Original Article

Evaluation of Blood Loss and Transfusion Requirements in Diabetic and Non-Diabetic Patients Undergoing Coronary Revascularization

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ABSTRACT

Hematological abnormalities associated with the diabetic state include reduced platelet function, enhanced factor activation and hyperfibrinolysis. The present investigation evaluated blood loss and the transfusion requirements in diabetic and non-diabetic patients undergoing extracorporeal circulation and cardiac surgery.

A retrospective study of 151 consecutive patients undergoing primary coronary revascularization were classified into one of three groups based on their diabetic disease state: Insulin Dependent Diabetics (IDDM, n=5), Non-Insulin Dependent Diabetics (NIDDM, n=33), and Non-Diabetics (NDM, n=113). IDDM patients were significantly younger than either NIDDM or NDM patients, were 80% female, and had lower preoperative red cell mass. The IDDM group had a greater red blood cell transfusion volume (1650 ± 1485 ml) than NIDDM (958 ± 593 ml) and NDM (997 ± 827 ml) patients (p=0.21). There were no significant differences in homologous blood exposure or chest tube drainage in any group. Mean length of hospital stay (days) in the IDDM group (19.8 ± 18.3) was significantly greater than the NIDDM (9.9 ± 3.7), and the NDM (10.8 ± 5.8) patients (p<0.001).

In conclusion, diabetic patients do not appear to be at increased risk for developing post-cardiotomy coagulopathies, despite having greater morbidity, which results in increased hospital stay.

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INTRODUCTION

Coronary artery disease is the major cause of morbidity and mortality in patients afflicted with insulin-dependent diabetes mellitus (1,2). Diabetic patients have increased morbidity post acute myocardial infarction, resulting in a higher incidence of congestive heart failure and "sudden death" (3). Despite the increased risk, coronary revascularization is commonly used to treat coronary artery disease in the diabetic patient. One of the common complications of coronary artery bypass grafting (CABG) is perioperative hemorrhage secondary to impaired hemostasis. Platelet function abnormalities, along with intrinsic coagulation and fibrinolytic derangements, are common in the diabetic patient (3-15), yet the absolute relationship of such hematological abnormalities with the risk of postoperative hemorrhage has not been defined. In addition, the diabetic patient has been shown to have an activated coagulation state with increased platelet activity (12). Platelet aggregation is increased in the diabetic patient due to a greater concentration of platelet specific proteins (platelet factor 4 and thrombomodulin) (5,12). Hyperglycemia affects metabolism of prostaglandins, which in turn produce less prostacyclin (PGI_2) from arachidonic acid. Consequently, platelets have increased aggregation properties in the diabetic patient (7), and increased concentrations and/or activities of blood coagulation factors, all of which contribute to a hypercoagulable state.

Conflicting views exist regarding specific fibrinolytic activity as resulting from diabetes (12). Decreased fibrinolysis is common in the diabetic patient, resulting from increased plasminogen activator inhibitor (PAI) activity (5,13,14). The diabetic patient has been shown to have increased concentrations of vonWillebrand's factor, fibrinogen, and enhanced thrombus formation (5,10). Known derangements in the endothelium of diabetic patients affect coagulation factor production, as well as activation of the clotting cascade and thrombus formation (7). When physiological inhibitors of coagulation are investigated, discrepancies occur due to lack of pertinent data (6,8,11,13,15).

Postoperative bleeding is always a concern after cardiopulmonary bypass (CPB) and can be occasionally life threatening. Hemorrhage occurs in 5% to 18% of patients who have undergone open heart procedures (16-18), with a high percentage of those patients requiring transfusion and, occasionally, reoperation to correct bleeding problems. The intent of this investigation is to examine the relationship and clinical relevance of the hemorrhagic and subsequent transfusion requirements associated with diabetes in patients undergoing coronary artery bypass grafting.

