 2003, two patients received corneal transplant of the right eye on the same day in the same facility. The corneas used for both patients were recovered from one donor, a woman aged 55 years who died from metastatic colon cancer.

The first patient, a man aged 64 years, had severe eye pain, nausea, and vomiting within 12 hours after surgery. He had increased intraocular pressure and decreased light perception in the eye in which the cornea was transplanted. Eye examination was consistent with endophthalmitis without evidence of periorbital or orbital involvement. The patient underwent a vitrectomy and was treated with intraocular vancomycin and ceftazadime. Two days after the surgery, inflammation of the eye persisted, but no evidence of systemic illness was found. Repeat vitrectomy was performed, and clindamycin and gentamicin were injected for treatment of suspected bacillus endophthalmitis; systemic penicillin G and clindamycin were started. Cultures of fluid inside the eye yielded *C. perfringens*. With treatment, the patient's infection resolved; however, he continued to have minimal light perception and retinal detachment and necrosis.

The second patient, a man aged 80 years, was determined on routine evaluation 1 day after surgery to have decreased visual acuity (20/400) and probable early endophthalmitis in the eye in which the cornea was transplanted. Infection progressed to severe endophthalmitis; however, he had no evidence of periorbital or orbital extension of the infection and no signs of systemic illness (Figure). Intraocular vancomycin and ceftazadime were administered. Two days after surgery, the patient's visual acuity had diminished to only light perception. The patient underwent an additional vitrectomy and was administered intraocular clindamycin and gentamicin with systemic clindamycin and penicillin G. Intraocular cultures also yielded *C. perfringens*. On follow-up, he recovered 20/200 vision, which was consistent with his preexisting maculopathy.

Cultures of both donor corneas, collected immediately before transplantation, subsequently grew *C. perfringens*. Review of data from the eye bank indicated that the donor body was refrigerated within 3 hours after death; eyes were recovered approximately 8 hours after death. The corneal tissues had undergone tissue processing as recommended by the Eye Bank Association of America (EBAA).[4] The donor tissue had been maintained in a solution of gentamicin and streptomycin, and transplantation was completed within 48 hours of tissue recovery. The eye bank and the surgeon had evaluated the donor tissue by slit lamp examination and found no abnormalities. No other tissues were recovered from this donor. Both cases were reported by the eye bank to EBAA as recommended.

Reported by: KG Banull, MD, JW Janelle, MD, Div of Infectious Diseases; WT Driebe, MD, Dept of Ophthalmology; LL Fauerbach, MS, Shands Hospital; L Archibald, MD, Div of Epidemiology, Univ of Florida, Gainesville. A Srinivasan, MD, D Jernigan, MD, Div of Healthcare Quality Promotion, National Center for Infectious Diseases, CDC.

CDC Editorial Note: This report describes the first reported cases of clostridial endophthalmitis associated with transplantation of contaminated corneal tissue. During 1991-2002, a total of 414,648 donor corneas were distributed for keratoplasty in the United States by EBAA-member eye banks.[5] Of 230 cases of culture-positive or clinically suspected microbial endophthalmitis among corneal transplant recipients reported during 1991-2002, no cases of endophthalmitis were reported to be caused by clostridia (EBAA, unpublished data, 2003). These data indicate that corneal transplantation in the United States has a very low risk for endophthalmitis.

Clostridial infections after implantation of contaminated allografts were first reported in 2001 among recipients of musculoskeletal tissues from cadaveric donors.[6] In that investigation, clostridia were recovered both from tissue recipients and from the donors of the tissues. Difficulties in detecting bacteria in postprocessing cultures led to release of the contaminated allografts. Cultures of the corneas collected immediately before implantation yielded *C. perfringens*, indicating that the tissue donor likely had disseminated *C. perfringens* disease. The donor's death was attributed to metastatic colon cancer; abdominal cancer is a known risk factor for *C. perfringens* bloodstream infection.[7] Neither cornea recipient acquired systemic infection; however, both had serious complications from infection, and one experienced substantial vision loss. The findings from this investigation underscore the serious infectious complications that can occur from transplanted allografts containing clostridia.

EBAA recommends that corneal tissue should be recovered by specially trained personnel using sterile technique.[4] Methods used by eye banks for processing corneal grafts include treatment with antimicrobials or bactericidal washes (e.g., povidone iodine)[8]; however, these methods do not inactivate spores. Corneas used for transplant are not sterilized because existing methods (e.g., irradiation) make the tissues unsuitable for transplant. Food and Drug Administration (FDA) regulations regarding corneal tissue address the medical suitability of donors and screening for infections caused by human immunodeficiency virus types 1 and 2, hepatitis C virus, and hepatitis B virus.[9] Neither FDA nor EBAA provide guidance specifically for detecting or inactivating clostridial spores on corneal allograft tissues.

Cultures of corneal tissue are not performed routinely by eye banks before a corneal transplant procedure. Eye banks may elect to perform presurgical (e.g., corneal-scleral rim) cultures, and positive culture reports should be reported to the receiving surgeon or recipient eye bank. Cultures may be performed either

before or at the time of surgery.[4] However, presurgical cultures might not reliably predict endophthalmitis complicating corneal transplantation.[10] For the two cases described in this report, culture results were not available early enough in the infection to prevent disease in recipients. If a corneal culture obtained at surgery identifies a pathogen, clinicians should evaluate the patient's condition promptly and consider initiation of appropriate therapy.

