Figure 1

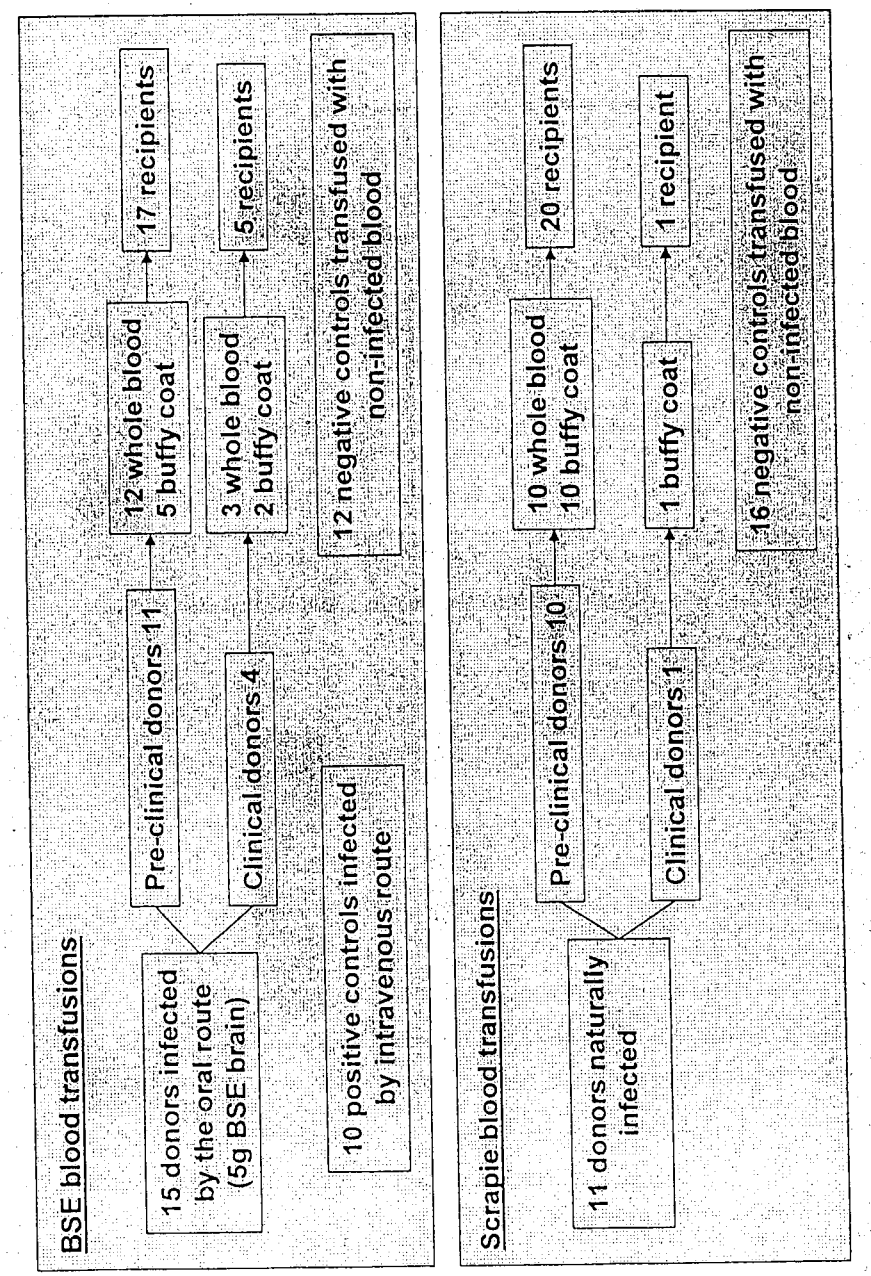
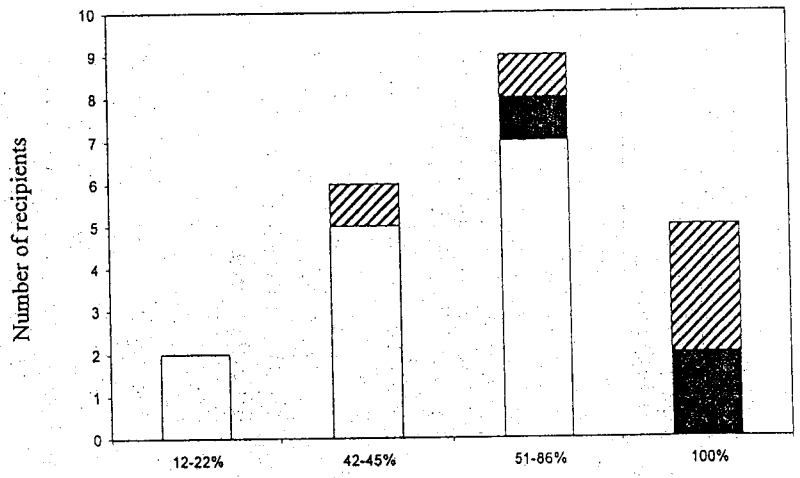


