

Frequency of allergy to ticlopidine among patients allergic to clopidogrel (and vice versa)

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Background: The two currently available thienopyridines, clopidogrel and ticlopidine, are structurally very similar, differing only by a carboxymethyl side group. In patients allergic to one thienopyridine, the likelihood that an allergy will also develop to the other is unknown.

Methods: We analyzed the electronic health record and performed a retrospective chart review of all patients with allergic or hematologic adverse reactions to clopidogrel or ticlopidine who were ever prescribed the other thienopyridine. Details of the allergy or adverse reaction and the subsequent clinical course were carefully reviewed. Dermatologic or hematologic side effects, angioedema, or anaphylaxis, which developed when the thienopyridine was initiated and resolved with its discontinuation, were included in this study. No patients with thrombotic thrombocytopenic purpura were challenged with the other thienopyridine. Gastrointestinal intolerance was *not* included in this study.

Results: We identified 76 patients with an allergy or hematologic adverse reaction to either clopidogrel ($n=52$) or ticlopidine ($n=38$) who also received the other thienopyridine. Their mean age was 69 years; 30% were female. The allergic or adverse reactions were dermatologic in 89%, angioedema in 5%, thrombocytopenia in 4%, and neutropenia in 3%; no patient developed anaphylaxis. Thirty-eight (73%) of the clopidogrel-allergic patients and 24 (63%) of the ticlopidine-allergic patients tolerated the other thienopyridine well. Fourteen (18%) patients had allergic or hematologic adverse reactions to both drugs. Therefore, among patients with allergic or hematologic adverse reactions to clopidogrel, 27% also had similar reactions to ticlopidine. None developed a life-threatening reaction requiring emergency therapy.

Conclusions: In patients with allergic or hematologic adverse reactions to a thienopyridine, there appears to be an increased frequency of such reactions to the other thienopyridine. In patients who develop what is believed to be an allergy (mostly rash) to clopidogrel, life-threatening reactions were not seen in patients subsequently treated with ticlopidine.

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Effect of rosiglitazone on endothelial function and inflammatory biomarkers in nondiabetics undergoing percutaneous intervention

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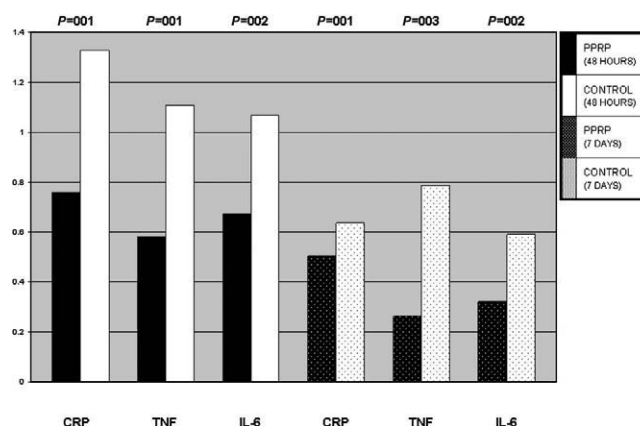
Objective: The goal of the study was to determine whether administration of rosiglitazone prior to percutaneous coronary intervention (PCI) blunts the rise of inflammatory biomarkers CRP, tumor necrosis alpha (TNF- α), and interleukin-6 (IL-6), and improves endothelial function.

Background: Elevation of inflammatory biomarkers surrounding PCI has been associated with increased death, nonfatal myocardial infarction, and restenosis.

Methods: Sixty nondiabetic patients undergoing PCI for a positive stress test or acute coronary syndrome were randomized to rosiglitazone therapy ($n=30$) or standard care ($n=30$). Samples were collected before PCI, 48 h, and 7 days, and concentrations of hsCRP, TNF- α , and IL-6 were measured. Brachial artery vasodilation was measured at the same time.

Results: Levels of hsCRP, TNF- α , and IL-6 were significantly lower in the rosiglitazone group at 48 h and 7 days. There was no statistically significant difference in brachial artery flow-mediated vasodilation.

Conclusion: The rise in inflammatory biomarkers following PCI is reduced by treatment with rosiglitazone in nondiabetics. There was no measurable change in endothelial function. This is the first study to demonstrate that treatment with the PPAR gamma agonist rosiglitazone can significantly attenuate this inflammatory response. Further studies are needed to assess whether this finding translates into clinical benefit.



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Outcomes of paclitaxel-eluting stent implantation as an initial treatment strategy for sirolimus-eluting stent failure

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Background: The long-term efficacy of paclitaxel-eluting stent (PES) implantation for the treatment of sirolimus-eluting stent (SES) restenosis is not known.

Methods: Patients were considered eligible for inclusion if (1) the initial lesion treated by the SES was de novo; (2) no other modality had been used to treat the SES restenosis previously; and (3) at least 1 year had passed since PES implantation.

Results: A total of 128 consecutive patients with 147 restenotic (ISR) SES lesions were treated with PES. Mean patient age was 64 ± 12 years, diabetes was present in 40%, and the target lesion was focal in 69%. Clinical follow-up was complete in 100% at a median of 454 days post-procedure (range, 12 to 30 months). Death occurred in two patients (1.5%) and myocardial infarction in one patient (0.8%). There were no episodes of stent thrombosis. The incidence of target lesion revascularization (TLR) was 23.8%. The median time to TLR was 251 days (range, 75 to 511 days). There was no difference in TLR between focal and diffuse lesions (25.5% vs. 26.7%, $P=ns$), although time to clinical TLR was significantly shorter in diffuse lesions ($P=.02$). The only characteristics associated with TLR were a smaller post-procedure minimal luminal diameter (MLD) and greater post-procedure diameter stenosis.

Conclusions: An initial treatment strategy of PES implantation for SES restenosis appears safe and provides adequate outcomes at more than 1-year follow-up. Special attention should be paid to achieving an optimal angiographic outcome, since smaller post-procedure MLD and greater residual stenosis are associated with longer-term failure. Given that TLR was not infrequent in our study, a randomized trial of PES vs. standalone angioplasty for SES restenosis should be considered.

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Successful treatment of refractory angina by minimally invasive delivery of autologous bone marrow mononuclear cells: long-term follow-up

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