vCJD and dental treatment

Fiona Ord* and **Pauline Watt*** highlight the existing knowledge of a fatal disease and its possible association with dental treatment.

BSE and CJD

In the last 15 years variant CJD (vCJD) emerged in the UK and is thought to have been acquired primarily through consumption of BSE-contaminated meat products. CJD is a fatal disease with no immediate prospect of treatment. Abnormal prion proteins associated with vCJD are found outwith the central nervous system, raising concerns for possible transmission of prion proteins via invasive medical (including dental) procedures from persons in the asymptomatic phase of the disease. This article seeks to highlight the existing knowledge of vCJD, the distribution in oral tissues, the possible association with dental treatment and to raise awareness of current research.

History

vCJD is a type of human prion disease that was first identified in the UK in 1996. To date, there have been 166 definite/probable cases of vCJD in the UK. The main route of infection is likely to have been dietary exposure to beef products contaminated with bovine spongiform encephalopathy (BSE). Effective measures were put in place to protect the human food chain from BSE in the UK from 1996 onwards. Although the number of new cases has declined in recent years, it remains unclear how many individuals will develop vCJD as a result of the dietary route. Sporadic CJD has been transmitted by neurosurgical procedures and via contaminated growth hormone. While there have been four instances of blood transfusion transmission of vCJD in the UK, there has been no evidence, to date, of transmission via dental treatment, organ transplantation or surgical intervention. However, it is important to assess whether dental treatment is a potential route of transmission of vCJD, especially as animal studies in the UK have shown evidence of transmission of infectious prion proteins via the oral cavity.

Prions – what are they?

Prion proteins (PrP) may exist in two forms – the normal cellular form (PrP^{c}) and the abnormal disease associated isoform (PrP^{Sc}) Everyone has the normal type of prion protein within brain cells and in other cells of their body, although its function is unknown. Abnormal prion proteins have many unusual properties: enzymes are unable to break down PrP^{Sc} and it forms aggregates and deposits in the brain, sometimes in the form of amyloid that are thought to contribute to the damage of nerve cells in the brain of cases of CJD. PrP^{Sc} is also associated with infectivity and is resistant to inactivation by common decontamination methods. The infectious agent is therefore neither bacterial nor viral in nature, but is thought to be a rogue protein.

Inactivation of prions

Prion diseases have a variable incubation period depending on the type of disease. The duration of illness of vCJD patients can be over a year. The disease is fatal and, as yet, there is no known cure. A major infection control challenge is that the infectious prions are strongly resistant to the usual forms of bacterial and viral inactivation agents, such as disinfectants and steam sterilisation. Prions can survive autoclaving at 134°C for 18 minutes. However, prions are not readily transmissible by routine physical contact.

Prions and the oral cavity

In animal studies, abnormal prion proteins have been found in the trigeminal nerve, tooth pulp, gingival tissue, salivary glands, saliva and the tongue. Syrian hamsters showed infectivity in the trigeminal ganglion, dental pulp and gingival tissue after injection with a strain of scrapie.[1] Cattle exposed orally to BSE agent showed signs of infectivity in the trigeminal ganglion[2] and also in the trigeminal ganglion of sheep and hamsters with experimental scrapie.[2,3]

In vCJD, unlike other types of human TSEs, infective proteins may be present in tissues outside the central nervous system, for example the appendix and other lymphoreticular tissues.[4] Table 1 shows that positive immunochemistry has been detected in the trigeminal ganglion but not the cranial nerve or salivary gland tissues.[5]

In humans, the first patient known to have vCJD died in 1995 and to date (1 June 2008) there have been 166 'definite' or 'probable' vCJD cases in the UK (Table 2). The mortality trend in vCJD suggests that it has slowed but it is too early to know whether this remains or to assess the final size of the epidemic. Of concern is the potential that patients incubating vCJD or those with subclinical infection may receive dental treatment leading to the possibility of iatrogenic transmission from inadequately decontaminated dental instruments (Table 3).

Prions and dental treatment

The design and intricacy of many dental instruments makes them difficult to clean, therefore the ability to ensure dental instruments are free from debris is a key factor in reducing the possibility of tissue adhering on used instruments

and subsequent possible transmission.

