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研究報告の概要

○クロイツフェルトヤコブ病の遡及調査:最新報告

背景:英国は輸血により感染したと考えられる変異型クロイツフェルト・ヤコブ病(vCJD)を3例報告しており、一番最近の症例は2006年に発症している。本試験は、1995年に米国の大規模血液供給システムならびに米国血液センターと共同でCJD供血者・受血者の特定を行っている米国疾病対策予防センター(CDC)により開始された。

目的:本試験の目的は、血液製剤の輸血による古典的CJDの伝播リスクの分析および特定を支援することである。

方法:米国血液センターや家族からの報告により、供血後に古典的CJDを発症した個人を特定した。血液センターは血液成分を受領した病院を探し出し、該当病院は血液成分の受血者を特定した。受血者は、国民の死亡索引(National Death Index、NDI)および必要であれば他のデータベースを用いて追跡を行った。受血者のフォローアップ調査は年1回ベースで行い、死亡(特にCJD関連死)についてNDIデータベースを検索し、死亡証明書の調査もいくつか行った。

結果:2006年2月までに、古典的CJDで死亡した供血者31名が試験に登録された。384名の受血者が特定され、合計1790人年の追跡を行った。1790人年の内訳は、生存者100名が1150人年、死亡者273名が606人年、追跡不能者11名が34 人年であった。輸血を受けてから5年以上生存した受血者は138名であった。これらの長期生存者の追跡は、計1569人年であった。138名中93名は最新報告時も生存し(1142人年)、42名は死亡(404人年)、3名は追跡不能であった(23人年)。長期生存者138名のうち7名は輸血後21年以上生存した。NDI調査によれば、2003年まではCJD発症患者はいない。さらに2名CJDの供血者が報告され、調査中である。

結論:現在実施中である本試験は、古典的CJDが輸血を介して受血者に伝播したという証拠を示さなかった。

報告企業の意見	今後の対応
供血後に古典的CJDを発症した個人を特定し、受血者の追跡調査を行ったところ、古典的CJDが輸血を介して受血者に伝播したという証拠は示されなかったとの報告である。	今後も引き続き、プリオン病に関する新たな知見及び情報の収集に努める。

使用上の注意記載状況・その他参考事項等

人全血液CPD「日赤」
照射人全血液CPD「日赤」
人全血液-LR「日赤」
照射人全血液-LR「日赤」

血液を介するウイルス、細菌、原虫等の感染
vCJD等の伝播のリスク

Results: In a total of 1728 blood samples from university or technical school students, 14 samples (0.8%) were found positive for HBsAg, whereas 75 samples (4.34%) were positive for anti-HBc. 15 (20%) out of these anti-HBc positive samples contained very low titre of anti-HBs (<100mIU/ml) and 14 serum samples (18.67%) had no protective antibodies against HBV and were characterized 'anti-HBc-only'. From the group of immigrants, 3 donors were HBsAg positive and totally 37 serum samples tested positive for anti-HBc. 12 samples out of the 37 anti-HBc positive (32.43%) were described as 'anti-HBc-only', since they had no anti-HBs antibodies. All blood components having the 'anti-HBc-only' profile were discarded.
Conclusions: It has previously been described that HBV can be transmitted by anti-HBc-only blood components or organs. Preliminary data of this study suggest that the percentage of anti-HBc-only donors in Greece appears to be increased, especially among the group of immigrants, signifying that immigration from less to more developed countries could contribute to reemergence of a number of infectious diseases. Experiments are underway to determine the presence of HBV DNA in these serum samples, verifying the infectivity of the blood components.

