Transfusion-Associated Cytomegalovirus Mononucleosis

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Transfusion-associated cytomegalovirus mononucleosis is generally considered only as a complication of extracorporeal circulation following cardiac surgery. Three cases following trauma were recognized in less than one year. Both massive and limited volume blood transfusions were involved. Hectic fever was a characteristic feature in these otherwise remarkably asymptomatic individuals, without the classic features of heterophile-positive infectious mononucleosis. Since the illness developed several weeks into the post-operative period after extensive thoracic or abdominal trauma surgery, the presence of an undrained abscess was naturally the major diagnostic concern. Atypical lymphocytosis, markers of altered immunity (cold agglutinins, rheumatoid factor) and moderate hepatic dysfunction were important laboratory clues. In one case, focal isotope defects in the spleen scan misleadingly suggested a septic complication. A false-positive monospot test initially obscured the correct serologic diagnosis in the same patient. Failure to consider this self-limited viral infection may be a critical factor leading to unnecessary surgery. Other viral agents capable of eliciting a similar syndrome are cited.

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INCREASING USE OF extracorporeal circulation (ECP) in cardiac surgery called attention to a self-limited viral-like systemic syndrome that complicated these procedures in a significant percentage of cases.[23,27] Scattered specialty journal reports now document the occurrence of cytomegalovirus (CMV) infection as a consequence of blood transfusion in many situations, but most physicians still consider this viral infection a complication seen exclusively following ECP employed during cardiac surgery. Three cases of transfusion-associated CMV mononucleosis were recognized in our hospital in less than one year. Noteworthy features included (1) both massive and limited volume blood transfusions, (2) surgery for trauma without the use of ECP, (3) a false-positive monospot test and (4) a hitherto unreported pattern of isotope activity in the enlarged spleen of one of these patients.

Case 1 (M.W.). A 19-year-old white man received a gunshot wound to the abdomen on 10/3/74 requiring closure of gastric and jejunal perforations, hepatic lacerations, splenectomy, distal pancreatectomy and a left nephrectomy. Six drains were inserted in the upper abdomen. Thirteen units of blood were transfused intraoperatively and cephalothin and chloramphenicol were started postoperatively. On 10/6, his hematocrit abruptly fell from 35% to 25%, but stabilized after transfusion of two units of blood. On 10/11 his temperature rose to 38.3° and purulent drainage was noted from a left upper quadrant drain. His white blood count was 23,600/mm³ with a left shift, and he had hepatic dysfunction (glutamic oxalacetic transaminase, SGOT—70 u, normal—40 u; alkaline phosphatase—282 International units, normal 101 I u; bilirubin—2.9 mg%). On 10/13 he vomited fresh blood; endoscopy revealed multiple erosions of the gastric mucosa. He stabilized temporarily on iced saline lavage, but required 7 additional units of blood. Massive bleeding on 10/16 could not be controlled with a pitressin infusion. He was taken to surgery for plication of a bleeding gastric ulcer, vagotomy and pyloroplasty and received 12 units of blood. The left subphrenic space contained clotted blood which subsequently grew *E. coli*, pseudomonas and enterococci. Postoperatively he received both clindamycin and gentamicin but a low-grade fever persisted. On 10/27 all drains were removed and his temperature promptly rose above 38°; irrigation of the left upper quadrant wound yielded 200 ml of pus. In the face of continued clinical improvement, his temperature fluctuated between 39–40°, despite antibiotic therapy. His white blood count was 9,000/mm³ with no shift to the left. Isotope scans, echography and a sinogram of the left upper quadrant wound yielded no clues to the source of fever. He was again explored, but no abscess was found. Liver biopsy revealed nonspecific periportal inflammation. Postoperatively his white blood count rose to 28,000/mm³ with a 72% lymphocytosis, 60% of which were atypical cells and hepatic enzymes rose (SGOT—70 u, alkaline phosphatase—282 I u). Heterophile and toxoplasma antibody studies were negative, but his serum titer for CMV-CF antibody rose from zero to 1:64 over the next two weeks. He became afebrile after three weeks, and his white blood count, which peaked at 32,000/mm³ on 11/25/74, finally returned to normal 6 weeks later.