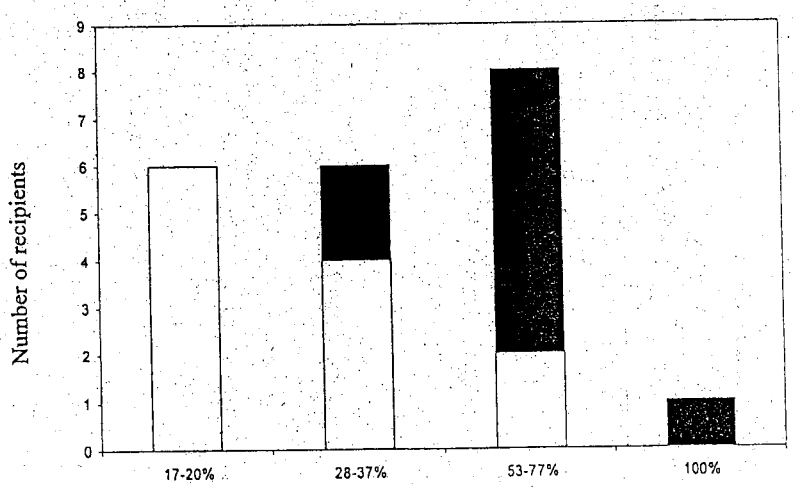
Figure 2.

A.



Donor – estimated percentage of incubation period at donation

B.



Donor – estimated percentage of incubation period at donation

医薬品 研究報告 調査報告書

識別番号・報告回数			報告日	第一報入手日	新医薬品等の区分	総合機構処理欄
				2008. 10. 17	該当なし	
一般的名称	人全血液		研究報告の公表状況		公表国	
販売名(企業名)	人全血液-LR「日赤」(日本赤十字社) 照射人全血液-LR「日赤」(日本赤十字社)			Morgan AE. Am J Infect Control. 2008 Oct;36(8): 602.	米国	

研究報告の概要

○耳鍼による緑膿菌感染
両耳用置き鍼治療(Stapling)は、効果的な減量法としてメディアで大きく取上げられている。鍼師は食欲抑制を目的として耳介軟骨の「つぼ」に鍼を留置する。現在多くの保険会社が鍼治療を保険適用にしている。
2週前に鍼治療院を訪れ両耳軟骨の置き鍼治療を受けた病歴のない16歳の女性は、左耳の鍼周囲の紅斑および圧痛がみられた。鍼を除去し、アモキシリン・クラブラン酸の経口投与を行ったが、1週間後、紅斑および圧痛が進行し膿瘍が現れた。ドレナージ検体を培養と感受性試験に供した。もう片耳の鍼も除去し排膿を認め検体を採取した。試験の結果が得られるまで、トリメトプリム・スルファメトキサゾール(TMP/SMX)の経口投与を行った。両耳で著しい緑膿菌の生育が認められたため、シプロフロキサシンの経口投与を行い、治療21日目に完全消失となった。
外耳軟骨は、血流に乏しく特に感染しやすい。さらに、鍼刺による周囲軟骨膜の破損は、耳軟骨の完全性に損傷を与える可能性がある。耳介軟骨炎で最も一般的な感染は、黄色ブドウ球菌と緑膿菌によるものである。緑膿菌は治療が困難であり、長期入院や再建手術を要する重度感染を引き起こす場合がある。
減量のための耳鍼は非常に人気のある方法になりつつあるが、患者はプラセボ効果の可能性と感染のリスクを考慮すべきである。もっとも重要なことは、耳鍼が危険な緑膿菌感染を起こす可能性があることを医師が認識することである。

使用上の注意記載状況・その他参考事項等

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

報告企業の意見	今後の対応
減量法として両耳用置き鍼治療(Stapling)を受けた女性の鍼周辺に緑膿菌が感染したとの報告である。	日本赤十字社は、細菌・ウイルス等の血液を介する感染防止の目的から、献血時にピアスについて確認し施術後1ヵ月ないし1年間献血延期としている。鍼治療についても申告があった場合は「鍼治療における感染防止の指針」に準拠していることを確認し、そうでない場合は1年間献血延期としている。今後も細菌感染に関する新たな知見及び情報の収集に努める。

(17)

MedDRA/J Ver.11.0J

Pseudomonas aeruginosa infection due to acupunctural ear stapling

To the Editor:

Bilateral ear stapling is widely advertised in the media (including the Internet) as a popular and successful weight reduction strategy. Acupuncture providers performing the technique place staples into ear cartilage "reflex points" to decrease craving.[1] Many insurance carriers now provide coverage for most acupuncture treatments.