MATERIALS AND METHODS

After approval by the Institutional Review Board, charts were retrospectively examined of 151 patients who underwent CABG for ischemic heart disease from July 1, 1993, to June 30, 1994. These included elective and emergent operations for surgical myocardial revascularization and were performed by two primary surgeons. The total number of patients operated on was 218 for that twelve-month period, but the criteria excluded 67 patients who had undergone repeat sternotomy procedures, valvular operations, combination valve/CABG, and orthotopic heart transplants.

Patients in this study were assigned to one of three groups based on their diabetic disease state, or lack thereof. These groups are based on the selection criteria of the National Diabetes Data Group and are as follows (19):

Group 1 (insulin dependent IDDM): Patients who have Type I diabetes mellitus with abrupt onset of classic symptoms. These patients suffer from total insulinopenia and require daily exogenous insulin injections for maintenance of health and of life itself and are insulin dependent.

Group 2 (insulin treated NIDDM): Patients who have Type II diabetes mellitus with insidious onset have few or no classic symptoms of the disease. These patients are not dependent upon exogenous insulin for survival and are insulin treated. Treatment methods included single or multiple pharmalogical interventions with insulin, acetohexamide, chlorpropamide, glipizide, glyburide, tolazamide, and/or tolbutamide. Diet is also used alone or in conjunction with pharmalogical intervention in some cases.

Group 3 (NDM): Patients who are non-diabetic without personal or family history of diabetes and with normal fasting glucose levels to serve as controls

Standard CPB circuitry with a membrane oxygenator[a], centrifugal pump[b], venous reservoir bag[a], and arterial line filter[a] were used in all patients. The prime consisted of a 2000 ml balanced electrolyte solution containing 100 ml 25% albumin and 10,000 iu of bovine lung heparin. During CPB arterial pressures were maintained between 70-90 mm Hg, with flow rates between 2.0 and 2.4 L/minute/m^2. Patients were cooled to 32°C core temperature. Both retrograde and antegrade blood cardioplegia (8:1 blood to crystalloid ratio) was used for arresting and maintenance doses. A terminal dose of warm (37°C) cardioplegia was administered to all individuals over a 10 to 20 minute period prior to removal of the cross clamp.

Patient parameters measured included anthropormorphic, operative, postoperative and laboratory values. Patients were monitored throughout their entire hospitalization and the following indices for hemorrhage recorded: chest tube drainage, intake and output volumes, and transfusion requirements.

Patients were transfused with packed red blood cells when the hemoglobin level fell below 7 gm/dL if the patient was less than seventy years of age, and 8 gm/dL if the patient was more than seventy years of age, or if the patient became hemodynamically unstable due to a suspected anemia. Patients were

a Baxter Healthcare Corp., Bentley Division, Irvine, CA 92714
b Medtronic Biomedicus, Eden Prairie, MN 55344

transfused with coagulation factors in the form of fresh frozen plasma (FFP), platelets (PLT), and cryoprecipitate (CRYO) only when bleeding was uncontrollable according to the following protocol: FFP when the fibrinogen (FIB) was less than 100 mg/dl, PLT when platelet counts were less than 100,000/ul, and CRYO when fibrinogen levels were less than 100 mg/dl and fibrin split products were greater than 40 ug/ml.

Table 1: Anthropomorphic Data

	NON-DIABETIC n=113	IDDM n=5	NIDDM n=33	P VALUE*
GENDER	M=91; F=22*	M=1; F=4	M=21; F=12*	0.0023 vs IDDM
AGE (YEARS)	65.3 ± 10.3*	50.8 ± 13.3	63.4 ± 10.3*	0.0094 vs IDDM
HEIGHT (CM)	173 ± 13.4	170 ± 14.0	169.58 ± 10.3	NS
WEIGHT (KG)	81.7 ± 17.6	71.0 ± 14.2	84.6 ± 18.0	NS
BSA (M^2)	1.96 ± 0.23	1.82 ± 0.26	1.96 ± 0.24	NS
PREOP ASA	72/113=64%	4/5=80%	24/33=73%	NS
MORTALITY	2/113=2%	0/5=0%	0/33=0%	NS
RBC MASS PREOP (ML)	1921.7 ± 531.6 (n=111) (MEDIAN, 1828)	1401.6 ± 603.2 (n=5) (MEDIAN, 1467)	2063.4 ± 457.5 (n=33) (MEDIAN, 2066)	0.0336

BSA = Body Surface Area; ASA = Acetylsalicylic Acid. All data is mean ± stdev.

