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Outcomes of Severity-Stratified Antithrombotic Management of Imaging-Detected Thrombosis After Simultaneous Pancreas-Kidney Transplantation

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Abstract

Background

Pancreas allograft thrombosis is an important cause of early graft dysfunction and loss after simultaneous pancreas-kidney (SPK) transplantation, and postoperative antithrombotic practices vary among centers. We describe outcomes of an institutional severity-stratified antithrombotic approach in which treatment intensity was linked to imaging-defined thrombus burden.

Materials and methods

We performed a single-center, single-arm, descriptive, retrospective cohort study of 79 adult SPK recipients who were transplanted between January 1, 2018, and September 17, 2025. Postoperative Doppler ultrasound and, when indicated, computed tomography angiography were used to identify vascular abnormalities. Management generally consisted of aspirin for normal vascular findings, clopidogrel for limited venous thrombus involving <50% of the vessel, and systemic anticoagulation for more extensive, progressive, or arterial thrombosis. Outcomes included thrombosis, study-defined major bleeding, thrombosis-attributed pancreas graft loss, and one-year patient and graft survival. Rotational thromboelastometry EXTEM was evaluated exploratorily when available.

Results

Venous thrombosis occurred in 18 of 79 recipients (22.8%), including 10 with <50% involvement and eight with ⩾50% involvement. Arterial thrombosis occurred in one patient (1.3%). Clinically significant thrombosis, which included persistent or progressive venous thrombus on repeat imaging, occurred in 14 patients (17.7%), while thrombosis-attributed pancreas graft loss occurred in three (3.8%). Study-defined major bleeding occurred in seven patients (8.9%). Among 76 recipients with at least one year of potential follow-up, one-year patient, kidney graft, and pancreas graft survival rates were 92.1%, 89.5%, and 81.6%, respectively (death was counted as graft loss). A classifiable EXTEM profile was available in 57 patients (72.2%), with no consistent association between coagulation profile and thrombosis or study-defined major bleeding.

Conclusions

Severity-stratified antithrombotic management based on imaging-defined thrombus burden was implemented in this cohort, and thrombosis-attributed pancreas graft loss occurred in three of 79 patients (3.8%). However, the retrospective design, small numbers of events, absence of a contemporaneous comparator, and possible variation in institutional practice preclude a conclusion regarding comparative effectiveness or safety. Prospective studies using standardized imaging and treatment criteria are warranted.

Categories: Transplantation, Internal Medicine, Nephrology
Keywords: antithrombotic therapy, doppler ultrasound, graft survival, pancreas allograft thrombosis, rotational thromboelastometry, simultaneous pancreas-kidney transplantation, venous thrombosis

Introduction

Simultaneous pancreas-kidney (SPK) transplant is an established treatment for selected patients with diabetes mellitus and end-stage kidney disease, restoring endogenous insulin production and providing

renal replacement [1]. Despite advances in surgical technique, organ preservation, and perioperative care, early complications of pancreas allografts remain clinically important because they can compromise graft function and patient outcomes [2].

Pancreas allograft thrombosis is one of the most serious early technical complications after SPK transplantation, involving venous outflow, arterial inflow, or both, and can lead to graft dysfunction, pancreatectomy, reoperation, and loss of insulin independence [2,3]. Thrombosis risk is multifactorial, influenced by donor factors, recipient comorbidities, ischemia time, surgical technique, graft pancreatitis, hemodynamic instability, and postoperative coagulation status [3].

Antiplatelet and anticoagulation strategies are widely used after pancreas transplantation, but there is no universally accepted regimen [4]. Heparin thromboprophylaxis may reduce early pancreas thrombosis and pancreas loss, though optimal patient selection and monitoring remain uncertain [5].

The major clinical challenge is balancing thrombosis prevention against bleeding risk: empiric full-dose anticoagulation may expose patients without early thrombotic findings to unnecessary hemorrhagic complications, while delayed treatment of early thrombus may allow progression to occlusion and graft loss. This tension supports a risk-adapted approach in which antithrombotic intensity is escalated according to objective postoperative findings rather than applied uniformly.

Postoperative imaging can identify early graft vascular abnormalities: Doppler ultrasound is the primary imaging modality for baseline assessment and evaluation of graft vascular flow, while cross-sectional computed tomography (CT) or CT angiography can further characterize suspected vascular compromise and thrombus burden [6]. Partial pancreatic graft thrombosis may be clinically silent, detected only on imaging, and, in some cases, treated with anticoagulation while preserving graft function [7]. Splenic vein thrombosis is particularly relevant, as a non-occlusive thrombus may represent an early, reversible stage; prophylaxis and treatment vary across institutions, from re-exploration to conservative anticoagulation depending on clinical and imaging factors [8].

Rotational thromboelastometry (ROTEM), using the extrinsically activated (EXTEM) assay, may add physiologic information to imaging-based decision-making by providing a whole-blood viscoelastic assessment of clot initiation, propagation, and strength [9]. In SPK transplantation, thromboelastography (TEG)-guided anticoagulation has been associated with less anticoagulation-related bleeding and fewer thrombosis-related graft losses than management guided by conventional coagulation testing [10]; earlier experience also supports the use of TEG to individualize anticoagulation in pancreas transplantation [11]. Whether an analogous ROTEM EXTEM classification improves clinical outcomes after SPK transplantation remains uncertain.

At our institution, postoperative antithrombotic therapy was stratified according to imaging-defined thrombus burden, with aspirin used for normal vascular findings, clopidogrel for limited venous thrombosis, and systemic anticoagulation for more extensive, progressive, or arterial thrombosis.

This single-arm, descriptive, retrospective cohort study, without a control group, describes the real-world implementation and outcomes of this severity-stratified antithrombotic management approach among adult SPK recipients. It does not compare treatment strategies, nor does it evaluate the comparative effectiveness or safety of imaging surveillance or any antithrombotic agent. The objectives were as follows: primary objective: to describe the occurrence of clinically significant thrombosis (the primary outcome), a composite of persistent or progressive venous thrombus on repeat imaging, arterial thrombosis with graft compromise, reoperation for thrombosis, or thrombosis-attributed graft loss, as defined in the Methods; secondary objectives: to characterize imaging-detected thrombosis (severity, timing, persistence, and resolution), treatment patterns according to thrombus severity, study-defined major bleeding, reoperation and pancreatectomy, and graft and patient outcomes, including one-year survival; and exploratory objective: to describe available ROTEM EXTEM measurements in relation to thrombosis and study-defined major bleeding. Because ROTEM testing and sampling times were not standardized during the study period, these analyses were considered hypothesis-generating.

Materials And Methods

Study design and setting

This was a single-center retrospective cohort study of adult SPK recipients managed at Rutgers University/Robert Wood Johnson (RWJ) Medical Center between January 1, 2018, and September 17, 2025, with an analytic data lock of August 7, 2026. The study was single-arm and descriptive and had no control group or contemporaneous alternative-treatment comparison. Because formal protocol versioning was unavailable, the approach represents institutional practice rather than a fixed prospective protocol. Severity-stratified management is documented from July 2019 onward; its use in the two earlier recipients could not be confirmed. The study follows Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidance [12].

Ethical approval

This retrospective chart review was approved by the Rutgers University Institutional Review Board (approval number: Pro2025002041), which waived the requirement for informed consent because the study used existing clinical data without direct patient contact. All data were de-identified before analysis.

Study population

Adult SPK recipients aged ⩾18 years who were identified in the institutional transplant database during the study period were assessed for eligibility. Patients were excluded if they underwent pancreas-transplant-alone, pancreas-after-kidney, or other multiorgan transplantation; had an incomplete record; lacked postoperative imaging or antithrombotic therapy data; or had insufficient follow-up to assess early outcomes. The analytic dataset did not retain the total number screened or patient-level exclusion counts.