A 16-year-old female with no medical history presented with a complaint of external ear pain. Two weeks earlier, she visited an acupuncture parlor, where she underwent bilateral ear stapling of her upper ear cartilage to induce weight loss. Examination revealed erythema and tenderness around the left ear staple. The staple was removed, and the patient was placed on oral amoxicillin/clavulanic acid. One week later, the erythema and tenderness had progressed, and an abscess was present. The lesion was drained, and a specimen of the drainage was sent for culture and sensitivity testing. At this time, the staple on the other ear was removed, and pus drainage was identified and collected. The patient was placed on oral trimethoprim/sulfamethoxazole (TMP/SMX) pending culture and sensitivity results.

Laboratory evaluation subsequently revealed heavy growth of *Pseudomonas aeruginosa* on both ears. The patient was placed on oral ciprofloxacin. Complete resolution occurred after 21 days of treatment.

The cartilage of the external ear is particularly vulnerable to infection due to its limited blood supply. In addition, disruption of the surrounding perichondrium due to stapling can damage ear cartilage integrity. The most common infectious agents in auricular chondritis are *Staphylococcus aureus* and *P aeruginosa*.[2] In this case, the patient failed a 1-week course of amoxicillin/clavulanic acid, which is highly effective against methicillin-sensitive *S aureus*. Due to the high prevalence of methicillin-resistant *S aureus* skin infections,[3] the patient was started on TMP/SMX before laboratory testing confirmed the *P aeruginosa* infection. *P aeruginosa* can be particularly difficult to treat because of its high resistance to oral antibiotic regimens.[4] In addition, auricular chondritis due to this organism can cause severe infection, necessitating prolonged hospitalization and reconstructive surgery.[4]

Studies on ear stapling have demonstrated that patients who strictly monitor their daily food consumption experienced comparable weight loss to those who undergo ear stapling.[5] Another study requiring patients to wear a simple wrist device to remind them of their dietary restrictions found comparable weight loss to ear stapling.[6] These studies indicate that the presence of an ear staple may have a placebo effect and that the increased attention to daily food consumption, possibly through daily logging, is actually responsible for the enhanced weight loss.

Ear stapling for weight loss is becoming an increasingly popular modality. The possibility of a placebo effect and the risk of infection should be considered in a patient's decision to receive the treatment. Most importantly, physicians should be aware that acupunctural ear stapling can cause dangerous *P aeruginosa* infection.

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doi:10.1016/j.ajic.2007.07.014

Hand hygiene in Iranian health care workers

To the Editor:

Hand hygiene (HH) remains the single most important measure to prevent nosocomial infections.[1] Despite universal awareness of HH role in reducing nosocomial infection, compliance among health care

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医薬品　研究報告　調査報告書

識別番号・報告回数			報告日	第一報入手日 2008年10月24日	新医薬品等の区分 該当なし	総合機構処理欄
一般的名称	別紙のとおり		研究報告の公表状況	MMWR. 2008;57:1145-1148	公表国 米国	
販売名（企業名）	別紙のとおり					

研究報告の概要	使用上の注意記載状況・その他参考事項等
問題点：輸血によるアナプラズマ症感染事例。過去に輸血によるアナプラズマ症の報告はあったが、本症例は血液ドナーに感染源が確認された初の事例。 2007年11月、入院中のミネソタ州住民が *Anaplasma phagocytophilum* に感染しているとの報告を受けた。患者は68歳男性、慢性腎不全、乾癬性関節炎、強直性脊椎炎の既往があり、ステロイド投与を受けていた。入院する3週間前にマダニのいる地域へ旅行したが、咬まれたかどうかは不明である。2007年10月12日、膝関節形成術および滑膜切除術が行われたが、数時間後に手術部位から出血、INRおよびPPT上昇を伴う凝固障害を来たし、フィブリノーゲンおよび血小板数が減少、外科処置と輸血が行われた。10月12〜21日、赤血球34単位、血小板4単位、新鮮凍結血漿14単位、寒冷沈降物7単位が輸血され、19日、敗血症および多臓器不全をきたし、セファゾリン、ピペラシリン/タゾバクタム、バンコマイシン、レボフロキサシンが投与された。10月18、20、31日の血液培養、19、25日の尿培養検査はいずれも陰性であった。31日、血小板減少が進行(31日:178,000/mm³、11月5日:54,000/mm³)、翌11月1日には低血圧、尿路感染症による発熱を来たし、レボフロキサシンとST合剤が投与された。入院22日目(11月3日)、末梢血塗抹検体から *A. phagocytophilum* の桑実胚が認められ、11月3〜5日のPCRによるDNAアッセイにて *A. phagocytophilum* が確認され、CDCによりIgG抗体陽性も確認された。11月5日よりドキシサイクリンが投与され、血小板数は回復、10日には163,000/mm³となり、13日にリハビリ病棟へ移動、12月3日に退院した。この患者に輸血された血液ドナー(59名)の調査を行ったところ、64歳女性の血液がPCR、IFA検査により *A. phagocytophilum* 陽性と確認されたが、この女性は献血の前後1ヵ月間、発熱などの症状は認めていなかった。輸血後の発熱を伴う急性血小板減少症は、アナプラズマ症の可能性を考慮し、輸血による感染の疑いを州や地方の保険局に報告すべきと考える。	記載なし