Comment. This young man's final laparotomy might have been avoided had atypical lymphocytes in larger numbers appeared in his peripheral blood earlier. In the week prior to this third procedure, 6 to 7% atypical lymphocytes were recorded, but this early suggestion of a mononucleosis syndrome was overlooked despite continued clinical improvement in the face of a persistent and significant fever.[16] The entire sequence of events pointed strongly to yet another undrained

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collection of pus. The transfusion of 34 units of blood during the first two weeks should have suggested the possibility of this syndrome. The absence of a spleen may have accounted for the markedly elevated white blood count.[16,20]

Case 2 (F.H.). This 44-year-old white man entered MSH on 6/7/75 with a gunshot wound to the heart. At surgery, bleeders were ligated and a myocardial laceration was sutured. He received 8 units of blood, but re-bled 12 hours later. At re-exploration, the left internal mammary artery was ligated; seven more units of blood were required. Cefazolin, chloramphenicol and gentamicin were begun. On the 6th hospital day, the medial sternotomy wound dehisced. Cultures of the mediastinal space, at the time of the sternal reclosure, grew pseudomonas species. Three more units of blood were transfused. Cefazolin and chloramphenicol were discontinued, carbenicillin was added and gentamicin continued. Drainage from from the mediastinal chest tube gradually decreased, despite continued growth of pseudomonas and persistent low-grade fever. He became afebrile for 5 days at the end of June and the beginning of July, but fever (37.8°) reappeared on the 3rd of July and gradually peaked at 40° 6 days later. The presence of 22% atypical lymphocytes in his peripheral blood at this time (7/9/75) prompted studies for mononucleosis; although his monospot test was repeatedly positive, heterophile determinations were not diagnostic (Table 1).

The spleen was scanned on 7/23/75 with Technetium 99; the enlarged organ showed defects in the middle and lower segments (Fig. 1) although it was neither clinically enlarged nor tender. Uric acid, alkaline phosphatase and SGOT levels were elevated (9.4 mg%, 135 I u, 80 u, respectively). At the time of discharge (7/29/75) significant titers of RA latex and cold agglutinins were noted, but CMV-CF titers were negative. One month later, the patient was asymptomatic and his physical examination was normal. Spleen scan revealed a normal-sized organ with residual evidence of the previous defects. His serum now contained a significant titer for CMV-CF antibody. The cold agglutinin titer had decreased and RA latex activity was absent. EBV antibody titers on three serial sera were identical (1:10) (TABLE 1).

Comment. This patient's hectic fever pattern one month after admission was viewed in the perspective of a significant atypical lymphocytosis (22%–28%), a relative bradycardia and lack of clinical toxicity despite daily 39.4–40° temperature elevations. Noteworthy features were the false-positive monospot test and focal splenic defects by scan.

Case 3 (R.S.). This 72-year-old woman suffered a left subtrochanteric hip fracture on 6/26/75. The following day, she underwent an open reduction and internal fixation with a Jewett nail and sideplate implantation. Single units of blood were given during surgery, and 5 and 6 days later (7/2 and 7/3/75). Her past history included hypertension, pulmonary tuberculosis, Parkinson's disease and pituitary irradiation for ablation of a chromoprobe adenoma. The postoperative course was complicated briefly by a pseudomonas urinary infection, a staphylococcal wound infection, and transient liver dysfunction during the second week. She was afebrile during the final month of her 7 week hospitalization and was discharged (8/16/75) with a normal white blood count, differential and liver function tests.

She was readmitted on 8/21/75 because of confusion, weakness and a temperature of 39.2°. There was no adenopathy, rash nor splenomegaly. The blood contained 4500 white cells/mm³ with 36% atypical lymphocytes. Urine cultures grew *E. coli* (>100,000 colonies/ml). Liver function tests revealed elevations of alkaline phosphatase (230 I u) and SGOT (217 u).