Institutional severity-stratified antithrombotic management practice

Postoperative antiplatelet and anticoagulation management was guided by Doppler ultrasound, generally performed on postoperative day 1 with repeat imaging within 24-48 hours as warranted, and CT angiography when arterial abnormalities, progressive thrombus, or uncertain findings required further characterization (Figure *1*).

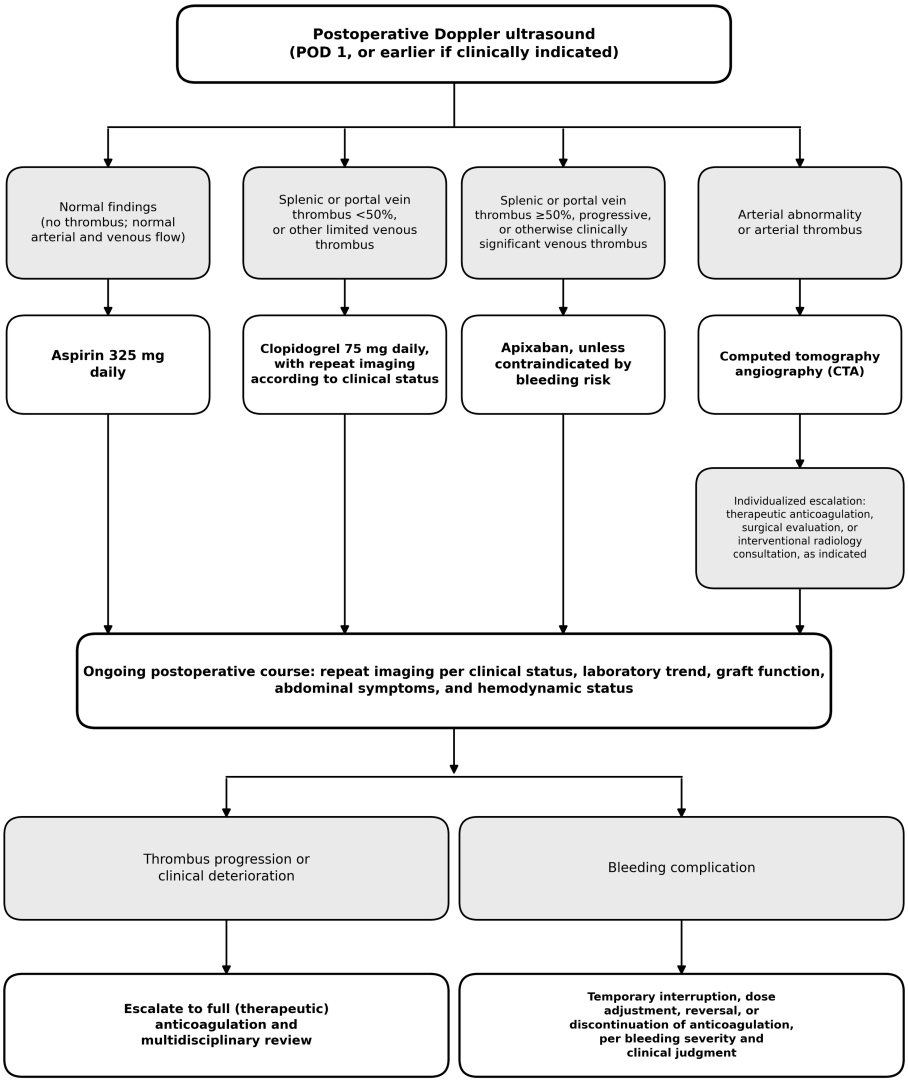


FIGURE 1: Reconstructed institutional severity-stratified antithrombotic management approach

Postoperative Doppler ultrasound findings guided treatment intensity, with aspirin for normal vascular findings, clopidogrel for limited venous thrombosis, and apixaban for more extensive or progressive thrombosis. Arterial abnormalities prompted CT angiography and individualized therapeutic anticoagulation, surgical evaluation, or interventional radiology consultation. Management was modified for progression, clinical deterioration, or bleeding. The figure summarizes the predominant practice reconstructed from clinical records and is not a prospectively version-controlled protocol.

CT: computed tomography; POD: postoperative day

Normal findings were managed with aspirin 325 mg daily; splenic or portal vein thrombus <50% with clopidogrel 75 mg daily and repeat imaging as clinically indicated; thrombus ≥50%, progressive, or otherwise clinically significant with apixaban unless contraindicated by bleeding risk; and arterial abnormalities with CT angiography and individualized therapeutic anticoagulation, surgical evaluation, or interventional radiology consultation (Figure *1*). Progression prompted anticoagulation and multidisciplinary review; bleeding prompted treatment modification as clinically indicated. Actual treatment received, including deviations from the reconstructed algorithm, was analyzed. In this stepwise design, clopidogrel served as an intermediate step for limited venous thrombus, and therapeutic anticoagulation was reserved for thrombus that was extensive, progressive, or arterial. This was a center-specific practice rather than a guideline-based treatment for venous thrombosis; its rationale and limitations are discussed in the Discussion.

Imaging assessment

Postoperative imaging data were abstracted from radiology and operative reports, including arterial and

venous flow, splenic and portal vein thrombus, arterial thrombus, graft edema, and peripancreatic fluid; when available, CT angiography findings were reviewed to characterize suspected abnormalities further.

For analysis, imaging data were converted to patient-level variables: presence and maximum severity of venous thrombus, day of first detection, persistence or resolution on serial imaging, and presence of arterial thrombus.

Venous thrombosis was defined as any postoperative duplex or CT angiography finding of splenic or portal vein thrombus, categorized as <50%, ⩾50%, or none. Time to thrombosis was calculated from transplant day 0 to the first evidence of venous thrombosis on imaging. Persistent thrombosis was defined as venous thrombus present on more than one postoperative imaging study, and thrombus resolution was assessed only among patients with follow-up imaging (see Definitions subsection below). Arterial thrombosis was any postoperative imaging evidence of arterial thrombus or occlusion.

Antiplatelet and anticoagulation variables

Medication data, recorded as delivered rather than as intended by the reconstructed management algorithm, were abstracted from the medical record, including the agent and, when documented, duration, partial thromboplastin time (when available), escalation due to imaging findings, and interruption due to bleeding. The abstracted dataset classified each patient by a single principal antithrombotic category (aspirin, clopidogrel, apixaban, or heparin); the aspirin and clopidogrel doses are those specified in the reconstructed algorithm. Apixaban dose and start day were not abstracted as reliable discrete variables, treatment-duration data were incomplete and did not permit the reconstruction of an exact apixaban duration for each patient, and whether aspirin was continued alongside another agent was not systematically abstracted.

ROTEM EXTEM assessment and coagulation profile classification

ROTEM EXTEM data were collected when available: clotting time (CT), clot formation time (CFT), alpha angle, amplitude at 10 minutes (A10), amplitude at 20 minutes (A20), and maximal clot firmness (MCF). Because A20 was not captured as a discrete variable, classification used CT, CFT, alpha angle, and MCF; A10 was recorded but not used, as it lacked a discrete assay-specific reference interval in the abstracted dataset. Manufacturer-labeled adult US EXTEM reference intervals, as summarized in prior ROTEM literature [9], were used: CT 43-82 seconds, CFT 48-127 seconds, alpha angle 65-80 degrees, and MCF 52-70 mm.

Unlike TEG, ROTEM EXTEM has no validated composite coagulation index, so classification uses these individual reference intervals. Patients were hypocoagulable if one or more available parameters were abnormal exclusively in the hypocoagulable direction, namely, CT >82 seconds, CFT >127 seconds, alpha angle <65°, or MCF <52 mm; normocoagulable if all available parameters were within range; and hypercoagulable if abnormal exclusively in the hypercoagulable direction, namely, CT <43 seconds, CFT <48 seconds, alpha angle >80°, or MCF >70 mm. Profiles with abnormalities in both directions were classified as mixed/indeterminate. Four parameters were preferred; profiles with three of four parameters available were classified only when the available parameters were concordant and did not indicate opposing directions, the sensitivity analysis required all four parameters.