報告企業の意見	今後の対応
別紙のとおり	今後とも関連情報の収集に努め、本剤の安全性の確保を図っていきたい。

MedDRA/J ver.11.1

別紙

一般的名称	①人血清アルブミン、②人血清アルブミン、③人血清アルブミン*、④人免役グロブリン、⑤乾燥ペプシン処理人免疫グロブリン、⑥乾燥スルホ化人免疫グロブリン、⑦乾燥スルホ化人免疫グロブリン*、⑧乾燥濃縮人活性化プロテインC、⑨乾燥濃縮人血液凝固第Ⅷ因子、⑩乾燥濃縮人血液凝固第Ⅸ因子、⑪乾燥抗破傷風人免疫グロブリン、⑫抗HBs人免疫グロブリン、⑬トロンビン、⑭フィブリノゲン加第ⅩⅢ因子、⑮乾燥濃縮人アンチトロンビンⅢ、⑯ヒスタミン加人免疫グロブリン製剤、⑰人血清アルブミン*、⑱人血清アルブミン*、⑲乾燥ペプシン処理人免役グロブリン*、⑳乾燥人血液凝固第Ⅸ因子複合体*、㉑乾燥濃縮人アンチトロンビンⅢ
販売名（企業名）	①献血アルブミン20“化血研”、②献血アルブミン25“化血研”、③人血清アルブミン“化血研”*、④“化血研”ガンマーグロブリン、⑤献血静注グロブリン“化血研”、⑥献血ベニロンーⅠ、⑦ベニロン*、⑧注射用アナクトC2,500単位、⑨コンファクトF、⑩ノバクトM、⑪テタノセーラ、⑫ペバトセーラ、⑬トロンビン“化血研”、⑭ボルヒール、⑮アンスロビンP、⑯ヒスタグロビン、⑰アルブミン20%化血研*、⑱アルブミン5%化血研*、⑲静注グロブリン*、⑳ノバクトF*、㉑アンスロビンP1500注射用
報告企業の意見	アナプラズマ症はマダニにより媒介される発熱性疾患で、その病原体は顆粒球に特異的に感染する0.2〜2μmの大きさの球状もしくは楕円状の偏性寄生性のグラム陰性桿菌である。1994年、米国で発熱性疾患患者の好中球の中にエーリキア様細菌の感染が認められ、ヒト顆粒球エーリキア症病原体［Human Granulocytic Ehrlichiosis (HGE) agent］と呼ばれるようになった。その後、1996年にはその病原体が分離報告され、さらに2001年にはEhrlichia属からAnaplasma属へと配置換えされて、*Anaplasma phagocytophilum* という学名が付された。それに伴って、昨今ではその病名もヒト顆粒球アナプラズマ症［Human Granulocytic Anaplasmosis (HGA)］と呼ばれている。*A. phagocytophilum* は、ヒトの他、ウマやヒツジなどにも感染し、アナプラズマ症を引き起こすことから「人獣共通感染症」病原体としても知られている。(http://idsc.nih.go.jp/iasr/27/312/dj312d.html) *A. phagocytophilum* によるアナプラズマ症の発生は欧米が中心であるが、2006年に日本においても *A. phagocytophilum* がマダニから検出されたことが初めて報告された。 弊所で製造している全ての血漿分画製剤の製造工程には、約0.2μmの「無菌ろ過工程」および、*A. phagocytophilum* よりも小さいウイルスの除去を目的とした平均孔径19nm以下の「ウイルス除去膜ろ過工程」が導入されているので、仮に製造原料に *A. phagocytophilum* が混入していたとしても、これらの工程により除去されるものと考えられる。更に、これまでに本剤によるアナプラズマ症感染の報告例は無い。 以上の点から、本剤はアナプラズマ症感染に対して一定の安全性を確保していると考えるが、今後とも関連情報の収集に努め、本剤の安全性の確保を図っていきたい。