Her temperature peaked at 38.7° the first evening (pulse, 90/min). Defervescence and complete symptomatic recovery occurred over the next 48 hours, prior to therapy for her urinary infection. Liver function abnormalities returned to normal. Toxoplasma, monospot and heterophile studies were negative. CMV-CF antibody was present in a dilution of 1:32 on 8/21 (admission), 8/27 (discharge) and 10/9/75 (late convalescent). Repeat monospot, heterophile and toxoplasma antibody determinations on the late convalescent serum were unchanged. EBV titer was negative on the late convalescent serum.

TABLE 1. *Serial Serologic Studies in Case 2*

Date	7/11	7/21	7/25	7/29*	8/27
Monospot	+	+	+	+	+
Heterophile					
Presumptive	1/28	1/28	1/28	1/28	1/14
Kidney	1/28	1/14	1/14	1/14	1/7
Beef	0	1/7	1/14	1/14	0
Cold agglutinin	0			1/160	1/40
Toxoplasma IHA		1/256			1/256
RA Latex				1/640	0
CMV-CF					
(Lab 1)	0	0			1/64
(Lab 2)	<1/2				1/192
EBV Titer	1/10		1/10		1/10

* Discharged this date.

Comment. This case may not represent symptomatic post-transfusion cytomegalovirus mononucleosis. Her febrile course was brief and an accompanying urinary tract infection may have been responsible for the fever. However abnormal liver function studies, the pronounced atypical lymphocytosis and a borderline elevated but persistent and probably significant CMV-CF titer (1/32) suggested the presence of CMV activity. The occurrence of this "syndrome" 6 weeks after the transfusion of only three units of blood is noteworthy.

Discussion

Iatrogenic CMV infection following transfusion of fresh whole blood from latently infected donors,[13,17] the postperfusion syndrome (PPS), was first broadly recognized and defined in patients undergoing cardiac surgery with extracorporeal circulation, although it was probably first reported as a complication of noncardiac surgery as early as 1957 in Scandinavia.[2] These authors described 24 cases with fever and a hematologic picture resembling infectious mononucleosis 16 to 38 days following surgery for pulmonary tuberculosis. Adenopathy, pharyngitis, rash, and hepatospleno-

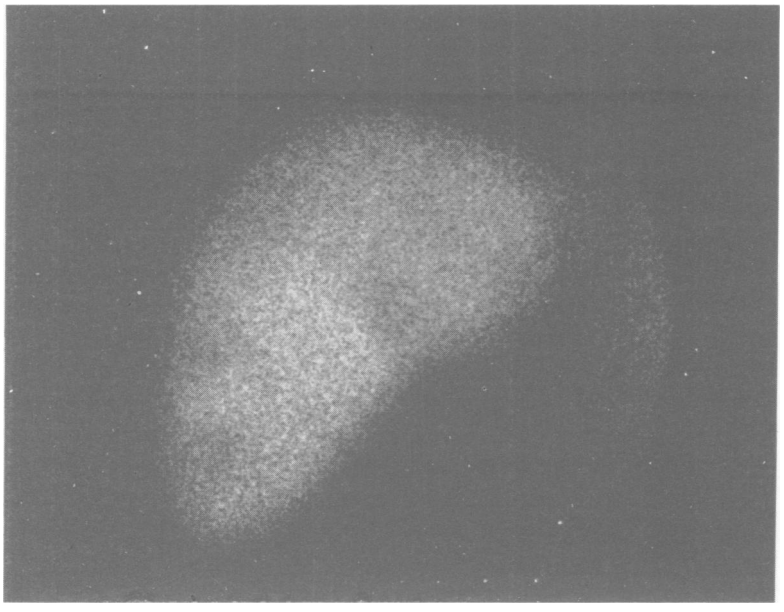

FIG. 1. Focal splenic defects, Technetium 99 Scan (7/23/75).