01.3 Blood Safety - TTD - TSEs

P-086
HIGH RATE OF VCJD TRANSMISSION IN MICE BY INTRAVENOUS INOCULATION OF WHOLE BLOOD

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Background: Variant Creutzfeldt-Jakob disease (vCJD), a newly emerged disease resulting from animal-to-human transmission though dietary exposure to bovine spongiform encepahlopathy, belongs to the group of transmissible spongiform encephalopathies (TSE)/prion diseases. Patients with vCJD accumulate TSE infectivity and disease-associated prion protein (PrPd) in lymphoreticular tissues more extensively than patients with classical CJD. These findings indicate that blood may also carry higher levels of infectivity. Recently, three cases of possible iatrogenic transmission of vCJD through blood transfusion have been reported in the UK, increasing the concern about safety of blood.
Aims: The present study is designed to elucidate the transmission of vCJD by intravenous (i.v.) inoculation of whole blood using a mouse model.
Methods: Swiss mice were inoculated intracerebrally (i.c.) with mouse-adapted vCJD agent. When mice developed clinical disease, they were euthanized and their blood was collected, pooled and inoculated i.v. (0.1 ml per mouse) into an experimental group of 19 mice. A sample of that blood was also diluted 1:4 in physiological saline and inoculated i.c. (30 ul per mouse) into 7 additional mice. Appropriate controls received i.v. blood from healthy animals. Brains and spleens of deceased and euthanized animals were examined for the presence of PrPd using western blotting.
Results: In the group of mice that received i.v. blood from vCJD infected animals, eight out of 19 mice were confirmed to be positive for infection by western blotting: five mice developed clinical signs of the diseases after 260 days and three died earlier from other causes. None of the mice in the i.c. inoculated group or control group developed clinical disease even after being followed up for more than 2 years after inoculation.
Summary and Conclusion: We previously have shown that at the clinical phase of the disease, blood of mice i.c. inoculated with vCJD contains low levels of TSE infectivity, which is present in buffy coat and plasma but not in RBC. We also showed that infection is efficiently transmitted by the i.c. route and the i.v. route both for buffy coat and for platelet poor plasma. In this study using a mouse model of vCJD, we showed that the disease is transmitted at a high rate by i.v. inoculation of whole blood from animals infected with vCJD. Absence of infections in a small group of animals inoculated i.c. with the same but diluted blood sample points to the limitations of an animal model when small numbers of animals are used in bioassay of samples with low levels of infectivity.

P-087
PERFORMANCE CHARACTERISTICS OF PALLS ENHANCED LEUKOTRAP AFFINITY PRION REDUCTION FILTER

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Background and Objectives: Three recent probable cases of transmission of a variant of human Creutzfeldt-Jakob Disease (vCJD) through blood transfusion suggest that the disease can be transmitted through transfusion of blood components from pre-symptomatic blood donors. In the present study, we investigated the performance of a new filter for reducing the levels of infectious prions (PrPSc) in red cell concentrates (RCC).
Materials and Methods: Endogenous Infectivity: A pool of 500mL of whole blood was collected from 263K-strain scrapie-infected hamsters into CPD anticoagulant, processed into either leucocyte-reduced (LR-RCC) or non-leucoreduced RCC (NL-RCC), and then passed through a prion reduction filter. Pre- and post-filtration samples were tested for PrPSc by Western blot and infectivity by inoculation of healthy hamsters Exogenous (Spiking)
Study: Different preparations of scrapie infected hamster brain homogenates containing PrPSc (clarified suspension, microsomal and pure forms) were added to human RCC and then filtered. Levels of PrPSc were determined by Western blot assay. The effects of different red cell preparative procedures on prion removal efficiency of the filter were evaluated. In addition, the effect of prior leucodepletion of 'spiked' RCC on PrPSc removal by the prion removal filter was also assessed.
Results: In the exogenous (spiking) study, the levels of all the different forms of PrPres were significantly reduced in RCC by about 2.5 to 3.7 logs, $p<0.05$. Prior leucodepletion of the RCC with a leucoreduction filter did not significantly reduce the concentration of exogenously spiked PrPSc, $p>0.05$.
Conclusion: The use of this new prion reduction filter should reduce the risk of vCJD transmission through transfusion of RCC, the most widely transfused blood component.

P-088
CREUTZFELDT-JAKOB DISEASE LOOK-BACK STUDY: AN UPDATE

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Background: The UK has reported three cases of probable transfusion-transmission of variant Creutzfeldt-Jacob disease (vCJD), the most recent case occurring earlier this year (2006). The present study was initiated in 1995 by a large US blood supply system and the Centers for Disease Control and Prevention (CDC), who work in collaboration with US blood centers to locate and identify CJD blood donors and

their recipients.
Aims: The aim of this study is to help assess and define the potential risk of transmission of classic CJD by transfusion of blood products.
Methods: Individuals who donated blood and subsequently developed classic CJD were identified by reports from US blood centers and family members. Blood centers located the hospitals that received the blood components and the hospital identified the recipients of those components. Tracking of these recipients is carried out by using the National Death Index (NDI) and, if necessary, other databases. Follow-up of the recipients occurs on an annual basis by querying the NDI database for deaths, particularly CJD-related deaths, and reviewing some death certificates.
Results: By February 2006, 31 blood donors who died of classic CJD were enrolled in the study. A total of 384 recipients were identified and followed for a total of 1,790 person-years (py). Of the latter, 1,150 py were from 100 survivors, 606 py were from 273 deceased recipients, and 34 py were from 11 recipients who were lost to follow up. 138 recipients survived 5 or more years after their transfusion. These long-term survivors were followed for a total of 1,569 py. Ninety-three of these 138 persons were still alive at last report and accounted for 1142 py, 42 were deceased (404 py) and 3 were lost to follow up (23 py). Seven of these 138 long term survivors lived 21 years or more after their transfusion. (Table 1) Through 2003, based on the NDI search, none of the recipients developed CJD. Two additional CJD blood donors were reported and are under investigation
Conclusions: This ongoing study shows no evidence that classic CJD was transmitted to the recipients through blood transfusion.