Table 2: Operative Data

	NON-DIABETIC n=113	IDDM n=5	NIDDM n=33	P VALUE
# OF GRAPHS	3.5 ± 1.0	3.8 ± 1.1	3.9 ± 0.9	NS
IMA-ONE	62/113=55%	2/5=40%	12/33=36%	NS
IMA-BOTH	13/113=11%	0/5=0%	1/33=3%	NS
IMA-NONE	38/113=34%	3/5=60%	20/33=61%	0.0039
CPB TIME (MIN)	92.4 ± 24.6	106.4 ± 22.8	99.1 ± 23.2	NS
CC TIME (MIN)	66.2 ± 17.9	75.4 ± 17.4	72.5 ± 17.2	NS
VENTILATOR HOURS	23.3 ± 37.8	29.6 ± 11.6	20.5 ± 11.5	NS
ICU DAYS	3.5 ± 2.3	9.2 ± 14.4	3.1 ± 0.7	0.004
HOSPITAL DAYS	10.8 ± 5.8	19.8 ± 18.3	9.9 ± 3.7	0.0045
IABP	5/113=4%	0/5=0%	2/33=6%	NS

IMA = Internal Mammary Artery; CPB = Cardiopulmonary Bypass; CC = Cross Clamp; ICU = Intensive Care Unit; IABP = Intra Aortic Balloon Pump. All data is mean ± stdev.

Laboratory Assessment

Preoperative laboratory assessment includes a prothrombin time (PT), partial thromboplastin time (PTT), PLT count, and template bleeding time. Coagulation profiles were performed on admission and throughout the perioperative period. Following heparinization (300 units/kg body weight), activated clotting time (ACT) values were maintained at > 480 seconds. Laboratory analysis including arterial blood gases, electrolytes, glucose, and ACT was performed 10-15 minutes after CPB was initiated, and then routinely every 30 minutes while the patient was maintained on CPB. Postoperative laboratory assessment occurred upon patient admission to the intensive care unit and then again on the morning of the first postoperative day. Chemistry profiles were performed on admission, throughout the surgical procedure and postoperatively. Included were routine electrolytes, glucose, blood urea nitrogen, and creatinine.

Statistics

Statistical analysis was performed by loading all data onto a personal computer in a spread sheet format. Parametric data was analyzed using a one-way analysis of variance (ANOVA) for intergroup statistics. Additional multiple comparison tests (Fisher's least significant difference) were performed when significant "F" ratios were achieved ($p<0.05$) Non-parametric data was analyzed using a Chi-square analysis. All data are reported as mean ± standard deviation of the mean.

RESULTS

There were 5 patients in the IDDM group, 33 in the NIDDM group, and 113 in the NDM group. Clinical characteristics of the diabetic and non-diabetic groups are shown in Table 1. The IDDM patients were significantly younger than either the NIDDM or NDM patients. There was also a significantly higher proportion of females in the IDDM group. Total hospital length of stay is characterized in Table 2 and was significantly longer for IDDM patients when compared to NIDDM patients and NDM patients. Packed red blood cells infused while on pump was significantly higher in the NIDDM compared to either IDDM or NDM patients ($p<0.04$) (Figure 1). The patients in the IDDM group all received packed cells

while on pump, while only 33% of NIDDM patients and 20% of NDM patients received blood during the same time. Chest tube drainage is shown in Table 5. There were no significant differences between groups. Heparin and protamine doses were not significantly different in any of the groups.

Total volume transfused during the duration of hospital stay is shown on Table 3. None of the parameters listed in Table 3 were significantly different. Overall, 100% of our IDDM group received packed red blood cells during their hospital stay, while 73% of NIDDM patients and 76% of NDM patients received packed red blood cells.