Study Size

The number of eligible SPK transplants determined the fixed study size during the study period; no prospective power calculation was performed. The small number of thrombotic and bleeding events limited inference between groups and the use of multivariable modeling.

Primary Outcome

The primary outcome was clinically significant pancreas allograft thrombosis, defined as any of the following: arterial thrombosis with graft vascular compromise (imaging-confirmed arterial thrombus or occlusion associated with impaired arterial flow and/or prompting therapeutic anticoagulation, surgical evaluation, or interventional radiology review), persistent or progressive venous thrombus on repeat imaging (present on more than one postoperative imaging study), reoperation for thrombosis, or thrombosis-attributed pancreas graft loss (including pancreatectomy performed for thrombosis). Escalated antithrombotic therapy was analyzed as a secondary rather than a primary outcome because it was determined by imaging findings and its inclusion would have introduced circularity.

Secondary Outcomes

Secondary outcomes included the individual components of the primary composite endpoint, any venous or arterial pancreas allograft thrombosis, splenic vein thrombosis, time to first imaging-detected thrombosis, thrombus persistence, imaging-confirmed thrombus resolution, escalated antithrombotic therapy due to imaging findings, interventional radiology procedures, reoperation for thrombosis, reoperation for any cause within 30 days, pancreatectomy, study-defined major bleeding, surgical washout for hematoma, length of

hospital stay, 30-day readmission, insulin independence at discharge, kidney delayed graft function, and one-year patient, pancreas graft, and kidney graft survival. Portal vein thrombosis, minor bleeding, transfusion, and interruption of antithrombotic therapy due to bleeding were described when available, but were not analyzed as formal secondary outcomes due to incomplete or non-discrete data capture.

Definitions

Study-defined major bleeding was a clinically documented hemorrhage or hematoma requiring reoperation or surgical washout; minor bleeding was documented bleeding not meeting this criterion. Pancreas graft loss was graft removal, permanent return to insulin therapy, or documented non-function; kidney graft loss was permanent return to dialysis, transplant nephrectomy, or documented non-function. Thrombus progression was defined as increased extent, worsening occlusion, new territory involvement, or the development of combined arterial/venous thrombosis on repeat imaging. Because serial imaging recorded whether thrombus was present but not its extent, the venous criterion of the primary outcome was applied to thrombus present on repeat imaging (persistent or progressive); extension could not be quantified separately. Pancreatectomy and thrombosis-attributed pancreas graft loss were ascertained at any time from transplantation through the data lock, without a prespecified window; the date of graft failure was recorded, but the date and indication of pancreatectomy were not abstracted separately, so the recorded cause of graft failure is reported. One-year graft survival refers to graft function at one year after transplantation, with death counted as graft loss (all-cause graft loss). A patient with both graft failure and death before 12 months was counted once. Return to the operating room within 30 days was abstracted as a yes/no field with a free-text cause; for reporting, each reoperation was assigned to a category based on its first-listed cause. Thrombus resolution was defined as partial or complete improvement in thrombus burden on follow-up imaging; resolution status could not be determined for patients without follow-up imaging, and a functioning graft without insulin dependence was not considered evidence of resolution.

Data Collection and Missing Data

Data were collected retrospectively from the electronic medical record, transplant database, operative records, radiology and laboratory reports, and discharge summaries, entered under a unique study code and stored securely. Donor type (donation after brain death (DBD) versus donation after circulatory death (DCD)) was derived from separate source fields recording brain death and circulatory death status; when these two fields were discordant, donor type was classified as indeterminate rather than inferred, to avoid introducing misclassification. Missing data for key variables (ROTEM EXTEM profile, partial thromboplastin time, antithrombotic therapy duration, follow-up imaging, bleeding, and one-year outcomes) were recorded; complete-case analysis was used, with denominators reported for all proportions. To reduce measurement and reporting bias, outcome and exposure variables were operationally defined (see Definitions subsection), and denominators were explicitly reported for every proportion rather than defaulting to the full cohort. As a fixed-cohort chart review, loss to follow-up in the prospective sense does not apply; instead, follow-up duration varied by transplant date relative to the data lock on August 7, 2026. One-year patient, kidney graft, and pancreas graft survival were assessed only among recipients with at least one year of potential follow-up at the data lock (transplanted on or before August 7, 2025; n=76); the three recipients transplanted after that date were excluded from these denominators.

Statistical analysis

Continuous variables were summarized as mean±standard deviation or median (interquartile range), and categorical variables as counts and percentages, for the overall cohort. Analyses were performed in Python (version 3.12.3) using pandas (version 3.0.2) and SciPy (version 1.17.1); Wilson 95% confidence intervals were calculated from the standard formula.

Comparisons between patients with and without venous thrombosis and exploratory ROTEM EXTEM comparisons were analyzed using Fisher's exact test, Welch's unequal-variance t-test, or Mann-Whitney U test, as appropriate. Because treatment escalation was determined by imaging-detected thrombosis, escalated and non-escalated treatment groups were not analyzed as independent exposure groups.

Given the limited number of events, multivariable regression was not performed. Between-group analyses were exploratory and unadjusted; p-values were considered hypothesis-generating, with no adjustment for multiple comparisons. ROTEM EXTEM classification was repeated as a sensitivity analysis, requiring all four parameters to confirm normocoagulable status. One-year survival outcomes were summarized as counts and percentages with Wilson 95% confidence intervals among recipients with at least one year of potential follow-up (n=76).

Results

A total of 79 SPK transplant recipients were included in the final analysis (Figure *2*); the abstracted database did not separately record the number of patients excluded during initial screening, so the total number of SPK transplants performed during the study period, before exclusions, is not available from this dataset. Venous-thrombosis status, antithrombotic therapy status, and donor type were documented for all 79

patients; a classifiable ROTEM EXTEM profile was available for 57 patients. Denominators are reported explicitly throughout, since some variables (recipient BMI, intraoperative transfusion, 30-day readmission, one-year outcomes) have their own, smaller documented denominators. The recipients' mean age was 46.8±10.3 years, and the recipients' mean BMI was 26.1±3.8 kg/m^2 (n=75 documented). Donor mean age was 30.5±11.3 years, and donor mean BMI, calculated from donor weight and height (the recorded "Donor BMI" field was found to be unreliable and was not used), was 26.2±5.1 kg/m^2. Median follow-up, calculated from transplant date to the analytic data lock (August 7, 2026), was 45.4 months. Donor type (DBD, DCD, or indeterminate) differed between groups (overall Fisher's exact test across the three categories; Cramér's V=0.35; p=0.021), with DCD donors more frequent among recipients who developed venous thrombosis (six of 18, 33.3%) than among those who did not (four of 61, 6.6%) (Table *1*).

