DISTAL ARTERIOVENOUS FISTULA INHIBITS EXPERIMENTAL ANASTOMOTIC INTIMAL THICKENING

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ANASTOMOTIC INTIMAL hyperplasia is recognized as a major cause of prosthetic bypass failure. This nonatherosclerotic form of intimal thickening typically involves distal end-to-side arterial anastomoses where major alterations in the near wall flow velocity profiles and wall shear stress occur as a result of the geometric configuration and outflow conditions. Regions of low and oscillatory shear have been shown to be associated with anastomotic intimal thickening (AIT). To test the hypothesis that increased flow and shear stress prevent AIT, we measured AIT in polytetrafluoroethylene (PTFE) anastomoses with a distal arteriovenous fistula and compared the findings to control vein and PTFE anastomoses.

MATERIALS AND METHODS

Bilateral iliofemoral grafts were implanted in 11 male mongrel dogs using a standard distal end-to-side anastomotic geometry with a hood length-to-vessel diameter ratio of 4:1. In four animals (Group I), a unilateral side-to-side superficial femoral arteriovenous fistula (AVF) was constructed 15 cm downstream from the PTFE anastomoses on one side; the contralateral PTFE anastomosis without an AVF served as a control. In seven animals (Group II) reverse saphenous vein or PTFE grafts were implanted without an AVF. Volumetric flow rate was measured in all animals. After 12 weeks, animals were killed and the anastomoses pressure perfusion fixed. Sequential histologic sections from each anastomosis (n = 60–80) were studied, and regions of intimal thickening were quantitated using computer-assisted morphometry.

Table 1—Effect of arteriovenous fistula on flow and anastomotic intimal thickening

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<tr>
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<th>Group I (n = 4)</th>
<th>Group II (n = 7)</th>
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<td>PTFE with AVF</td>
<td>PTFE</td>
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<td>Flow (mL/min)</td>
<td>392 ± 40*</td>
<td>173 ± 40</td>
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<tr>
<td>AIT (μm)</td>
<td>0.05 ± 0.01†</td>
<td>0.24 ± 0.02</td>
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*P < 0.005; †P < 0.03. Values expressed as mean ± SEM.

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RESULTS

Mean blood flow (mL/min) was increased to more than twofold in PTFE grafts with AVF (n = 4) compared to control vein (n = 7) or PTFE grafts without AVF (n = 11, P < 0.005). There was marked reduction in AIT in PTFE anastomoses with AVF (0.05 ± 0.01 μm) compared to both Group I and Group II PTFE anastomoses without AVF (0.24 ± 0.02 and 0.19 ± 0.01, respectively, P < 0.0001). AIT in PTFE with AVF was also significantly less than in Group II vein anastomoses (P < 0.03) (Table 1).

CONCLUSIONS

An inverse relationship between the magnitude of shear stress and experimental intimal thickening has been previously demonstrated. The findings of the present study indicate that increasing blood flow and shear by creating a distal AVF virtually eliminates AIT in experimental PTFE anastomoses. Similar interventions, or other means, such as exercise, that enhance volume flow may prevent the development of occlusive intimal hyperplasia and improve long-term patency of PTFE grafts. Further study of the hemodynamically induced molecular and cellular events associated with progression and regression of AIT in relation to wall shear stress is warranted.

REFERENCES