Laboratory values are depicted in Table 4. Glucose levels were all significantly elevated from baseline values in the three groups of patients. Preoperative hematocrits were also significantly different between groups. The hematocrits of the patients in the IDDM group were significantly lower than those in the NIDDM group and the NDM group. Red cell mass was calculated using weight in kg multiplied by hematocrit multiplied by an average blood volume of 60ml/kg. Preoperative red cell mass was significantly less in IDDM patients when compared to NIDDM patients and NDM patients (Table 1). Discharge hematocrits, platelet counts, protimes and PTT's were not significantly different between the three groups of patients.

None of our IDDM patients required a preoperative transfusion, 100% required perioperative transfusions, and 80% postoperative transfusions. Additionally, of the NIDDM patients, 3% required preoperative transfusions, 73% surgical transfusions, and 76% postoperative transfusions. In our control group of NDM patients, 6% received preoperative transfusions, 74% received blood during surgery, and 65%

Figure 1: Packed RBC Infusions

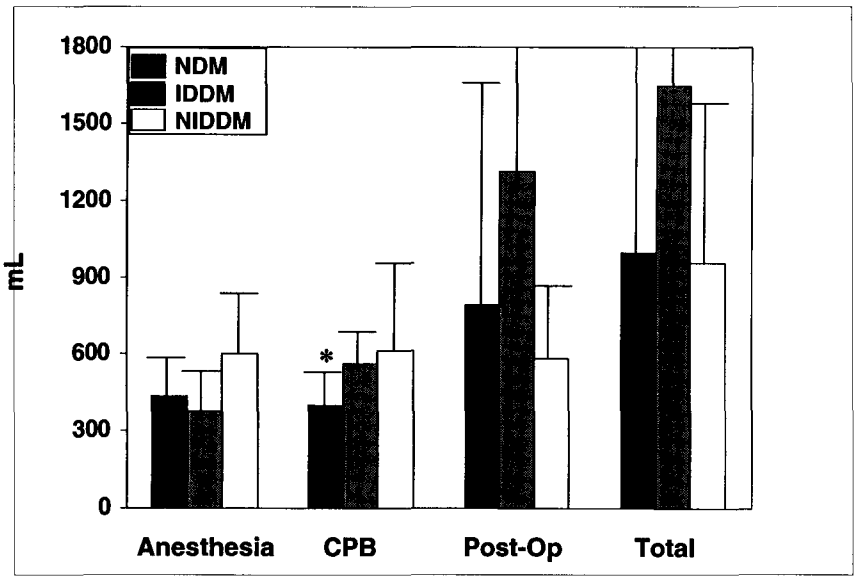

Table 3: Total Volume

	NON-DIABETIC	IDDM	NIDDM	P VALUE
PRBC (ML)	997.1 ± 827.4 (n=86) (MEDIAN, 750)	1650 ± 1485.4 (n=5) (MEDIAN, 1250)	958.3 ± 592.8 (n=24) (MEDIAN, 750)	NS
PRBC (# UNITS)	4.0 ± 3.3 (n=86) (MEDIAN, 3)	6.6 ± 5.9 (n=5) (MEDIAN, 5)	3.8 ± 2.4 (n=24) (MEDIAN, 3)	NS
PLTS (ML)	872.6 ± 579.6 (n=81) (MEDIAN, 600)	700 ± 346.4 (n=4) (MEDIAN, 600)	906.0 ± 452.2 (n=20) (MEDIAN, 700)	NS
PLTS (# UNITS)	14.0 ± 9.2 (n=81) (MEDIAN, 10)	10.5 ± 7.4 (n=4) (MEDIAN, 10)	14.9 ± 8.1 (n=20) (MEDIAN, 11)	NS
FFP (ML)	848.0 ± 799.2 (n=75) (MEDIAN, 600)	700 ± 200 (n=4) (MEDIAN, 800)	800.0 ± 549.4 (n=23) (MEDIAN, 800)	NS
FFP (# UNITS)	4.2 ± 4.0 (n=75) (MEDIAN, 3)	3.5 ± 1.0 (n=4) (MEDIAN, 4)	4.0 ± 2.7 (n=23) (MEDIAN, 4)	NS
CRYO (ML)	601.5 ± 316.3 (n=17) (MEDIAN, 500)	500 ± 0 (n=1) (MEDIAN, 500)	635.7 ± 184.2 (n=7) (MEDIAN, 600)	NS
CRYO (# UNITS)	24.1 ± 12.7 (n=17) (MEDIAN, 20)	20 ± 0 (n=1) (MEDIAN, 20)	25.4 ± 7.4 (n=7) (MEDIAN, 24)	NS
% REC'D PRBC	76%	100%	73%	NS
% REC'D PLTS	72%	80%	61%	NS
% REC'D FFP	66%	80%	70%	NS
% REC'D CRYO	15%	20%	21%	NS
COLLOID (ML)	426.6 ± 382.8 (n=111)	300.0 ± 215.1 (n=5)	454.7 ± 371.9 (n=32)	NS
COLLOID/KG	5.4 ± 5.1 (n=111)	4.6 ± 3.7 (n=5)	5.5 ± 4.3 (n=32)	NS