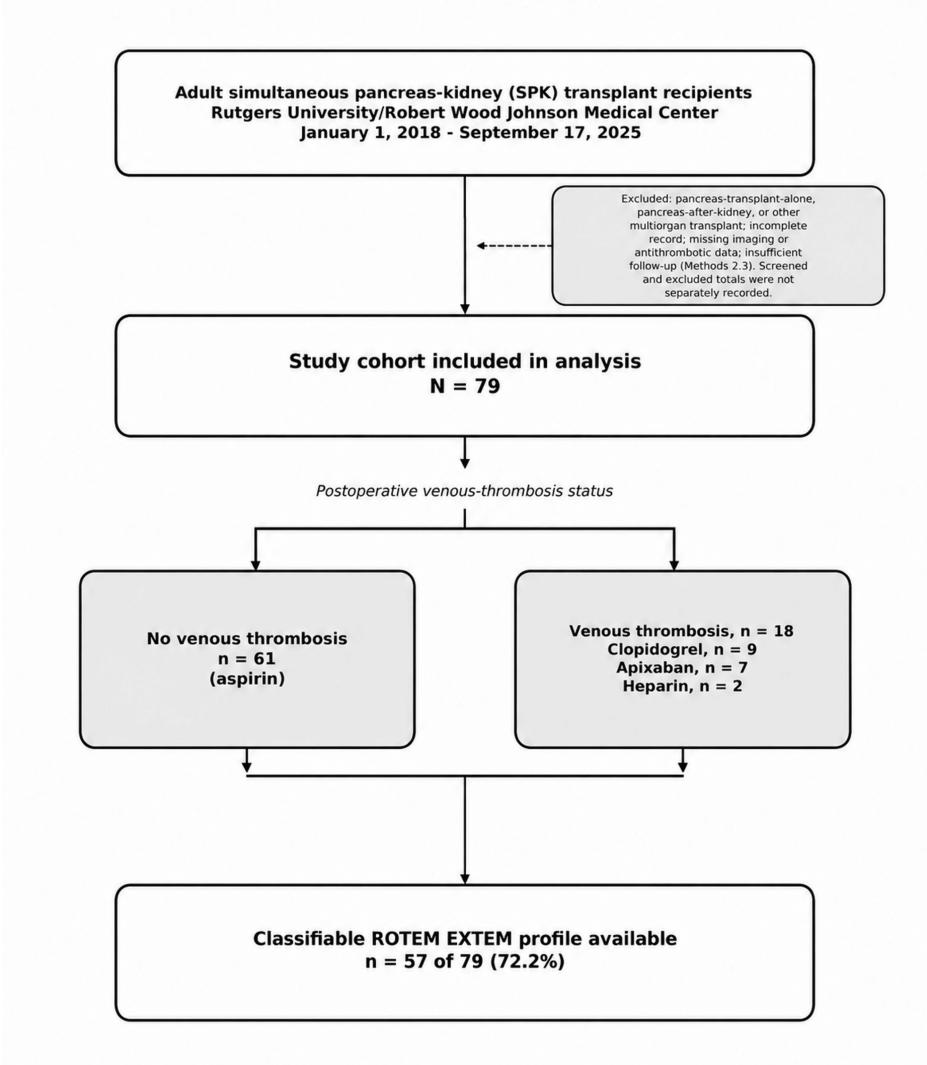


FIGURE 2: Study participant flow

Flow of adult simultaneous pancreas-kidney transplant recipients from the source population to the final analytic cohort (N=79), including the venous-thrombosis groups used in Tables 1-2 and the subset with classifiable ROTEM EXTEM profiles used in Table 4. The total number screened and patient-level exclusion counts were not available in the abstracted dataset; eligibility and exclusion criteria are described in the Methods.

ROTEM: rotational thromboelastometry; EXTEM: extrinsically activated

Variable	Overall N	Overall value	Overall SD/%	No-VT N	No-VT value	No-VT SD/%	VT N	VT value	VT SD/%	Statistical test	Test statistic	df	Effect size	P-value
Recipient characteristics														
Recipient age, years	79	46.8	10.3	61	47.8	9.8	18	43.4	11.3	Welch's t-test	t=1.50	25.2	—	0.147
Recipient sex, male, n (%)	79	63	80	61	50	82	18	13	72	Fisher's exact test	Not applicable (exact test)	—	OR 0.57	0.504
Recipient BMI, kg/m²	75	26.1	3.8	58	26.2	3.9	17	26.0	3.6	Welch's t-test	t=0.19	28.0	—	0.849
Diabetes type, n (%)														
Type I, n (%)	79	31	39	61	25	41	18	6	33					
Type II, n (%)	79	47	59	61	35	57	18	12	67	Fisher's exact test (2×3)	Not applicable (exact test)	—	Cramér's V=0.09	0.685
Other, n (%)	79	1	1	61	1	2	18	0	0					
Dialysis duration, months	75	22.3	20.6	57	23.5	22.7	18	18.3	11.3	Welch's t-test	t=1.30	59.1	—	0.197
Donor characteristics														
Donor age, years	79	30.5	11.3	61	30.4	11.3	18	30.6	11.7	Welch's t-test	t=-0.05	27.0	—	0.957
Donor BMI, kg/m²	79	26.2	5.1	61	26.0	5.3	18	27.1	4.4	Welch's t-test	t=-0.90	32.7	—	0.373
KDPI, %	79	17.5	13.4	61	17.9	13.8	18	16.4	12.6	Welch's t-test	t=0.42	30.1	—	0.675
Donor type, n (%)														
DBD, n (%)	79	66	83.5	61	54	88.5	18	12	66.7					
DCD, n (%)	79	10	12.7	61	4	6.6	18	6	33.3	Fisher's exact test (2×3)	Not applicable (exact test)	—	Cramér's V=0.35	0.021
Indeterminate, n (%)	79	3	3.8	61	3	4.9	18	0	0.0					
Pancreas cold ischemia time, hours	71	11.8	2.4	55	11.9	2.5	16	11.6	2.0	Welch's t-test	t=0.50	30.0	—	0.624
Kidney cold ischemia time, hours	77	13.9	2.7	61	13.8	2.8	16	14.1	2.3	Welch's t-test	t=-0.44	28.4	—	0.663
Intraoperative transfusion, n (%)	54	15	27.8	41	10	24.4	13	5	38.5	Fisher's exact test	Not applicable (exact test)	—	OR 0.52	0.478

TABLE 1: Baseline donor, recipient, and operative characteristics

Continuous variables are presented as mean±SD and categorical variables as n (%). P-values were calculated using Fisher's exact test for categorical variables and appropriate parametric or nonparametric tests for continuous variables. A single overall exact p-value (the Freeman-Halton extension of Fisher's exact test) is reported for both diabetes type and donor type. Donor BMI was calculated from the recorded donor height and weight. Donor type was indeterminate in three patients because DCD/DBD classifications were discordant. Intraoperative transfusion status was documented for 57 patients; three values did not match the coded responses ("yes"=1; "no"=2) and were treated as missing, leaving 54 patients (41 without and 13 with venous thrombosis) for this row. Each cell contains one value. Continuous variables are reported by group as N (documented), mean, and SD and categorical variables as N (denominator), n, and %. For continuous variables, Welch's unequal-variance t-test statistic and degrees of freedom are reported. Fisher's exact test provides an exact p-value without a conventional test statistic; therefore, the statistic is listed as "Not applicable (exact test)". Odds ratios (venous-thrombosis versus no venous-thrombosis group) are reported for 2×2 comparisons and Cramér's V for the 2×3 diabetes-type and donor-type comparisons. Category sub-rows report only category-specific N/n/% because test statistics, effect sizes, and p-values apply to the overall three-level comparison (—). All values were recalculated from the underlying study dataset. Kidney cold ischemia time was undocumented for two patients with venous thrombosis, leaving 77 patients with documented times (61 without and 16 with venous thrombosis).

VT: venous thrombosis; DBD: donation after brain death; DCD: donation after circulatory death

Imaging surveillance and thrombosis detection

Postoperative duplex ultrasound was performed during the index admission, with a median of 2 ultrasounds per patient (interquartile range: 2-3); patients with venous thrombosis underwent more inpatient ultrasounds than those without (3 (2-4) versus 2 (1-3); Mann-Whitney U=332.0; p=0.009), most likely reflecting additional surveillance after thrombosis was detected rather than a risk factor for its development. Venous thrombosis was identified in 18 patients (22.8%; 95% CI: 14.9-33.2%), including splenic vein thrombus less than 50% in 10 patients and splenic vein thrombus greater than or equal to 50% in eight

patients. Portal vein thrombus was not separately captured as a distinct variable in the abstracted dataset. Among the 13 patients with detailed serial imaging available, the median time to detection of the first venous thrombosis was 0 days after transplantation (interquartile range: 0-0; range: 0-2), reflecting predominantly early postoperative detection on days 0-1. Arterial thrombosis was identified in one patient (1.3% of the overall cohort; 95% CI: 0.2-6.8%), who also had splenic vein thrombus <50% and was managed with heparin-based anticoagulation (Table 2).