*現在製造を行っていない

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Anaplasma phagocytophilum Transmitted Through Blood Transfusion — Minnesota, 2007

Anaplasma phagocytophilum, a gram-negative, obligate intracellular bacterium of neutrophils, causes human anaplasmosis, a tickborne rickettsial disease formerly known as human granulocytic ehrlichiosis (*1*). In November 2007, the Minnesota Department of Health was contacted about *A. phagocytophilum* infection in a hospitalized Minnesota resident who had recently undergone multiple blood transfusions. Subsequent investigation indicated the infection likely was acquired through a transfusion of red blood cells. This report describes the patient's clinical history and the epidemiologic and laboratory investigations. Although a previous case of transfusion-transmitted anaplasmosis was reported (*2*), this is the first published report in which transfusion transmission of *A. phagocytophilum* was confirmed by testing of the recipient and a donor. Although polymerase chain reaction (PCR) assays provided reliable evidence of transmission in this case, no cost-effective method for screening blood donors for *A. phagocytophilum* exists. Screening donors for a recent history of tick bite is not likely to be sensitive or specific because such exposures are common and often not recalled by persons with anaplasmosis (*3*). Physicians should consider the possibility of anaplasmosis in patients who develop posttransfusion acute thrombocytopenia, especially if accompanied by fever, and should report suspected transfusion-associated cases to health authorities.

Case Report

The patient, a male aged 68 years with a medical history of chronic renal insufficiency, psoriatic arthritis, ankylosing spondylitis, and corticosteroid therapy, underwent elective knee arthroplasty and synovectomy on October 12, 2007. Three weeks before his hospitalization, the patient had traveled to an area where blacklegged ticks (*Ixodes* spp.) were endemic, but he did not spend time outdoors and had no known tick bites. Several hours after the procedure, the patient developed bleeding at the surgical site and associated coagulopathy, indicated by elevated international normalized ratio (INR) and partial thromboplastin time (PTT) and by decreased fibrinogen and platelet counts. The extensive hemorrhage required two surgical evacuations of hematoma from the knee, popliteal artery embolization, and transfusion of multiple blood components. During October 12–21, the patient received 34 units of nonleukoreduced red blood cells (RBC), 4 units of leukocyte-reduced apheresis platelets, 14 units of fresh frozen plasma (FFP), and 7 units of cryoprecipitate. The components came from 59 individual blood donors; all donations were collected by Memorial Blood Centers (St. Paul, Minnesota). On October 19, the patient developed sepsis and multisystem failure. He was treated empirically with antibiotics (cefazolin, piperacillin/tazobactam, vancomycin, and levofloxacin). Blood cultures were negative on October 18, 20, and 31, and urine cultures were negative on October 19 and 25.

On October 31, the patient was found to have worsening thrombocytopenia. His platelet count declined from 178,000/mm^3 on October 31 to 54,000/mm^3 on November 5. On November 1, he developed hypotension and fever attributed to urinary tract infection. He was treated with levofloxacin and sulfamethoxazole/trimethoprim and was afebrile by November 3. On November 3, 22 days after admission, a peripheral blood smear from the patient demonstrated inclusions compatible with

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The *MMWR* series of publications is published by the Coordinating Center for Health Information and Service, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

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A. phagocytophilum morulae in neutrophils. Retrospective review of an October 15 blood smear from the patient showed no evidence of intracellular morulae. Whole blood specimens from November 3–5 were positive for *A. phagocytophilum* DNA by PCR assays conducted at the Mayo Medical Laboratory, Minnesota Department of Health, and CDC. Serum from November 3–5 was tested at CDC and found to be weakly positive by indirect immunofluorescence assay (IFA) (titer 1:64) for immunoglobulin G (IgG) antibodies to *A. phagocytophilum*. Doxycycline treatment was begun on November 5. The patient's platelet count steadily improved and returned to a normal level of 163,000/mm^3 on November 10. Pretransfusion blood samples and serum from the patient's convalescence period were not available for further testing. The patient improved clinically and was transferred to a rehabilitation unit on November 13. After rehabilitation, the patient was discharged on December 3, 2007.

Epidemiologic and Laboratory Investigation

In early November, Memorial Blood Centers began an investigation to identify whether any of the 59 blood donors associated with the 34 RBC, 4 platelet, 14 FFP, and 7 cryoprecipitate units had evidence of *A. phagocytophilum* infection. Paired whole blood specimens from the original donations had been retained from all 34 RBC donors and eight of 14 FFP donors and were available for PCR testing. During November 2007–March 2008, Memorial Blood Centers also collected postdonation blood samples for serologic testing and information on recent illness history and potential tick exposure from 53 of the 59 donors. In addition, plasma components from two FFP donors and two cryoprecipitate donors who donated again during December 2007–January 2008 were retained for serologic testing. The whole blood specimens retained from initial donation were tested by PCR, followed by sequencing of the PCR amplicons at CDC. Serum and plasma specimens were tested by IFA for IgG antibodies to *A. phagocytophilum*.