megaly were absent, and the Paul-Bunnell and toxoplasma tests negative in these generally asymptomatic individuals; fevers lasted from one to three weeks. Similar syndromes have followed a host of surgical conditions not utilizing ECP. An early example occurred in a 59-year-old man given three units of blood during a bypass graft with a homologous freeze-dried vessel; one month later, he developed a mononucleosis-like illness.[12] Gynecologic surgery and obstetrical complications[9,10,18], aborted heart surgery where the pump was never employed[10,18], cholecystectomy, appendectomy[33], lumbosacral fusion[30], splenectomy for idiopathic thrombocytopenic purpura[29], and trauma and burns[5,25] have been followed by this syndrome. Patients transfused for bleeding ulcers, esophageal varices and ulcerative colitis are also at risk.[20,38]

The spectrum of response to post-transfusion CMV infection ranges from asymptomatic seroconversion to a self-limited mononucleosis-like syndrome beginning approximately one month (two to six weeks) after surgery and lacking the features of classic infectious mononucleosis—pharyngitis, adenopathy, rash and a positive Paul-Bunnell test; splenomegaly and liver dysfunction are commonly present. High, often spiking, and long-lasting fever may dominate the clinical picture, usually without significant physical impairment.[33] Post-transfusion CMV mononucleosis is also to be contrasted with spontaneous CMV mononucleosis where generalized adenopathy, sore throat without exudative pharyngitis and a variety of usually non-fatal complications (ascending polyneuritis, pulmonary infiltrates, splenic infarction, myocarditis, meningoencephalitis) have been reported.[17,31] Persistent viruria is usually noted.[17,21] Markers of altered immunity include increased cold agglutinins, cryoglobulins, rheumatoid factor, Coombs-positive hemolytic anemia[4] and antinuclear antibodies.[20] The liver is usually involved but overt jaundice is uncommon.[4,21] Biopsy reveals a nonspecific inflammatory reaction and distinctive CMV inclusions are not commonly recognized in hepatocytes except in patients with disseminated CMV infection consequent to malignancy or other immunosuppressed states.[15] Granulomas have also been described and CMV infection should be excluded in all cases of acute granulomatous hepatitis with fever of unknown etiology.[4]

Fatal disseminated CMV infection in adults appears to be limited, for the most part, to patients who are immunosuppressed, either by their underlying disease or its therapies. Diffuse interstitial pneumonia may be responsible for death following renal,[19] bone marrow[26] and liver[1] transplantation. Active CMV infection developed in 96% of renal transplant recipients in one study.[8]

CMV resides in the leukocyte fraction of peripheral blood during viremic phases of infection.[24,36] Armstrong et al. studied patients with documented post-transfusion cytomegaloviremia.[1] All had received multiple blood transfusions or fresh frozen plasma, but only one had received fresh blood. They isolated virus from the blood, erythrocyte layer, the leukocyte layer and the plasma and serum, in that order of frequency. In vitro studies demonstrated persistence of inoculated CMV in the presence of erythrocytes in tissue culture media for up to 21 days. In whole blood, under banking conditions, inoculated virus was recovered after 28 days, and in fresh frozen plasma, after 97 days.

Diosi et al. found CMV-CF antibody titers ranging

from 1/8 to 1/64 in 21 of 32 healthy adult donors.[6] Virus grew from leukocyte cultures of fresh peripheral blood in two seropositive subjects; neither demonstrated recent illness or viruria. Other investigators have not been able to recover virus from unwashed leukocytes or leukocyte-rich plasma of large numbers of volunteer blood donors despite the fact that 58% to 75% of the donors had antibody titers for CMV (CF or indirect hemagglutinating);[19,24] one group included seven asymptomatic donors with CMV viruria at the time of blood donation.[19] Estimates of the viremic carrier rate range as high as 5–12% when patient seroconversion rates are analyzed, presupposing that the natural host is a more sensitive indicator than any culture system.[13,27]