Variable	Overall N	Overall V1	Overall V2	Overall V3	No-VT N	No-VT V1	No-VT V2	No-VT V3	VT N	VT V1	VT V2	VT V3	Statistical test	Test statistic	df	Effect size	P-value
Length of stay, days — median, Q1, Q3	79	9	6	12.5	61	9	6	12	18	8.5	6.25	13	Mann-Whitney U test	U=504.0	—	—	0.601
Number of inpatient ultrasounds — median, Q1, Q3	79	2	2	3	61	2	1	3	18	3	2	4	Mann-Whitney U test	U=332.0	—	—	0.009
Any postoperative venous thrombus, n (%)	79	18	22.8	—	61	0	0	—	18	18	100	—	—	—	—	—	—
Splenic vein thrombus <50%, n (%)	79	10	12.7	—	61	0	0	—	18	10	55.6	—	—	—	—	—	—
Splenic vein thrombus ≥50%, n (%)	79	8	10.1	—	61	0	0	—	18	8	44.4	—	—	—	—	—	—
Arterial thrombus, n (%)	79	1	1.3	—	61	0	0.0	—	18	1	5.6	—	Fisher's exact test	Not applicable (exact test)	—	Not estimable (zero cell)	0.228
Median time to first thrombus, days — median, Q1, Q3	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a	13	0	0	0	n/a	n/a	n/a	n/a	n/a
Persistent thrombus on repeat imaging, n (%)	n/a	n/a	n/a	—	n/a	n/a	n/a	—	13	11	84.6	—	n/a	n/a	n/a	n/a	n/a
Imaging-confirmed thrombus resolution, n (%)	n/a	n/a	n/a	—	n/a	n/a	n/a	—	13	4	30.8	—	n/a	n/a	n/a	n/a	n/a
Aspirin, n (%)	79	61	77.2	—	61	61	100	—	18	0	0	—	—	—	—	—	—
Clopidogrel, n (%)	79	9	11.4	—	61	0	0	—	18	9	50.0	—	—	—	—	—	—
Apixaban, n (%)	79	7	8.9	—	61	0	0	—	18	7	38.9	—	—	—	—	—	—
Heparin, n (%)	79	2	2.5	—	61	0	0	—	18	2	11.1	—	—	—	—	—	—
Antithrombotic therapy escalated due to imaging, n (%)	79	18	22.8	—	61	0	0	—	18	18	100	—	—	—	—	—	—

TABLE 2: Imaging findings and antithrombotic management

Continuous variables are presented as median (interquartile range (IQR)) and categorical variables as n (%). P-values were calculated using Fisher's exact test for categorical variables and the Mann-Whitney U test for continuous variables. P-values are not reported (—) for variables defining venous-thrombosis groups or for antithrombotic treatments assigned based on imaging findings because these comparisons would be circular. Fisher's exact test was used for arterial thrombus, which is independent of group definition. Clopidogrel is an antiplatelet agent; apixaban and heparin are anticoagulants. Serial imaging outcomes use available-case denominators. Thrombus resolution was confirmed in four of 13 patients with serial imaging; status was unavailable in five without follow-up imaging. The single patient with arterial thrombosis also had <50% splenic vein thrombosis and is included in both categories. V1/V2/V3 represent row-specific quantities: for n (%) rows, V1=n and V2=% (V3=—); for median/Q1/Q3 rows, V1=median, V2=Q1, and V3=Q3. Mann-Whitney U statistics (for the no-venous-thrombosis group) are reported for continuous-variable comparisons; df and odds-ratio-type effect sizes are not applicable (—). Fisher's exact test has no conventional test statistic ("Not applicable (exact test)"); for arterial thrombus, the odds ratio was not estimable because one 2×2 cell was zero. Rows marked — or n/a have no formal statistical comparison, as noted above.

VT: venous thrombosis

Antithrombotic management

Antithrombotic management varied according to imaging findings. Aspirin was administered to 61 patients (77.2%), clopidogrel to nine (11.4%), and apixaban to seven (8.9%); the remaining two patients (2.5%), including the arterial-and-venous thrombosis patient noted above, received heparin-based anticoagulation. No patient was managed without antithrombotic therapy. Antithrombotic therapy was escalated based on imaging findings, including CT angiography, in 18 patients (22.8%) (Table 3). Among the nine patients managed with clopidogrel, none had arterial thrombosis, reoperation for thrombosis, or study-defined major bleeding; however, one lost the graft to thrombosis on postoperative day 8, with thrombus present on all

five serial studies. Across the seven with serial imaging, thrombus was present on more than one study in six and was no longer detected on the last study in three. A second patient underwent pancreatectomy, with the graft failure recorded as DM1. Because treatment was assigned based on thrombus severity and the group is small, these descriptive findings cannot demonstrate that clopidogrel was adequate. Partial thromboplastin time values during admission were not captured as a discrete variable in the abstracted dataset.

Imaging-detected category	N	Aspirin n	Aspirin %	Clopidogrel n	Clopidogrel %	Apixaban n	Apixaban %	Heparin n	Heparin %
Normal imaging	61	61	100	0	0	0	0	0	0
Splenic vein thrombus <50%	9	0	0	9	100	0	0	0	0
Splenic vein thrombus ≥50%	8	0	0	0	0	7	87.5	1	12.5
Arterial thrombus	1	0	0	0	0	0	0	1	100

TABLE 3: Maximum imaging-detected thrombosis category and antithrombotic therapy received

Each patient is classified once, by the maximum imaging-detected category, and therapy is the principal antithrombotic category recorded for that patient. The patient with arterial thrombus also had splenic vein thrombus <50% and appears only in the arterial row; the <50% row (n=9) therefore differs from the 10 patients with <50% thrombus in Table 2. Percentages for the four therapy columns use each row's own N as the denominator. No statistical tests are reported because therapy was assigned based on imaging findings, which would make the comparison circular.

ROTEM EXTEM findings

A classifiable ROTEM EXTEM profile, based on clotting time, clot formation time, alpha angle, and maximal clot firmness (amplitude at 20 minutes was not captured as a discrete variable), was available for 57 of 79 patients (72.2%). Of the 22 patients without a classifiable profile, 21 had no ROTEM EXTEM measurement recorded, and one had a timestamped measurement with no usable parameter values. Using the last available measurement as the principal exposure and expressing results as a proportion of the 57 classifiable patients, 42 (73.7%; 53.2% of the overall cohort) were normocoagulable, eight (14%; 10.1%) were hypocoagulable, five (8.8%; 6.3%) were hypercoagulable, and two (3.5%; 2.5%) had a mixed/indeterminate profile (Table *4*).

ROTEM EXTEM profile	Patients n	Patients N	Patients %	Venous thrombosis n	Venous thrombosis %	Arterial thrombosis n	Arterial thrombosis %	Escalated therapy n	Escalated therapy %	Major bleeding n	Major bleeding %
Hypocoagulable	8	57	14.0	3	37.5	0	0.0	3	37.5	1	12.5
Normocoagulable	42	57	73.7	12	28.6	0	0.0	12	28.6	3	7.1
Hypercoagulable	5	57	8.8	0	0.0	0	0.0	0	0.0	1	20.0
Mixed/indeterminate	2	57	3.5	0	0.0	0	0.0	0	0.0	0	0.0
ROTEM EXTEM not performed	21	79	26.6	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a
ROTEM EXTEM performed, but insufficient parameters to classify	1	79	1.3	n/a	n/a	n/a	n/a	n/a	n/a	n/a	n/a

TABLE 4: Exploratory ROTEM EXTEM profiles and observed clinical outcomes

ROTEM EXTEM profiles were classifiable in 57 of 79 patients using clotting time, clot formation time, alpha angle, and maximal clot firmness; A20 was unavailable as a discrete variable, and A10 was not used. All four parameters were available in 54 patients; three had three of four available with concordant classification. For patients with multiple ROTEM measurements, the last intraoperative or immediate postoperative value was used as the principal exposure. Clinical outcome percentages were calculated within each ROTEM profile. Escalated antithrombotic therapy included clopidogrel, apixaban, or heparin. No inferential or causal interpretation is intended because ROTEM testing was incomplete, sampling times were not protocolized, and subgroups were small. "Patients N" is 57 for the four classifiable profiles and 79 for the two non-classifiable rows, matching the denominator for "Patients %". Percentages for venous thrombosis, arterial thrombosis, escalated therapy, and major bleeding use each row's "Patients n" as the denominator.