Table 1: Summary of Long-Term Survivors

Recipients	Deceased	Survivors	Lost to Follow Up	Total
5-10 Years	34	41	3	78
11-15 Years	4	37	0	41
16-20 Years	3	9	0	12
21+ Years	1	6	0	7
Total	42	93	3	138
Person-Years	403.5	1142.25	23.25	1569

P-089
ABILITY OF THE MACOPHARMA PRION CAPTURE (P-CAPT(TM) FILTER TO REMOVE BRAIN PRPRES IN LEUKOREDUCED HUMAN RBC

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Background: Transmissible spongiform encephalopathy (TSE) diseases, also known as prion diseases, are neurodegenerative illnesses that can be transmitted by the transfusion of infected blood and blood products. Blood-borne TSE infectivity can harbor undetected for years in the blood of an asymptomatic variant Creutzfeldt-Jakob disease (vCJD) patient who, during this period, could donate blood. Precautionary measures against the spreading of TSE through the blood supply have been implemented, but alone those measures are not sufficient.
TSE pathogen removal is probably one of the most achievable options to date to better safeguard the blood supply. Pathogen Removal and Diagnostic Technologies Inc. (PRDT) developed a strategy in which resins that best adsorbed the TSE causative agent from blood were selected. One resin was shown to capture brain PrPres in red blood cell concentrate (RBC), whole blood, and plasma. The same resin also reduced brain-derived scrapie infectivity by approximately 4 log(10) and more recently we have demonstrated that the resin captured all detectable endogenous infectivity from hamster whole blood as measured by the bioassay. The resin was incorporated into membrane layers that were developed by MacoPharma into a prion capture filter termed P-CAPT(TM).
Aim: To investigate the performance of the P-CAPT(TM) filter to capture PrPres from scrapie infected hamster brain homogenate mixed in leukoreduced human RBC as assessed by Western blot analysis.
Methods: Hamster brain homogenate infected with the 263K strain of scrapie was added to a unit of leukoreduced (LST1 MacoPharma whole blood filter) human RBC. The unit bag was sterile docked to a P-CAPT(TM) kit containing an in-line P-CAPT(TM) filter and a transfer bag to collect the treated RBC. The spiked RBC was applied to the P-CAPT(TM) filter by gravity at ambient temperature according to the manufacturer's instructions. At the end of the run, the filter was extensively washed until most of the red blood cells were removed. The proteins captured by the filter were eluted for Western blot analysis using 3F4 antibody to specifically detect PrP. In some studies, two P-CAPT(TM) filters in series were tested to investigate the level of PrPres removal provided by the filter. Other parameters such as reproducibility of binding and filtration time were also analyzed.
Results: Western blot analysis of the filter-bound proteins indicated that the P-CAPT(TM) filter captured brain PrPres from a unit of human leukoreduced RBC. The binding was reproducible within the experimental variability of the Western blot signal and the filtration time was also reproducible. Additional studies also indicated that the input PrPres was not detected adsorbed in a second, in series filter.
Conclusions: P-CAPT(TM) filters contain a resin that, when tested in column format, captured brain PrPres and brain-derived infectivity as well as endogenous hamster blood infectivity. The studies here investigated whether the same resin incorporated into a filter was still capable of capturing brain PrPres. The results indicated that the resin maintained those properties under the current new format. Furthermore, one P-CAPT(TM) filter removed all PrP input to the limit of detection of the Western blot.