Metastatic colon cancer alone is not a factor that prompts deferral of a donor; however, the medical director should evaluate information about any potential donor with metastatic colon cancer to determine whether the donation should proceed. The risk for clostridial disease from corneas should be a consideration for tissue bank directors when evaluating potential donors with metastatic colon cancer. EBAA recommends that surgeons report adverse events, including cases of *C. perfringens* endophthalmitis, to eye banks and subsequently to EBAA within 30 days of the occurrence for review by a medical advisory board.[4] State health departments, CDC, and FDA should be notified to assist with investigations.

Acknowledgments

This report is based in part on data provided by EJ Holland, MD, KR Wilhelmus, MD, Eye Bank Association of America, Washington, DC.

REFERENCES

1. Crock WC, Wilson JH, Janakiraman P, Weiner JM. Gas gangrene infection of the eyes and orbits. Br J Ophthalmol 1985;69:143-8.
2. Nangia V, Hutchinson C. Metastatic endophthalmitis caused by *Clostridium perfringens*. Br J Ophthalmol 1992;76:252-3.
3. Romsaitong DP, Grasso CM. *Clostridium perfringens* endophthalmitis following cataract surgery. Arch Ophthalmol 1999;117:970-1.
4. Eye Bank Association of America. Medical Standards. Washington, DC: Eye Bank Association of America, 2003.
5. Eye Bank Association of America. 2002 Eye Banking Statistical Report. Washington, DC: Eye Bank Association of America, 2003.
6. CDC. Update: allograft-associated bacterial infections. MMWR 2002;51:207-10.
7. Bodey GP, Rodriquez S, Fainstein V, et al. Clostridial bacteremia in cancer patients: a 12-year experience. Cancer 1991;67:1928-42.
8. Pels E, Vrensen GF. Microbial decontamination of human donor eyes with povidone-iodine: penetration, toxicity, and effectiveness. Br J Ophthalmol 1999; 83:1019-26.
9. U.S. Food and Drug Administration. Human tissue intended for transplantation. Federal Register 1997; 62:40429-46.
10. Everts RJ, Fowler WC, Chang DH, Reller LB. Corneoscleral rim cultures: lack of utility and implications for clinical decision-making and infection prevention in the care of patients undergoing corneal transplantation. Cornea 2001;20:586-9.

Update: Creutzfeldt-Jakob Disease Associated With Cadaveric Dura Mater Grafts—Japan, 1979-2003

MMWR. 2003;52:1179-1181

2 figures omitted

IN 1997, A NONGOVERNMENT SURVEILlance group for Creutzfeldt-Jakob disease (CJD) in Japan supported financially by the Ministry of Health and Welfare* (MHW) reported 43 cases of CJD associated with receipt of cadaveric dura mater grafts.[1] In all but one case, the most probable vehicle of transmission was a single brand of dural graft (LYODURA® [B. Braun Melsungen AG, Melsungen, Germany]) produced before May 1987. As of March 2003, ongoing surveillance in Japan had identified an additional 54 dura mater graft–associated cases. This report summarizes the investigation of the 97 cases, which indicated that during 1983-1987, the estimated minimum risk for CJD within 17 years of receipt of the implicated product in Japan was approximately one case per 1,250 grafts. No cases have been reported among patients who received their first dural graft after 1991; however, because of the long latency period between graft placement and symptom onset, additional cases of graft-associated CJD are likely to be reported.

During 1996-2003, cases of CJD were identified in Japan by using (1) a mail survey of neurologic, psychiatric, and neuropathologic institutions (overall response rate: 74%)[1] and (2) subsequent reporting of CJD patients by clinicians to MHW. During this period, 97 cadaveric dura mater graft–associated CJD cases were identified. A case of dura mater–associated CJD was defined as a case in which a patient received a cadaveric dura mater graft and subsequently had CJD diagnosed by a physician and reviewed and accepted as CJD by a surveillance panel of neurologists.

The 97 CJD patients had illness onset during September 1985–April 2002 (Figure 1). Median age at onset was 58 years (range: 15-80 years); mean age was 55 years. Mean age at onset was younger than that reported for sporadic CJD in Japan (66 years). A total of 58 (60%) patients were female. Neuropathologic confirmation of CJD diagnosis was obtained for 20 (21%) patients; 65 (84%) of the other 77 patients with physician-diagnosed CJD had an electroencephalogram with a periodic synchronous discharge pattern consistent with CJD.

All 97 patients received dura mater grafts during 1978-1991 (Figure 2). Three patients received more than one dural graft during this period, including one patient reported previously.[1] In all three cases, the first graft was considered to be the source of infection. Medical conditions leading to the use of dural grafts in these patients included tumor (n=46), brain hemorrhage (n=14), Jannetta procedure for facial palsy (n=13) and for trigeminal neuralgia (n=six), intracranial aneurysm (n=eight), unspecified anomalies (n=five), hematoma (n=three), injury (n=one), and ossification of the spinal posterior longitudinal ligament (n=one).

Latency periods ranged from 14 months (receipt in 1987 and onset in 1989) to 275 months (receipt in 1978 and onset in 2001). The median and mean latency periods were 122 and 125 months, respectively. A total of 93 patients received dural grafts during 1978-1987. In 1987, the manufacturer revised collection and processing procedures for the implicated product to reduce the risk for CJD transmission. Four patients received grafts during 1988-1991. No cases have been reported among patients who received their first dural graft after 1991. A total