ROTEM: rotational thromboelastometry; EXTEM: extrinsically activated

Primary outcome

Clinically significant thrombosis, defined as any of the following (arterial thrombosis with graft vascular

compromise (imaging-confirmed arterial thrombus or occlusion associated with impaired arterial flow and/or prompting therapeutic anticoagulation, surgical evaluation, or interventional radiology review), persistent or progressive venous thrombus on repeat imaging (present on more than one postoperative imaging study), reoperation for thrombosis, or thrombosis-attributed pancreas graft loss (including pancreatectomy performed for thrombosis; antithrombotic therapy escalation was analyzed separately as a secondary management outcome to avoid circularity, since antithrombotic therapy was itself determined by imaging findings)), occurred in 14 of 79 patients (17.7%; 95% CI: 10.9-27.6%), based only on documented events meeting these criteria; one of these 14 met the outcome solely through thrombosis-attributed graft loss without antecedent imaging-detected thrombosis (see Discussion). By component, 11 patients had persistent or progressive venous thrombus on repeat imaging, one had arterial thrombosis with graft vascular compromise, and three had thrombosis-attributed pancreas graft loss. The components overlap in two patients: one patient with graft loss also underwent reoperation for thrombosis, and one patient with graft loss also had venous thrombus on repeat imaging. Each of the 14 patients is counted once overall, and no patient qualified through reoperation alone. Serial imaging to assess the persistence or progression of venous thrombus was unavailable for five of the 18 thrombosis-positive patients. Of these, one had documented arterial thrombosis and met the primary outcome through the arterial-thrombosis-with-compromise criterion, and one had pancreas graft failure and met the primary outcome through the graft-loss criterion; the remaining three had a functioning pancreas graft without insulin dependence at the last follow-up, a reassuring clinical course that does not, however, confirm thrombus resolution, and their status on repeat imaging could not be determined. Pancreas graft loss attributable to thrombosis occurred in three patients (3.8%) (Table 5).

Outcome	n	N	%	95% CI lower	95% CI upper	Median	Q1	Q3
Clinically significant thrombosis, n (%)	14	79	17.7	10.9	27.6	—	—	—
Pancreas graft loss due to thrombosis, n (%)	3	79	3.8	—	—	—	—	—
Pancreatectomy, n (%)	11	79	13.9	—	—	—	—	—
Interventional radiology procedure, n (%)	3	79	3.8	—	—	—	—	—
Reoperation for thrombosis, n (%)	1	79	1.3	—	—	—	—	—
Intraoperative transfusion, n (%)	15	54	27.8	—	—	—	—	—
Study-defined major bleeding, n (%)	7	79	8.9	4.4	17.2	—	—	—
Surgical washout for hematoma, n (%)	5	79	6.3	—	—	—	—	—
Kidney delayed graft function (DGF), n (%)	20	79	25.3	—	—	—	—	—
Insulin independence at discharge, n (%)	67	79	84.8	—	—	—	—	—
30-day readmission, n (%)	47	66	71.2	—	—	—	—	—
Length of stay, days	—	79	—	—	—	9	6	12.5
One-year patient survival, n (%)	70	76	92.1	83.8	96.3	—	—	—
One-year kidney graft survival, n (%)	68	76	89.5	80.6	94.6	—	—	—
One-year pancreas graft survival, n (%)	62	76	81.6	71.4	88.7	—	—	—

TABLE 5: Clinical outcomes and safety in the overall cohort

Values are presented for the overall cohort only. Study-defined major bleeding was reoperation for confirmed hemorrhage or hematoma. Kidney delayed graft function (DGF) was recorded as dialysis within the first week after kidney transplantation. Insulin independence was recorded as not being on insulin at discharge. Pancreatectomy is counted at any time from transplantation to the data lock (see Table 6). One-year graft survival counts death as graft loss. Wilson 95% confidence intervals are reported for the primary outcome, study-defined major bleeding, and one-year survival; no CIs were calculated for the other rows. Intraoperative transfusion and 30-day readmission use available-case denominators of 54 and 66 patients, respectively, rather than the full cohort of 79. One-year survival outcomes are based on the 76 recipients with at least one year of potential follow-up at the data lock (August 7, 2026). Treatment-group comparisons were omitted because treatment escalation was driven by imaging-detected thrombosis, creating substantial confounding by indication. "N" is the documented denominator for each row. CI lower/upper bounds and, for length of stay, median/Q1/Q3 are reported in separate columns rather than as combined ranges.

Treatment values reflect the actual antiplatelet or anticoagulant received, based on each patient's maximum imaging-detected thrombosis category. The single patient with arterial thrombosis also had <50% splenic vein thrombosis and is included only in the arterial-thrombosis category. Observed treatment reflects clinical practice during the study period and may differ from the idealized management algorithm shown in Figure 1.

Secondary outcomes

Study-defined major bleeding, defined here as reoperation for confirmed hemorrhage or hematoma, occurred in seven patients (8.9%). Minor bleeding was not captured as a discrete variable in the abstracted dataset. Surgical washout for hematoma was required in five patients (6.3%). Interventional radiology procedures were performed in three patients (3.8%).

Pancreatectomy was performed in 11 patients (13.9%) at any time from transplantation to the data lock (Table 6). Seven of these 11 had a graft that failed within the first year (on postoperative days 8-248 in the six with a recorded date; one had no recorded date), and four had a functioning graft at one year. Because pancreatectomy was counted over the entire follow-up period and its date was not separately abstracted, the number of pancreatectomies is not directly comparable with the number of one-year graft losses. The 14 one-year pancreas graft losses were 14 unique patients: 11 with pancreas graft failure (three of whom died before one year) and three who died with a functioning graft, which counts as graft loss. Seven involved a pancreatectomy, and seven did not. Thrombosis-attributed graft loss occurred in three patients on postoperative days 8, 16, and 120.

Patient	Day of graft failure	Recorded cause of graft failure	Attributed to thrombosis	Graft status at one year
1	8	Non-functioning	Yes	Failed
2	11	Non-functioning	No	Failed
3	16	Thrombosis	Yes	Failed
4	50	Vascular calcifications	No	Failed
5	120	Graft thrombosis	Yes	Failed
6	248	Infection	No	Failed
7	Not recorded	Not recorded	Not recorded	Failed
8	617	DM1	No	Functioning
9	887	DM1	No	Functioning
10	1537	Persistent leak	No	Functioning
11	Not recorded	Not recorded	Not recorded	Functioning

TABLE 6: Pancreatectomies and recorded causes of pancreas graft failure

Each row is one of the 11 patients who underwent pancreatectomy, numbered in order of day of graft failure (not study identifiers). The day of graft failure is counted from the date of transplantation. The date and indication of pancreatectomy were not abstracted separately, so the recorded cause of graft failure is shown. "Not recorded" means the abstracted data contained no failure date or cause. "DM1" is reproduced as recorded in the source data. Graft status at one year refers to pancreas graft function one year after transplantation; patients 8-11 had functioning grafts at one year. Patient 1's graft failure was recorded as non-functioning in the abstracted data and was attributed to thrombosis on clinical review.