PCR and IFA tests on samples from a female RBC donor aged 64 years were positive for *A. phagocytophilum* infection (Table). *A. phagocytophilum* DNA was found in an RBC product donated by this woman on September 28 and transfused to the patient on October 13. IgG IFA titers to *A. phagocytophilum* were 1:512 and 1:256, respectively, in subsequent sera collected November 17 and December 18. The donor did not recall being bitten by a tick, but had spent time in wooded areas of northeast Minnesota where anaplasmosis is endemic within the month before her donation. She reported no history of fever during the month before or after her donation. No other patients received blood components from her donation.

TABLE. Polymerase chain reaction (PCR) and immunofluorescence assay (IFA) results* for *Anaplasma phagocytophilum* testing of transfusion blood products from 59 donors — Minnesota, 2007

Blood product	PCR	IFA	No. of donors
Red blood cells (n = 34)	+	1:512†	1
	–	1:64	2
	–	<1:32	31
Apheresis platelets (n = 4)	NA§	<1:32	4
Fresh frozen plasma (n = 14)	–	<1:32	6
	–	NA	2
	NA	<1:32	6
Cryoprecipitate (n = 7)	NA	<1:32	7

* Results from PCR testing by CDC of 42 whole blood segments retained from the original donations and IFA testing of 57 serum or plasma specimens submitted after the original donation.
† IFA titers 1:64 and higher were considered positive.
§ Test results not available.

No whole blood samples from other tested donors were PCR positive for *A. phagocytophilum*. Sera from two RBC donors were weakly positive by IFA (titer 1:64), but their respective whole blood samples from the original transfused units were PCR negative. These two donors did not live on wooded property and reported they had no tick exposure or illness during the 2 months before donation. Available postdonation serum samples from other donors were negative for *A. phagocytophilum* by IFA (titer <1:32).

Reported by: *M Kemperman, MPH, D Neitzel, MS, Minnesota Dept of Health; K Jensen, J Gorlin, MD, E Perry, MD, Memorial Blood Centers, Saint Paul; T Myers, MD, T Miley, MD, Park Nicollet Methodist Hospital, Saint Louis Park, Minnesota. J McQuiston, DVM, ME Eremeeva, MD, PhD, ScD, W Nicholson, PhD, J Singleton, National Center for Zoonotic, Vector-Borne, and Enteric Diseases; J Adjemian, PhD, EIS Officer, CDC.*

Editorial Note: *A. phagocytophilum*, the causative agent of anaplasmosis, typically is transmitted to humans by infected *Ixodes* spp. ticks. In wooded areas of the United States, *A. phagocytophilum* is transmitted by the blacklegged tick (*Ixodes scapularis*) in the Northeast and upper Midwest and by the western blacklegged tick (*Ixodes pacificus*) on the West Coast. In infected persons who are symptomatic, illness onset occurs 5–21 days after a bite from an infected tick. Initial presentation typically includes sudden onset of fever, headache, malaise, and myalgia, often accompanied by thrombocytopenia, leukopenia, and elevated liver transaminases. Severe infections can include prolonged fever, shock, confusion, seizures, pneumonitis, renal failure, hemorrhages, opportunistic infections, and death (*1*). Anaplasmosis and other tickborne diseases, including human ehrlichiosis, Rocky Mountain spotted fever, and babesiosis, caused by *Ehrlichia chaffeensis* or *Ehrlichia ewingii*, *Rickettsia rickettsii*, and *Babesia* spp., respectively, represent a potential risk for transmission via blood transfusion in the United States (*2–6*).

The case described in this report provides strong presumptive evidence that *A. phagocytophilum* infection in this patient was acquired through blood transfusion. Pretransfusion blood samples and convalescent serum from the transfusion recipient were not available for PCR or serologic testing to demonstrate conclusively that the patient was free of *A. phagocytophilum* infection before his hospitalization on October 12. However, the patient reported limited outdoor exposure that might include potential tick contact during the 3 weeks before hospitalization, and a blood smear collected 3 days after hospital admission showed no evidence of intracellular morulae. The timing of events and the expected incubation period for anaplasmosis (5–21 days) suggest that the patient's exposure most likely occurred during hospitalization. *A. phagocytophilum* DNA was found in a retained sample from the implicated RBC product that was transfused to the recipient, providing strong evidence that this was the likely route of disease transmission to the blood transfusion recipient.