PRBC=Packed Red Blood Cells; PLT=Platelets; FFP=Fresh Frozen Plasma; CRYO=Cryoprecipitate. All data is mean ± stdev.

received blood products postoperatively.

DISCUSSION

Excessive postoperative bleeding continues to complicate cardiac surgery, with life-threatening hemorrhage occuring in 5-18% of the cardiac surgery population (16-18). Approximately 10-20% of patients exhibit inadequate hemostasis of varying duration and severity, requiring intervention that includes transfusions of blood products (16, 18). Many authors have suggested predictors and variables for hemorrhage and/or hemostatic derangements during and after CPB, including the following: pharmacological treatment prior to surgery, anesthetic techniques, complexity and duration of surgical procedure, extracorporeal surface exposure, hypothermia, hemodilution, prime constituents, heparinization and heparin neutralization, use of transfused blood products, and hypocalcemia (20-23). All of these factors can bring about a number of hemostatic changes that can eventually lead to excessive bleeding.

The major contributors to hemostatic derangements during CPB are alterations in platelet function and/or platelet number, resulting in an acute acquired defect in the formation of the platelet plug, as well as an increase in template bleeding time (21,22). Immediately after the initiation of CPB, platelet count decreases due to the hemodilution that occurs with crystaloid priming solutions, although the importance of this decrease is unknown. Exposure to the extracorporeal surface activates platelets and promotes aggregation and the release of alpha granules from the platelet (22). When the bleeding time is abnormal during CPB, indicating a functional platelet defect, the quantitative platelet count is usually greater that 80-90,000/mm^2 (17,20). A transient activation of platelets and/or a platelet membrane abnormality occurs when a depletion of platelet alpha granules is accompanied by an increase of beta thromboglobulin, platelet factor 4, and thromboxane B2 (24). Additional theories of platelet activation triggers include release of ADP from hemolyzed red blood cells, or the release of human neutrophil elastase by contact activation and protein mediated pathways (25). Platelets exposed in vitro to a membrane oxygenator show reduced binding to fibrinogen due to exposure of their fibrinogen receptor site and adherence to surface adsorbed fibrinogen (21,22,25). As bypass continues, some platelets become detached and leave a fragment of platelet membrane still adhered to the surface of the circuit. The detached platelets possess fewer fibrinogen