Among the 76 recipients with at least one year of potential follow-up, one-year patient survival was 92.1% (70/76), kidney graft survival was 89.5% (68/76), and pancreas graft survival was 81.6% (62/76) (Table 5), counting death as graft loss; six of the 76 recipients died within the first year.

Reoperation for any cause within 30 days occurred in 26 patients (32.9%). By first-listed recorded cause, these were hemorrhage or hematoma in seven, leaks in eight (urinary tract in four, pancreatic in three, and enteric or duodenal in one), intra-abdominal infection or abscess in four, and other or unrecorded causes in seven (Table 7). Thrombosis was the recorded or attributed reason in three patients (reoperation for thrombosis in one, exploration for suspected thrombosis with preserved flow in one, and a graft pancreatectomy attributed to thrombosis in one). In exploratory unadjusted comparisons, pancreas cold ischemia time (11.2±2.2 vs. 12.1±2.5 hours; Welch t=-1.48; p=0.145) and donor age (29.1±12.1 vs. 31.2±11.0 years; Welch t=-0.74; p=0.461) did not differ between patients with and without reoperation. Transfusion after postoperative day 1 was documented for 31 of 79 patients, among whom 17 (54.8%) received a transfusion; because a blank field could reflect either no transfusion or missing documentation rather than a confirmed absence of transfusion, this proportion should be treated as a minimum estimate among documented patients rather than a cohort-wide rate. The median number of packed red blood cell units was not captured as a discrete variable.

Recorded cause (first-listed)	Patients, n	Percent of 26
Confirmed hemorrhage or hematoma	7	26.9
Suspected bleeding or hematoma, none found	2	7.7
Urinary tract leak (ureteral or bladder)	4	15.4
Pancreatic leak or fistula	3	11.5
Enteric or duodenal leak	1	3.8
Intra-abdominal infection or abscess	4	15.4
Exploration for suspected graft thrombosis, flow-preserved	1	3.8
Graft pancreatectomy	1	3.8
Kidney graft complication (Page kidney after heparin)	1	3.8
Fascial dehiscence	1	3.8
Cause not recorded	1	3.8
All reoperations within 30 days	26	100.0

TABLE 7: Recorded causes of return to the operating room within 30 days

Each of the 26 patients is assigned to one category by the first-listed cause in the abstracted free-text field; where more than one cause was recorded, the first-listed cause was used. Patient-level examples: one patient with hemorrhage at the first operation had a second operation for pancreatic thrombosis and is classified under hemorrhage; the graft pancreatectomy was attributed to thrombosis on clinical review. The timing of each reoperation within the 30 days and its operative findings were not abstracted. "Cause not recorded" means the abstracted field contained no cause.

Association between ROTEM EXTEM profile and outcomes

Among patients with a classifiable ROTEM EXTEM profile, venous thrombosis was identified in three of eight hypocoagulable patients (37.5%), 12 of 42 normocoagulable patients (28.6%), 0 of five hypercoagulable patients (0%), and 0 of two mixed/indeterminate patients (0%). Arterial thrombosis was not identified in any of the 57 classifiable patients (the single arterial thrombosis case in the overall cohort did not have a classifiable ROTEM EXTEM profile). Study-defined major bleeding occurred in one of eight hypocoagulable patients (12.5%), three of 42 normocoagulable patients (7.1%), one of five hypercoagulable patients (20%), and 0 of two mixed/indeterminate patients (0%) (Table *4*). No consistent directional relationship between ROTEM EXTEM coagulation profile and either venous thrombosis or study-defined major bleeding was observed. Because the principal ROTEM EXTEM exposure used here is a proxy for the end-of-surgery time point rather than a confirmed, protocol-defined time point, these associations should be interpreted as exploratory and hypothesis-generating rather than as evidence of a temporal or causal relationship between coagulation profile and thrombosis.

Discussion

This study describes the outcomes of severity-stratified antithrombotic management in relation to imaging-defined thrombus burden after SPK transplantation. Because formal protocol versioning was absent, the reconstructed algorithm reflects predominant institutional practice (Figure *1*), and temporal variation cannot be ruled out. The contribution is not postoperative Doppler or CT angiography itself, but the description of outcomes under a treatment-escalation approach linked to imaging severity.

In this cohort, early detection of venous and arterial thrombosis is consistent with prior literature showing that pancreas allograft thrombosis remains an important early complication after SPK transplantation [2,3].

Postoperative imaging and conservative treatment of non-occlusive pancreas allograft venous thrombosis have been described previously and are not novel in isolation. This study instead describes outcomes when antithrombotic intensity was stratified from aspirin to clopidogrel to systemic anticoagulation according to imaging-defined thrombus burden.

Donor type was also associated with venous thrombosis, with DCD donors more frequent among recipients who developed thrombosis (overall Fisher's exact test; Cramér's V=0.35; p=0.021), consistent with donor factors previously implicated in thrombosis risk [3,13]; given the small number of DCD donors in the thrombosis group (n=6) (Table *1*), this association should be interpreted cautiously. One-year patient, kidney graft, and pancreas graft survival among recipients with at least one year of potential follow-up were 92.1%,

89.5%, and 81.6%, respectively (n=76; death counted as graft loss). Kidney and pancreas graft survival were lower than contemporary national benchmarks for SPK transplantation, which use the same all-cause definition (96.2% and 90.8% at one year) [14], and the 95% confidence intervals (Table *5*) did not include those values. Six of the 76 recipients died within the first year, and each death counted as a loss of both grafts. Only three of the 14 one-year pancreas graft losses were attributed to thrombosis, so most losses were attributed to other causes or to death. This single-center cohort is small and unadjusted, so the comparison is descriptive, and differences in case mix cannot be excluded.

Prior studies suggest that non-occlusive splenic vein thrombosis may not always require immediate surgical intervention and can be managed non-operatively with anticoagulation and imaging surveillance [8]. Splenic vein thrombosis here was evaluated for severity, timing, treatment response, persistence, and resolution, with evaluation of thrombus behavior over time rather than presence alone.

The use of clopidogrel for limited venous thrombus deserves explanation. Therapeutic anticoagulation is commonly used for venous thrombosis, including non-occlusive splenic vein thrombus after pancreas transplantation, for which intravenous heparin followed by warfarin has been used [8]. We are not aware of studies evaluating clopidogrel for this indication; our data cannot establish that it is adequate. The evidence for managing limited thrombus in the pancreatic allograft, however, is sparse and unsettled. No standard protocol has been shown to prevent thrombosis consistently [3], and management has ranged from watchful waiting with serial imaging to therapeutic anticoagulation [15]. In the Cambridge single-center series, graft and patient survival did not differ between anticoagulated and non-anticoagulated recipients with grade 1 or 2 thrombosis, and the authors concluded that therapeutic anticoagulation was not necessary for grade 1 venous thrombosis [16]; in a later SPK series, graft loss due to thrombosis did not differ between recipients with grade 0 and grade 1 thrombosis [17]. The closest evidence to our approach is an earlier single-center SPK series in which the authors suggested that aspirin may be sufficient for limited, partial splenic vein thrombosis, whereas extensive splenic or superior mesenteric vein thrombosis was treated with heparin followed by oral anticoagulation [18]; that report concerned aspirin, not clopidogrel. In that series, re-exploration for bleeding was numerically more frequent among anticoagulated recipients with grade 1 or 2 thrombosis, although the difference was not statistically significant [16]. Because bleeding remains a major concern when anticoagulation is used after pancreas transplantation [2], our center-specific practice used clopidogrel as an intermediate-intensity strategy for thrombus involving <50% of the vessel, with escalation to anticoagulation for more extensive or progressive thrombosis. The grading used in those studies is not equivalent to our <50% and ⩾50% categories, so they support the plausibility, but not the adequacy, of this step. Descriptively, thrombus persisted on repeat imaging in six of the nine patients managed with clopidogrel, and one lost the graft to thrombosis on postoperative day 8 (Results), which further limits any inference about its adequacy. The clopidogrel step reflects institutional practice; the present data do not establish clopidogrel as equivalent or preferable to anticoagulation, and it is not offered as a recommendation but warrants prospective comparison with anticoagulation.