The high incidence of positive reactors for CMV-CF antibody in the general population precludes the likelihood that effective screening measures can be developed on the basis of serological testing only.[24,35] Early reports implicated fresh donor blood as a source for CMV.[9] This is consistent with the lability of this virus, which is difficult to store or to isolate from other than fresh specimens, although the above cited in vitro studies cast some doubts about this.[1] More recent reports indicate that the incidence of CMV-CF antibody rise correlates best with the volume of blood received, but does not appear to be related either to the time of storage of the transfused blood or the type of operative procedure performed.[13,27,34] Some infections may represent activation of endogenous virus and it is possible that re-infection with antigenic variants of CMV may occur. The appearance of new antibody, especially of the IgM class, supports the diagnosis of primary infection.[23]

The most widely available assay for humoral immunity to CMV, i.e., the complement-fixing (CF) antibody test, may be the least reliable serologic method.[27,32,35] Fluorescent antibody determination was the most sensitive test employed by Stagno, et al.;[32] the indirect hemagglutinating antibody was only slightly less effective while complement-fixation determinations were significantly less reliable. The complete spectrum of CMV antibody responses in man may not be reflected in the CF reactivity to a single antigenic strain. Waner et al. employed three different strains and found fluctuating levels of CF activity in 50 donors over an 18-month period, with titers often ranging between significant and undetectable in the same individual.[35] Donor patterns varied and included individuals with consistently high CF titers against all three antigenic strains, some with depressed reactivity for one antigen as compared with the other two, and some with CF activity for only one or two strains.

Blood products can be contaminated with other viruses and agents which may produce similar syndromes. Heterophile-positive mononucleosis may follow blood transfusion; deliberate transmission to man was recorded as early as 1942.[37] More recently, an 86-year-old woman developed heterophile-positive, Epstein-Barr virus (EBV)—positive mononucleosis 5 weeks after receiving three units of blood.[3] Both CMV and EBV may be present and responsible for the postperfusion syndrome in the same patient.[11,13] The relative paucity of patients without pre-existing antibody to EBV, as compared to patients without anti-CMV antibody, probably accounts for the fact that CMV is more frequently involved in the postperfusion syndrome than EBV.[22] Despite appropriate studies, the etiology of heterophile-negative transfusion-associated mononucleosis remains obscure in a significant proportion of cases.[22] Leukocytes may also be the source of toxoplasma mononucleosis acquired by transfusion.[31]

Postoperative fever and atypical lymphocytosis was recently linked to rubeola virus transmission in this fashion.[16] A young man required laparotomy, splenectomy and multiple transfusions following a motorcycle accident. On the 23rd day post-injury, he developed a fever, his white cell count rose from 5400/mm^3 with 26% atypical lymphocytes to a peak of 45,000/mm^3 with 92% lymphocytes. Abdominal pain and rigidity led to a second laparotomy, which showed only reactive mesenteric adenitis. Rubeola virus was grown from a buffy-coat culture and rubeola CF titres rose to 1/32; all other viral studies were negative. Colorado tick fever virus transmission was confirmed fortuitously in an 82-year-old man with a prolonged and unexplained fever following exploratory laparotomy for bowel obstruction.[28]

Persistent postoperative fever following extensive thoracic or intraabdominal procedures for trauma usually initiates a search for an undrained collection of pus. As in our first case, several postoperative febrile episodes were associated with both hemorrhage and the evacuation of infected material yielding a mixture of aerobic intestinal flora. However, the final febrile episode coincided with the onset of a mononucleosis syndrome that was unfortunately not recognized despite the presence of 6–7% atypical lymphocytes in the peripheral blood in an asymptomatic patient gaining both strength and weight despite a hectic febrile pattern. Trauma and splenectomy may influence the course of postoperative viral infections acquired by transfusion.[16,28,29] CMV infection may be responsible for unnecessary surgery in some cases.[10] although this diagnosis does not exclude the simultaneous presence of an abscess.[14] With an increasing volume of trauma surgery and the extensive use of

blood replacement therapy, many patients may be at risk. CMV infection must be considered in the differential diagnosis of postoperative febrile states.

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