Some blood transfusion recipients (i.e., those who are immune compromised) likely are at increased risk for developing severe complications associated with tickborne diseases. Both *A. phagocytophilum* and *E. chaffeensis* can survive in refrigerated RBCs, and possible transfusion-transmission cases have been reported for anaplasmosis (Minnesota Department of Health, unpublished data, 1998) (*2,3,5,6*). However, because of the rarity of transfusion-associated cases, concerns regarding the specificity of available tests, (none of which are approved by the Food and Drug Administration), and the economic costs associated with implementation, the U.S. blood supply is not routinely screened for tickborne disease using laboratory methods (*7*).

As a method to reduce the risk for certain pathogens in blood products, blood banks often defer donations if the potential donor is ill at the time of donation. However, persons infected with tickborne disease might experience mild illness or have asymptomatic infection, as was the case with the implicated donor in this report (*1,3*). Screening donors for a recent history of tick bite is unlikely to identify high-risk donors, because this type of exposure frequently is not recalled by persons with anaplasmosis (*3*). In this case, the implicated donor did not recall a tick bite, although she did report contact with wooded habitat in an anaplasmosis-endemic area. Nearly 75% of the other blood donors in this investigation reported similar outdoor contact, making the screening of blood donors for tick-related exposures poorly predictive for possible infection. Because *Ehrlichia* and *Anaplasma* are associated with white blood cells, leukoreduction techniques would be expected to reduce the risk for *Ehrlichia* and *Anaplasma* transfusion-transmission through RBC components (*5,8*). In the absence of effective screening tools to identify donors or products infected with

the organisms, physicians should weigh the benefits of using leukoreduced blood components, to potentially reduce the risk for *Ehrlichia* and *Anaplasma* transmissions.

Although transfusion-associated transmission of *A. phagocytophilum* appears to be rare, reported incidences of anaplasmosis and other tickborne diseases are increasing in the United States (*1*). A record 322 cases of anaplasmosis were reported in Minnesota in 2007 (6.2 cases per 100,000 population) (*9*). As the incidence of tickborne diseases increases, physician vigilance for possible transmission of these agents via transfusions also should increase. In addition to other more common etiologies, physicians should suspect possible rickettsial infection if transfusion recipients develop acute thrombocytopenia posttransfusion, especially if accompanied by fever. Such signs should lead to rapid assessment for rickettsial agents and empiric treatment with doxycycline (*1*). Although insensitive, blood smear can provide timely support for a presumptive diagnosis of anaplasmosis, followed by IFA or PCR to confirm the diagnosis (*1*). Similarly, babesiosis should be suspected in patients who develop hemolytic anemia and fever posttransfusion (*3,4*).

Anaplasmosis and ehrlichiosis are nationally notifiable diseases. Suspected cases of tickborne rickettsial diseases should be reported promptly to the state or local health department, and suspected transfusion-associated transmission should be reported to the supplying blood center and appropriate public health authorities.

Acknowledgments

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Progress in Introduction of Pneumococcal Conjugate Vaccine — Worldwide, 2000–2008

Pneumococcal disease is a leading cause of childhood morbidity and mortality globally, causing an estimated 0.7–1.0 million deaths annually among children aged <5 years (*1*). A pneumococcal conjugate vaccine (PCV) that includes seven pneumococcal serotypes (PCV7) first became available in 2000. Studies in the United States have demonstrated that introduction of universal vaccination with PCV7 resulted in a 77% decrease in invasive pneumococcal disease among children aged <5 years and a 39% decrease in hospital admissions for pneumonia among children aged <2 years (*2,3*). A similar vaccine with two additional serotypes was highly efficacious against pneumonia and invasive disease in clinical trials in Africa and, in one trial, reduced all-cause mortality among children by 16% (*4*). Low-income countries, which account for >97% of pneumonia cases in children aged <5 years (*5*), will benefit most from introduction of PCV. This report summarizes the progress made in introducing PCV7 worldwide. As of August 2008, 26 countries offered PCV7 to all children as part of national immunization programs or had PCV7 in widespread use (i.e., with estimated national coverage >50%); however, none of these countries is a low-income or lower-middle income country. The World Health Organization (WHO) and UNICEF have recognized the safety and effectiveness of PCVs and recommend that these vaccines for young children be included in national immunization programs (*1*). Overcoming the challenges to global introduction remains an urgent public health priority.

WHO recommends including PCV in national immunization programs (i.e., routine vaccination of all young children with PCV), particularly in countries where all-cause mortality among children aged <5 years is >50 per 1,000 live births or where >50,000 children die annually from any cause (*1*). In addition, because persons infected with human immunodeficiency virus (HIV) are up to 300 times more likely to have pneumococcal disease than those who are HIV negative (*6*), WHO recommends that countries with a high prevalence of HIV infection make the introduction of PCV a priority.