P-090
IN VITRO EVALUATION OF RED CELL CONCENTRATES AFTER FILTRATION OVER THE NEW COMPOSAFE PR SYSTEM (SYSTEM FOR PRION REDUCTION FROM LEUKOCYTE DEPLETED RCC)

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Background: Although the risk for contamination of RCC with prions is unknown, several companies are developing filters for prion reduction of blood components. The prion filter for RCC recently developed by Pall (LAPRF), was combined with leukodepletion in a Fresenius whole blood system and tested for the in vitro quality of RCC during storage for up to 42 days.
Methods: Whole blood was collected in standard Fresenius in line systems with integrated whole blood filter. The whole blood was either leukoreduced after overnight storage at 22°C (group I; n=8) or within 4 h (group II; n=8) and subsequently separated into plasma and RCC in SAGM. For group I the RCC units were immediately filtered over the prion filter (Composafe Pr) and for group II the RCC were filtered over the prion filter after overnight storage at 4°C. After filtration over the prion filter all RCC were stored at 4°C and sampled at various time points during storage.
Results: The CompoSafe Pr filter had no effect on the total amount of protein in the supernatant of the RCC, but removed factor IX from the supernatant (before 0.14 IE/ml, after < 0.01 IE/ml) which has been indicated as a pseudomarker for prion removal. The RCC in both groups contained 54 ± 4 g Hb (mean±SD), with a combined loss due

医薬品 研究報告 調査報告書

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研究報告の概要

○臓器移植後のシャーガス病ーロスアンジェルス、カリフォルニア、2006年
ロスアンジェルスの心臓移植患者2名で、今年の初めに臓器移植によるシャーガス病伝播が見られた。米国における固形臓器移植によるTrypanosoma cruzi伝播としては4例目と5例目になる。
症例1:2005年12月、64歳の男性が心臓移植を受け、1月に拒絶反応を疑われて強い免疫抑制療法を受けた。2月に食欲不振、発熱、下痢で再入院した。血液培養ではT. cruzi陽性となり、心内膜心筋の生検では無鞭毛型が検出された。血清学検査はT. cruzi抗体陰性、PCRではDNA陽性であり、最近感染したことが示された。治療によって寄生虫血症は解消されたが、移植臓器の拒絶反応で4月に死亡した。遡及調査を行ったところ、臓器ドナーは米国生まれだがメキシコのT. cruzi汚染地域に渡航したことがあった。血清学検査の結果は、RIPAでT. cruzi抗体陽性、IFAで弱陽性だった。これに加えて3人の患者が肝臓と両腎臓を移植されていた。これらの患者はT. cruzi血清陰性でPCRでも寄生虫血症は確認されなかった。
症例2:2006年1月、73歳の男性が心臓移植を受け、2月に発熱、倦怠感、腹部の発疹で再入院した。血液検査ではT. cruzi陽性だった。患者は6月に死亡した。主な死因は心不全で、剖検は行われなかった。他の3人の臓器移植患者では血清学検査陰性でPCRで寄生虫血症は確認されなかった。ドナーはエルサルバドル生まれで死亡時はカリフォルニア在住であり、血清学検査はRIPAで陽性、IFAで陰性だった。いずれのドナーも供血の記録は確認されなかった。

報告企業の意見	今後の対応
ロスアンジェルスの心臓移植患者2名で、今年の初めに臓器移植によるシャーガス病伝播が見られたとの報告である。	日本赤十字社は、輸血感染症対策として献血時に海外渡航歴の有無を確認し、帰国後4週間は献血不適としている。また、シャーガス病の既往がある場合には献血不適としている。今後も引き続き情報の収集に努める。

使用上の注意記載状況・その他参考事項等

解凍赤血球濃厚液「日赤」
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血液を介するウイルス、
細菌、原虫等の感染
vCJD等の伝播のリスク

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MMWR

Weekly

July 28, 2006 / 55(29);798-800

Chagas Disease After Organ Transplantation --- Los Angeles, California, 2006

Chagas disease is an infection caused by the parasite *Trypanosoma cruzi*. Reduviids (i.e., "kissing bugs") transmit the parasite through infected feces. *T. cruzi* also can be transmitted congenitally and through blood transfusion or organ transplantation. The infection is lifelong if left untreated; the majority of infected persons are asymptomatic, and their disease remains undiagnosed. Although routine serologic testing of organ and blood donors is performed in areas of Latin America where Chagas disease is endemic, no *T. cruzi* screening test is licensed in the United States. However, seroprevalence studies using research tests have documented the presence of *T. cruzi* antibodies in U.S. blood (*1*) and organ donor populations (*2*). This report describes two cases of acute Chagas disease in heart transplant recipients reported by two Los Angeles County hospitals in February 2006. In the United States, one previous report documented *T. cruzi* transmission through solid organ transplantation, in which three organ recipients were infected (*3*).

