ABSTRACTS

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Antiplatelet effect of aspirin in patients with cerebrovascular disease

Alberts MJ, Bergman DL, Molner E, et al. Stroke 2004;35:175-8.

Conclusion: More than half of patients taking enteric-coated aspirin(ASA) or low-dose ASA have normal platelet function. Women and older patients are less likely to have a positive therapeutic response to aspirin independent of aspirin formulation or dose.

Summary: Platelet function in patients with cerebrovascular disease who were taking only ASA as an antiplatelet agent was analyzed. A platelet function analyzer (PFA-100), a commercially available benchtop device, was used to evaluate platelet function. ASA dose, formulation, and basic demographic factors were correlated with PFA platelet function results.

One hundred and twenty-nine patients with a diagnosis of stroke, transient ischemic attack, or cerebrovascular disease were entered in the study. A total of 39 patients were taking 81 mg of ASA every other day or daily. Of these, 22 (56%) had normal PFA results. Normal PFAs were also present in 24 of 87 patients (28%) taking 325 mg per day of ASA, ($P = .001$). An enteric-coated form of ASA was taken by 41 patients (32%). ASA resistance was 65% in patients taking an enteric-coated ASA preparation. ASA resistance was 25% in patients taking uncoated ASA preparations, ($P < .001$). Patients older than 63 years had a decreased therapeutic response to ASA independent of dose ($P = .048$) and preparation ($P = .034$). Men had a stronger association between ASA dose and a therapeutic PFA response than women (odds ratio, 5 versus 2.5).

Comment: Up to 40% of patients with stroke are taking ASA at the time of their event (Stroke 2001;32:2559-66). Failure of ASA therapy to prevent stroke may be related to patient compliance, drug interactions, and stroke mechanisms not responsive to ASA or ASA resistance. This study is critical in that it indicates that ASA resistance may be in part due to the dosage and formulation of ASA. In addition, older patients and women may be less likely to respond to ASA therapy. Demographic factors may identify patients with cerebrovascular disease who should be preferentially treated with antiplatelet agents other than ASA.

A polymer-based, paclitaxel-eluting stent in patients with coronary artery disease

Stone GW, Ellis SG, Cox DA, TAXUS-IV Investigators. N Engl J Med 2004;350:221-31.

Conclusion: Compared with bare metal stents, paclitaxel-eluting stents markedly reduce angiographic and clinical restenosis at 9 months following coronary stenting procedures.

Summary: This was a randomized, prospective, double-blind, multicenter trial conducted at 73 US centers. Patients with previously untreated coronary artery stenosis (vessel diameter, 2.5 to 3.75 mm; lesion length, 10-28 mm) who were to receive a stent for a single previously untreated coronary artery stenosis were randomly allocated to receive a bare metal stent or a paclitaxel-eluting stent. A total of 652 patients received the bare metal stent and 662 received the drug eluting stent. At 9 months, angiographic follow-up was prespecified in 732 patients.

Mean vessel diameter was 2.7 mm, mean lesion length was 13.4 mm, and diabetes was present in 24.2% of patients. At 9 months, ischemia-driven target vessel revascularization was 12.0% in the patients treated with bare metal stents and 4.7% in patients treated with the drug-eluting stent (relative risk, 0.39; 95% confidence interval, 0.26-0.59; $P < .001$). Target lesion revascularization was also significantly less in the drug-eluting stent group than in the bare metal stent group (3.0% versus 11.3%; relative risk 0.27; 95% confidence interval, 0.16-0.43; $P < .001$). Angiographic restenosis was reduced in the drug-eluting stent patients (7.9% versus 26.6%; $P < .001$). Stent thrombosis occurred in 0.6% of the patients receiving a drug-eluting stent and 0.8% of the patients receiving a bare metal stent.