Table 4: Laboratory Values

	IDDM (n=5)	NIDDM (n=33)	P VALUE
GLUC PREOP	243.3 ± 147.2	168.8 ± 65.4	0.0001
GLUC OPER 1	273.8 ± 69.1	238.6 ± 54.8	0.0001
GLUC OPER 2	358.2 ± 116.5	287.7 ± 85.3	0.0001
GLUC OPER 3	354.5 ± 64.3	284.1 ± 84.3	0.0470
HCT PREOP	33.4 ± 14.0	40.8 ± 5.7	0.0121
HCT POSTOP 1	25.9 ± 5.3	28.7 ± 4.7	NS
HCT POSTOP 2	28.4 ± 1.5	26.9 ± 3.1	NS
HCT DISCHG	32.6 ± 3.4	32.1 ± 4.5	NS
PLTS PREOP	270.0 ± 95.5	240.2 ± 59.4	NS
PLTS POSTOP 1	162.5 ± 42.4	163.8 ± 55.7	NS
PLTS POSTOP 2	136.0 ± 39.5	162.3 ± 50.0	NS
PROTIME PREOP	12.0 ± 0.6	11.9 ± 0.8	NS
PROTIME POSTOP 1	15.1 ± 1.5	14.2 ± 1.1	NS
PROTIME POSTOP 2	13.4 ± 0.5	12.9 ± 1.2	NS
PTT PREOP	35.2 ± 16.9	34.9 ± 16.8	NS
PTT POSTOP 1	33.1 ± 2.9	31.1 ± 6.2	NS
PTT POSTOP 2	31.0 ± 2.0	28.0 ± 6.9	NS

GLUC = Glucose (mg/dl); HCT = Hematocrit (%); PLTS = Platelets (10^3/uL); PROTIME = Prothrombin Time (sec); PTT = Partial Thromboplastin Time (sec); PREOP = Preoperatively; OPER 1 = Operative 1; OPER 2 = Operative 2; OPER 3 = Operative 3; POSTOP 1 = Postoperative 1; POSTOP 2 = Postoperative 2; DISCHG = Discharge.

Table 5: Chest Tube Drainage

	NON-DIABETIC n=113	IDDM n=5	NIDDM n=33	P VALUE
CT DRAIN—24 HRS	604.1 ± 481.0	550.4 ± 529.8	545.4 ± 505.6	NS
PER KG	7.9 ± 6.4	7.1 ± 5.6	6.3 ± 5.7	NS
CT DRAIN—48 HRS	453.3 ± 394.3 (n=110)	463.4 ± 294.4	366.7 ± 268.9 (n=31)	NS
PER KG	5.8 ± 5.1 (n=110)	6.1 ± 3.7	4.4 ± 2.8 (n=31)	NS
CT DRAIN—72 HRS	130.6 ± 160.0 (n=39)	127.0 ± 99.8 (n=3)	118.1 ± 174.2 (n=16)	NS
PER KG	1.7 ± 2.1(n=39)	1.6 ± 1.1 (n=3)	1.4 ± 2.0 (n=16)	NS
CT DRAIN—TOTAL	1097.7 ± 756.5	1090.0 ± 786.9	946.1 ± 716.4	NS
PER KG	13.9 ± 10.0	14.2 ± 8.2	11.1 ± 7.5	NS

CT DRAIN = Chest Tube Drainage

receptors and therefore are not fully functional (25). New platelets enter the circulation, as demonstrated by the normal or low normal quantitative platelet count, but do not adhere to the synthetic surface of the circuit for two reasons. One reason is that adsorbed fibrinogen has undergone conformational changes so that the molecule is not recognized by the platelet fibrinogen receptor (26,27). The second reason is that some adsorbed fibrinogen binding sites may be covered by platelet membrane fragments left behind by detached platelets (25). Therefore, at the conclusion of CPB, the platelet population is heterogenous, consisting of new, intact, functional platelets (24), partially degranulated platelets (22), platelets with damaged membranes that have resealed (25), and platelet fragments. The bleeding time and platelet count gradually return to normal after discontinuation of bypass, but there may be abnormal bleeding in patients with a functional platelet defect (22). The degree of impairment in platelet function is directly proportional to the duration of CPB (22). Additional abnormalities most frequently diagnosed after CPB include prolongation of clotting tests related to heparin, low fibrinogen titers, complex changes of clotting tests due to hemodilution or rarely disseminated intravascular coagulation, and enhanced fibrinolytic activity (20). The concentration of other coagulation factors also decreases due to crystalloid prime, but these reductions rarely impair clotting.