Case Reports

Case 1. In December 2005, a man aged 64 years with idiopathic cardiomyopathy received a heart transplant. In January 2006, he was treated with enhanced immunosuppression for suspected organ rejection. In February 2006, he was readmitted to the hospital with anorexia, fever, and diarrhea of 2 weeks' duration. A peripheral blood smear revealed *T. cruzi* trypomastigotes, blood cultures were positive for *T. cruzi*, and endomyocardial biopsy specimens contained amastigotes. The patient was interviewed about natural exposures, and organ procurement and transplantation records were reviewed. He had no identifiable risk factors for *T. cruzi* infection (e.g., travel to a country endemic for Chagas disease). He was seronegative for *T. cruzi* antibodies but positive for *T. cruzi* DNA by polymerase chain reaction (PCR), indicating recent infection. After initiation of nifurtimox therapy, his parasitemia rapidly cleared. However, in April 2006, the patient died from complications attributed to acute rejection of the transplanted organ.

To identify the source of infection, a traceback was conducted on all blood products transfused to the heart donor and recipient. All available blood donors tested negative for *T. cruzi* antibodies by immunofluorescence assay (IFA) and radioimmunoprecipitation assay (RIPA). However, blood from the organ donor tested seropositive for *T. cruzi* antibodies by RIPA and tested borderline-positive by IFA. The organ donor had been born in the United States but had traveled to a *T. cruzi*–endemic area of Mexico.

Three additional patients received a liver and both kidneys from the same donor. These patients are *T. cruzi*–seronegative by IFA and have no evidence of parasitemia by PCR. They continue to be monitored.

Case 2. In January 2006, a man aged 73 years with ischemic cardiomyopathy received a heart transplant. The patient was readmitted to the hospital in February 2006 with fever, fatigue, and an abdominal rash. A thin blood smear revealed *T. cruzi* trypomastigotes, and blood cultures were positive for *T. cruzi*. Organ procurement and transplantation records were reviewed. The patient had no identifiable risk factors for *T. cruzi* infection. He was seronegative but PCR-positive for *T. cruzi*, indicating recent infection.

The patient's rash and parasitemia resolved after 10 days of nifurtimox treatment. Serial endomyocardial biopsies did not reveal trypanosomes, and he remained seronegative by IFA for *T. cruzi*. The patient died in June 2006. The primary cause of death was cardiac failure; no autopsy was performed.

he source of infection was investigated with the same methods used for case 1. All available blood donors tested ɛronegative for *T. cruzi*. The organ donor, who had been born in El Salvador and was residing in Los Angeles at ıe time of his death, tested positive for *T. cruzi* antibodies by RIPA but had a negative IFA. Three other patients ɛceived solid organs from the same donor. These patients are *T. cruzi*-seronegative by IFA and have no evidence f parasitemia by PCR. They continue to be monitored. No record of previous blood donations by either organ ɔnor was found.

eported by: *L Mascola, MD, Acute Communicable Disease Control Program, Los Angeles Dept of Health Svcs; Kubak, MD, Univ of California; S Radhakrishna, MD, Univ of Southern California; T Mone, One Legacy, Los ngeles; R Hunter, California Dept of Health Svcs. DA Leiby, PhD, American Red Cross, Rockville, Maryland. M uehnert, MD, Div of Healthcare Quality Promotion, National Center for Infectious Diseases; A Moore, MD, F 'eurer, MS, G Lawrence, MPH, Div of Parasitic Diseases, National Center for Preparedness, Detection, and ontrol of Infectious Diseases (proposed); H Kun, ScD, EIS Officer, CDC.*

ditorial Note:

he two cases described in this report are the fourth and fifth cases of reported *T. cruzi* transmission through solid gan transplantation in the United States. The prevalence of infection with *T. cruzi* in the United States varies by gion and might now be higher than previously thought, especially in geographic areas such as Los Angeles ounty, where a substantial proportion of blood and organ donors have emigrated from Chagas-endemic countries ecause organ donors frequently receive blood transfusions, infection can be transmitted to recipients either by ınsfusion or transplant. Currently, no policies recommend laboratory screening for *T. cruzi*. Diagnostic tests ailable for research studies have variable sensitivities and specificities, and no licensed screening test exists.

ıysicians and laboratorians should maintain a high index of suspicion for *T. cruzi* infection in transplant and ınsfusion recipients who exhibit complications of an unknown etiology when more common sources have been ɛcluded. Acute Chagas disease in severely immunocompromised patients is of special concern because the inical course is often severe and rapidly progressive. If Chagas is suspected, manual microscopic examination of ɛripheral blood smears should be performed. Patients with acute Chagas disease should be treated as early as ɔssible in the course of the infection. Available treatments include nifurtimox (available from CDC Drug Service, lephone 404-639-3670) or benznidazole (only distributed outside of the United States).

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