Comment: Paclitaxel inhibits cellular division, signal transduction, and motility. Its effect on coronary artery restenosis when delivered via a coronary artery stent appears similar to that of sirolimus delivered via coronary stents. Cardiologists now have two drug-eluting stents from which to choose. It appears reasonable, and likely, that both types of drug-eluting stents will eventually be evaluated in peripheral arteries as well.

Explaining racial variation in lower extremity amputation: A five-year retrospective claims data and medical record review at an urban teaching hospital

Rucker-Whitaker C, Feinglass J, Pearce WH. Arch Surg 2003;138:1347-51.

Conclusion: Racial disparities in amputation rates between African American patients and white patients are due to higher rates of repeat amputation among African American patients and not a higher rate of primary amputation.

Summary: Population-based data suggests African American patients undergo major lower extremity amputation up to three times more frequently than white patients. To determine whether this disparity is due to treatment choice or severity of disease, the authors studied rates of primary major amputation and repeat amputation at a large Midwestern teaching hospital. The risk of primary amputation was derived from analyzing data for all patients undergoing lower extremity bypass, angioplasty, or major amputation over a 5-year period. Sixty African American major amputees were compared with 60 white major amputees for risk of repeat amputation. Primary and repeat amputation rates were controlled for age, sex, and diabetes mellitus.

African American patients hospitalized for lower extremity ischemia were younger ($P < .05$), more often female ($P < .01$), and more likely to undergo major amputation (odds ratio, 1.68; $P = .005$). After adjusting for age, sex, and diabetes prevalence, African American and white patients had an equal likelihood of primary amputation. Repeat amputees were 2.5 times more likely to be African American than white ($P = .04$).

Comment: Racial disparities in revascularization versus amputation are documented (Med Care 2002;40 Suppl:1106-16). The current study suggests these differences may be due to severity and progression of disease rather than access to treatment. Race, however, is clearly a surrogate for potential cultural and socioeconomic factors that can influence health care. Additional data evaluating effectiveness of social support and efficacy of management of atherosclerotic risk factors in the white versus African American amputees must be considered before conclusions derived from this study can considered definitive rather than speculative.

Preoperative and operative predictors of delayed neurologic deficits following repair of thoracoabdominal aortic aneurysm

Estrera AL, Miller CC III, Huynh TTT, et al. J Thorac Cardiovasc Surg 2003;126:1288-94.

Conclusion: Acute aortic dissection, Type II thoracoabdominal (TA) aortic aneurysms, and preoperative renal dysfunction are predictors of delayed neurologic deficit following thoracic and TA aortic aneurysm repair.

Summary: The authors analyzed the results of 828 consecutive patients undergoing thoracic or TA aortic repair surviving long enough for assessment of postoperative neurologic status. Demographic and preoperative physiologic, as well as intraoperative, data were evaluated with multivariable analysis to determine factors associated with delayed neurologic deficit. Delayed neurologic deficit was defined as paraplegia or paraparesis that occurred after a patient had recovered from anesthesia and was initially neurologically intact.

There were 38 patients with immediate neurologic deficits and 21 (2.7%) with delayed deficits following TA aorta repair. The univariate predictors of delayed neurologic deficit included acute dissection (odds ratio, 3.9; $P < .05$), preoperative renal dysfunction (odds ratio, 5.9; $P < .06$), Type II TA aorta (odds ratio, 3.0; $P < .03$) and use of cerebrospinal fluid drainage and distal aortic perfusion (odds ratio, 7.7; $P < .03$). With a multivariable model, cerebrospinal fluid drainage and distal aortic perfusion were not significant. With optimization of blood pressure and cerebrospinal fluid drainage, 12 of 21 (57%) of patients with delayed neurologic deficit recovered neurologic function.

Comment: The risk factors identified for delayed neurologic deficit following TA aortic repair are similar to those associated with immediate neurologic deficit following TA aortic repair. Since reinstitution of cerebrospinal fluid drainage results in a high rate of neurologic recovery, the obvious question is how long should cerebrospinal fluid drainage be maintained postoperatively to minimize development of a delayed neurologic deficit?