Many authors have shown hemostatic abnormalities in the diabetic population, which theoretically contribute to increased bleeding due to lack of platelet function (3-15,28). Studies have shown a reduction in heparin cofactor II (HCII) activity, decreased anti-thrombin III (AT-III) biological activity, and increased of fibrinopeptide A plasma levels in IDDM patients (6,8,9,11). Normal AT-III plasma levels have been reported in the diabetic patient despite decreased biological activity. This phenomenon could be explained on the basis of a structurally modified AT-III that retains immunoreactivity but is functionally depressed (5,6). HCII and AT-III are both glycoproteins that inhibit thrombin in the clotting cascade. Fibrinopeptide A is a byproduct of the fibrinogen to fibrin conversion. Therefore, a decrease in AT-III activity, coupled with an increase in fibrinopeptide A in IDDM patients, indicates a thrombin hyperactivity, leading to a hypercoagulable state (9). IDDM patients have a hypercoagulable state, which may be related to poor glycemic control (28). Hypercoagulability is manifested in IDDM patients by increased platelet aggregation and number, augmented plasma beta thromboglobulin and PF4 as a result of increased platelet activation, elevated vWF, raised coagulation factors, and impaired fibrinolysis (high levels of plasma and urine fibrinopeptide reflecting coagulation activation and fibrin formation). Increased blood viscosity has also been observed (28). The hypercoagulable diabetic state may predispose the diabetic patient to consumptive coagulapathies and influence the genesis of perioperative and postoperative bleeding diathesis. We were unable to confirm this state, but the small number of IDDM patients (n = 5) could play a role in our conclusions.

We demonstrated that patients with IDDM have both a significantly lower preoperative hematocrit and lower preoperative red cell mass. They required a larger volume of red cells during their hospital stay, although it was not significantly different from the volume transfused to the other two groups. In the IDDM population in our study, 80% were women, which could account for overall lower hematocrits in ths group. Cosgrove and associates identified preoperative hematocrit as a predictor of blood use in NDM patients (29). Diabetic patients in general have lower hematocrits than NDM patients, due to shortened red blood cell life span (30). Causes of shortened red cell life span in the diabetic population include the following: 1) microvascular disease where the red blood cell is physically damaged as it passes through arteries roughen by atherosclerotic plaque; 2) iron deficient anemia caused by inadequate iron intake, inefficient intestinal absorption, and blood loss; and 3) renal failure which causes inadequate erythropoietin production (30).

One recognized limitation of this study was the lack of hematological assessment available. Laboratory tests the authors would have found useful include the following:

1. In vitro platelet aggregation response to epinephrine, ADP, or collagen. IDDM and NIDDM have "hyperreactive" platelets which leads to a hypercoagulable state (31), and possibly a consumptive coagulaopathy as CPB progresses.

2. Total fibrinogen titer. Platelets from diabetics bind a greater amount of fibrinogen (30) and show increased fibrinolysis translating to low fibrinogen levels as CPB progresses.

3. von Willebrand factor. Initially diabetics have a high vWF level, but with a consumptive coagulaopathy, this level should fall on CPB. Correction of a low vWF requires CRYO infusion or desmopressin acetate administration. Levels need to be monitored throughout the case.

4. Beta thromboglobulin. High levels indicate increased platelet release. Beta thromboglobulin is also released during platelet aggregation. Diabetics have a high rate of platelet turnover.

The second limitation to our study is its retrospective nature. Ideally, a randomized prospective study is indicated with specific laboratory tests measured at defined intervals throughout the case and the CPB procedure. In addition, the small number of IDDM patients may have also influenced our findings, but reflects the distribution of patients afflicted with this disease at our institution.

In conclusion, insulin dependent diabetic patients present with lower red blood cell mass although their red blood cell transfusion requirements were not significantly different from the other groups. There was no significant difference in the quanity of coagulation factors replaced between diabetic or non-diabetic patients. The increased length of hospital stay seen in the insulin dependent diabetic group reflects the increased morbidity associated with this disease.

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