Only one PCV, the 7-valent formulation (PCV7), is currently licensed for use worldwide; new formulations of PCV (10-valent or 13-valent) are scheduled to become available

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No. 30

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研究報告の概要

○インフルエンザA型ウイルス(H1N1)、オセルタミビル耐性:南半球
タミフル(oseltamivir)耐性型の"通常の"季節性インフルエンザが急速に拡大しており、今年の冬(2008～2009)のインフルエンザ株の制御に当該薬剤が効果を示さない可能性がある。
H1N1株に感染した南アフリカ人患者107名全員がタミフルに耐性を示す変異株を保有していた。タミフルを服用していた患者は1名のみであった。
H1N1ウイルスの変異は、2007年の第4四半期～2008年3月31日に34カ国(主に北半球の国々)7528検体の検査では16%であったのに対し、2008年8月1日～20日の期間に、12カ国(主に南半球の国々)788検体の検査では、242名(31%)であった。
タミフル耐性型インフルエンザは、2007年1月に初めてノルウェーので蔓延がWHOに報告されて以来、ヨーロッパ、北米、南米、アフリカ、アジア、オーストラリアの40カ国で報告されている。
タミフル等の抗ウイルス製剤は、パンデミックが発現・蔓延後、ワクチンが開発されるまでの3ヶ月以上の期間、主要な治療手段であり、タミフルはWHOや世界各国の政府によって備蓄されている。
スウェーデンの研究者らは、ヒトで発症する別のウイルスと耐性型ウイルスが組み合わされた場合、タミフル耐性株に突然変異する可能性があると述べた。

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」
照射解凍赤血球濃厚液「日赤」
解凍赤血球-LR「日赤」
照射解凍赤血球-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

報告企業の意見

南アフリカをはじめとした南半球の各国において、タミフル(oseltamivir)耐性型の"通常の"季節性インフルエンザが急速に拡大しているとの報告である。

今後の対応

タミフル耐性インフルエンザウイルスが拡大しているという情報は、公衆衛生上及び血液事業への影響が大きく、厳重な注意が必要である。今後も引き続き情報の収集に努める。

(17)

Health Minister David Cunliffe said this year [2008] that 103 of the 1229 treatment courses of Tamiflu the Government had bought at a cost of [USD] 300 000 had reached their expiry dates.

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Communicated by:
ProMED-mail Rapporteur Mary Marshall

[Oseltamivir (brand name Tamiflu) is a medication that decreases the spread of influenza A and B viruses. Neuraminidase is an enzyme that enables the influenza virus to spread from infected cells to healthy cells. Oseltamivir blocks the action of neuraminidase (that is, Tamiflu is a neuraminidase inhibitor) thereby reducing the spread of influenza. By preventing the spread of virus from cell to cell, the symptoms and duration of influenza infection are reduced. On average, oseltamivir reduces the duration of symptoms by one and a half days if treatment is started within 48 hours after symptoms begin. Thereafter it becomes less effective.

As far as is known Tamiflu-resistant influenza A virus does not exhibit any enhanced or decreased virulence.

The final paragraph of the report above reveals a weakness inherent in the strategy of maintaining stockpiles of Tamiflu to combat seasonal and avian influenza; namely, the drug has a limited shelf life. - Mod.CP]

[see also:
Influenza A (H1N1) virus, oseltamivir resistance (05): China (HK) 20080203.0438
Influenza A (H1N1) virus, oseltamivir resistance (04): CA, USA 20080202.0428
Influenza A (H1N1) virus, oseltamivir resistance (03): corr. 20080203.0430
Influenza A (H1N1) virus, oseltamivir resistance (03) 20080201.0399
Influenza A (H1N1) virus, oseltamivir resistance (02): Europe 20080129.0371
Influenza A (H1N1) virus, oseltamivir resistance - Norway 20080128.0361
2007

Avian influenza, human (101): Indonesia, Tamiflu resistance 20070622.2021
Influenza B virus, neuraminidase inhibitor resistance 20070404.1143
Avian influenza, human (15): Egypt, drug resistance 20070119.0253
Avian influenza, human (15): Egypt, drug resistance 20070110.0238
2006

Avian influenza, human (162): oseltamivir resistance 20061010.2907
2005

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Influenza viruses, drug resistance (06) 20051016.3021
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Influenza viruses, drug resistance (03) 20051007.2924
Influenza viruses, drug resistance (02): RFI 20051001.2878
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2004

Avian influenza A (H5N1) virus, drug resistance (02) 20040127.0316
Avian influenza A (H5N1) virus, drug resistance 20040125.0298
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Influenza virus, neuraminidase inhibitor resistance (02) 20010928.2372
Influenza virus, neuraminidase inhibitor resistance 20010926.2350]
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