A risk assessment by the Department of Health (2006) concluded that, given the existence of a carrier state, a self sustaining epidemic of vCJD was feasible, but employing single use 'files' and 'reamers' in endodontic treatment could eliminate the risk.[6] However, subsequent findings from a mouse model of vCJD infection has led the Spongiform Encephalopathy Advisory Committee (SEAC) to conclude that the potential risk of transmission of vCJD via a range of dental procedures may be greater than previously anticipated.[7]

Dental treatment and vCJD risk

A case control study carried out by the National Creutzfeldt-Jakob Disease Surveillance Unit (NCJDSU) examined the potential links between vCJD and dental treatment.[8]

This study initially examined information reported by relatives and found no statistically significant associations between dental treatment and vCJD. However, the study was limited to data reported from relatives. Therefore, there is a need to examine dental records to obtain more accurate information. A pilot study has demonstrated the feasibility of collating information from dental records. In an attempt to gain a clearer understanding of how vCJD may be transmitted, a study (funded by the Department of Health) is being carried out by the NCJDSU and the University of Glasgow Dental Hospital & School. This research follows the pilot study methodology for retrieving and recording information from dental treatment records.

The study will review dental treatment for all vCJD cases (n = 166) and controls (up to 650) to determine whether there are temporal or geographic links between vCJD cases, and to compare dental treatment in cases and controls. The study has been given ethical approval by a Multi-Centred Research Ethics Committee. Two dental hygienists will collect treatment information on a standardised data collection form by attending dental practices or requesting copies of full treatment histories. Partial or unavailable dental histories will be accessed through NHS Dental payment schemes. Unique identifiers (eg date of birth) will enable the researchers to access dental treatment for patients provided it was claimed for under the National Health Service.

What do I do?

If a patient presents for treatment and reports they have vCJD or are 'at risk', treatment may be carried out as usual. Presently, there are no additional precautions required for these patients provided optimal standards of infection control and decontamination are maintained. General advice on infection control can be obtained from the BDA (Advice Sheet A12), whilst instrument decontamination advice can be obtained from the local decontamination unit guidelines produced by Health Protection Scotland.[9] It would be prudent to check with your local infection control team to confirm that your instrument reprocessing procedures are indeed optimal.

Current guidance

Infection control guidelines in the UK are produced by the Advisory Committee on Dangerous Pathogens and the Spongiform Encephalopathy Advisory Committee (ACDP/SEAC). The CJD Incidents Panel with secretariat provided by the Health Protection Agency (HPA) provide advice on individual incidents. See www.hpa.org.uk/infections/topics_az/cjd/incidents_panel.htm.

Table 1 Infectivity in oral tissues from humans with vCJD

Tissue	Prion strain	Finding	Reference
Trigeminal ganglion	vCJD	Positive	Ironside *et al.* (2002)
Cranial nerves	vCJD	Negative	Ironside *et al.* (2002)
Salivary glands	vCJD	Negative	Ironside *et al.* (2002)

Table 2 Summary of vCJD cases in the UK since 1995

Deaths	
Deaths from definite vCJD (confirmed)	115
Deaths from probable vCJD (without neuropathological confirmation)	48
Deaths from probable vCJD (neuropathological confirmation pending)	0
Number of deaths from definite or probable vCJD	163
Alive	
Number of definite/probable vCJD cases still alive	3
Total number of definite or probable vCJD cases (dead and alive)	166

Table 3 Clinical concerns for iatrogenic infection of vCJD

Patient classification	Infectivity
Symptomatic, with clinical features of the disease	Incubation period unknown Possible infectivity through iatrogenic transmission
Asymptomatic/undiagnosed carriers • pre-clinical infection • sub-clinical infection	Incubating disease Possible infectivity through iatrogenic transmission Carriers that eventually develop disease (incubation period unknown) Carriers that will never develop disease symptoms

1. Ingrosso L, Pisani, F, Pocchiari M. Transmission of the 236K scrapie strain by the dental route. *J Gen Virol* 1999; **61:** 3235-3240.
2. Wells G A, Hawkins S A, Green R B, Austin A R *et al.* Preliminary observations on the pathogenesis of experimental bovine spongiform encephalopathy (BSE); an update. *Vet Rec* 1998; **142:** 103-106.
3. Groschup M H, Beekes M, McBride P A, Hardt M *et al.* Deposition of disease associated prion protein involves the peripheral nervous system in experimental scrapie. *Acta Neuropathol* 1999; **98:** 453-457.
4. Hilton D A, Fathers E, Edwards P, Ironside J W, Zajiceck J. Prion immunoreactivity in appendix before the clinical onset of variant Creutzfeldt-Jakob disease. *Lancet* 1998; **352:** 703-704.
5. Ironside J W, McCardle L, Horsburgh A, Lim Z, Head M W. Pathological diagnosis of variant Creutzfeldt-Jakob disease. *APMIS* 2002; **110:** 79-87.
6. Department of Health. Dentistry and vCJD: the implications of a carrier state for a self sustaining epidemic. 2006.
7. SEAC (2007) Position statement on vCJD and endodontic dentistry.
8. Porter S R. Dental treatment and risk of vCJD. *Br Dent J* 2007; **202:** E19.
9. The HPS local decontamination unit guidelines – www.hps.scot.nhs.uk

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