Bleeding remains a major concern when anticoagulation is used after pancreas transplantation [2]. In this cohort, study-defined major bleeding occurred in seven patients (8.9%) and surgical washout for hematoma in five (6.3%). Because thromboprophylaxis practices vary widely, direct comparison of bleeding rates is challenging [4].

Return to the operating room within 30 days occurred in about one-third of recipients (26 of 79, 32.9%), which is high in absolute terms, and published series using broader early postoperative windows have reported rates in a similar range. In a series of 255 SPK recipients, early relaparotomy within three months, a longer window than ours, occurred in 32.9% overall, falling from 43% to 14.4% after the center restricted donor age and shortened pancreas cold ischemia time, and re-operative rates of 12-44% have been summarized across centers [19]. In our cohort, hemorrhage or hematoma, leaks, and intra-abdominal infection accounted for 19 of the 26 reoperations, and thrombosis was the recorded or attributed reason in three (Table *7*). The early reoperation burden was therefore driven predominantly by other postoperative surgical complications, whereas in that series, vascular complications, particularly thrombosis, were the most common indication [19]. Pancreas cold ischemia time was an independent predictor of early relaparotomy in that series [19]; neither it nor donor age differed between patients with and without reoperation in our small, unadjusted exploratory comparison. Because the timing and operative indication for each reoperation were not abstracted separately, and treatment was assigned based on imaging findings, these data cannot show whether antithrombotic therapy contributed to the seven hemorrhage or hematoma reoperations. The high early reoperation rate nonetheless indicates that imaging-guided antithrombotic management is set against a background of substantial early surgical morbidity, which is relevant when weighing the intensity of anticoagulation.

Exploratory ROTEM EXTEM analysis showed no consistent relationship with thrombosis or study-defined major bleeding. Incomplete testing, small subgroups, nonstandardized sampling, and possible treatment before measurement preclude the use of these data to guide therapy.

The relationship between the ROTEM EXTEM profile and imaging-detected thrombosis was explored descriptively in the 57 classifiable patients. Venous thrombosis was numerically more common among hypocoagulable (37.5%) and normocoagulable (28.6%) patients than among hypercoagulable or

mixed/indeterminate patients (0% in both groups). This non-directional pattern likely reflects small hypercoagulable/mixed profile counts (five and two patients), the exploratory classification used, and possible confounding by therapy escalation before sampling.

Bleeding rates did not show a consistent gradient across profiles (12.5% hypocoagulable, 7.1% normocoagulable, and 20% hypercoagulable, the last based on a single event among five patients), not supporting a simple monotonic relationship and likewise limited by small cell counts. Whether ROTEM EXTEM measured at a single, imprecisely timed point meaningfully adds to imaging surveillance cannot be determined here; a prospectively timed assessment with complete parameter capture would be needed before concluding its clinical utility, though sample size will likely remain limiting. In this sensitivity analysis, three patients were reclassified as insufficient-to-classify (54 of 79 were fully classifiable) (Table *4*); venous thrombosis and bleeding rates were materially unchanged (normocoagulable venous thrombosis: 30.8% versus 28.6% in the primary analysis), supporting the classification's robustness.

This study has several strengths: it addresses a clinically important problem using objective imaging findings, follows STROBE guidance for observational research, and describes real-world implementation of a severity-stratified antithrombotic management approach with detailed imaging, treatment, bleeding, and graft outcomes. Outcome and exposure variables were explicitly defined in the Methods, and denominators were reported for every proportion rather than defaulting to the full cohort, thereby limiting the risk that variable-level missingness would be obscured. The ROTEM EXTEM classification was additionally tested in a sensitivity analysis that required complete parameter capture, thereby supporting the robustness of that exploratory classification.

This study has important limitations. Its retrospective, single-center design limits causal inference and generalizability. The small cohort and event counts produced imprecise estimates and precluded reliable multivariable adjustment. Imaging timing varied with clinical status, potentially introducing surveillance bias, and one thrombosis-attributed graft loss occurred without antecedent imaging-detected thrombosis, indicating that imaging-based stratification did not capture all cases of clinically significant thrombosis. Treatment decisions may reflect unmeasured factors, and without a contemporaneous comparator, the observed thrombosis, bleeding, and graft outcomes cannot be attributed to the management approach. In addition, the use of clopidogrel for limited venous thrombus lacks direct supporting evidence (the available reports concern aspirin), and this study cannot determine whether it was adequate compared with therapeutic anticoagulation. ROTEM EXTEM was classifiable in only 57 of 79 patients, lacked standardized timing, and used individual reference intervals because no validated composite EXTEM index exists. Some treatment-duration, laboratory, and follow-up imaging data were incomplete: ROTEM EXTEM was unavailable in 22 of 79 patients (27.8%), and five of the 18 patients with venous thrombosis had no follow-up imaging, limiting the assessment of thrombus persistence and resolution. The number of patients screened and excluded was not recorded, so selection bias cannot be assessed. Serial imaging recorded whether thrombus was present but not its extent, so the venous component of the primary outcome reflects persistent thrombus on repeat imaging and may overstate true progression. One-year outcomes were assessed among the 76 recipients with at least one year of potential follow-up, so these estimates are based on a smaller denominator and have wide confidence intervals. The date and indication of pancreatectomy were not abstracted separately, so the date of graft failure and its recorded cause are reported instead; the 11 pancreatectomies are only partially explained by the recorded causes of graft failure (Table *6*). Pancreatectomy and graft loss were assessed over the full follow-up without prespecified time windows, so these cumulative counts are not directly comparable with the one-year outcomes. Early morbidity was substantial: 26 of 79 recipients (32.9%) returned to the operating room within 30 days, and 47 of the 66 with a documented readmission status (71.2%) were readmitted within 30 days; the causes of readmission were not abstracted, and neither rate can be attributed to the antithrombotic strategy. Reoperation was abstracted as any return to the operating room within 30 days with a free-text cause; the categories in Table *7* were assigned from that text, and the timing and operative findings of each reoperation were not abstracted. Because escalation was triggered by thrombosis, the treatment category was described as a management outcome rather than analyzed as an independent exposure. Donor type classification required reconciling two source fields that were occasionally discordant or inconsistently coded; because donor type was the only variable associated with venous thrombosis at conventional significance thresholds, this finding should be interpreted with particular caution. Because the management approach was reconstructed from clinical records rather than governed by a version-controlled protocol, and because imaging timing, ROTEM sampling, and treatment decisions followed clinical judgment, the study cannot be replicated under identical conditions; the definitions, thresholds, and denominators are reported so that the analysis can be reproduced from comparable records, but the management itself would require prospective standardization.

Despite these limitations, the study describes real-world severity-stratified management and its clinical outcomes. Treatment escalation based on imaging-defined thrombus burden was implemented, but the data do not establish superiority over alternative prophylaxis or anticoagulation strategies. ROTEM EXTEM findings were exploratory and non-directional. Prospective studies with standardized imaging and treatment criteria are needed to assess comparative effectiveness and safety.

Conclusions

In this single-center retrospective cohort, postoperative imaging guided antithrombotic intensity according to thrombus severity. Venous thrombosis occurred in approximately one-fifth of recipients and clinically significant thrombosis in 14 (17.7%), whereas arterial thrombosis occurred in one patient (1.3%); study-defined major bleeding occurred in seven patients (8.9%) and thrombosis-attributed pancreas graft loss in three (3.8%).

Exploratory ROTEM EXTEM profiles showed no consistent association with thrombosis or study-defined major bleeding. These findings describe the implementation and outcomes of severity-stratified management but do not establish comparative effectiveness or safety. Prospective studies with standardized imaging and treatment criteria are needed.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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