

## SUMMARY

During the last decades percutaneous coronary intervention (PCI) has become a major weapon against coronary artery disease. This approach was compromised by the fact that restenosis was the main limitation. Great efforts have been made in resolving this vexing problem. Although the angiographic restenosis rate has been substantially reduced by stenting, especially with the drug eluting stents, there still remains a problem particularly in a subset of patients and lesions. The predominant mechanism in the development of in-stent restenosis is intimal hyperplasia and anti-proliferative agents have been used for the elimination of restenosis (*Toutouzas et al., 2004*).

Inflammation plays an important role in the pathogenesis of atherosclerosis and restenosis. Several markers are known to represent the systemic inflammatory status, among them are C-reactive protein (CRP) and IL-6. Several studies have shown that high plasma levels of inflammatory markers predict the extent of atherosclerosis and subsequent coronary events among healthy individuals (*Segev et al., 2004*).

In order to assess the inflammatory response following coronary artery stenting with DES or BMS and the effect of in-stent restenosis on inflammatory markers hs-CRP and IL-6 documented with 6 month follow up coronary angiography in patients with stable coronary artery disease, a series of 100 patients with stable angina who underwent stenting in native single vessel coronary artery were included in this study.

**Patient with any of the following criteria were excluded from the study:**

1. Acute coronary syndrome.
2. Surgery within previous month.
3. Known inflammatory, neoplastic, or infectious disease.
4. Treatment with steroids, immunosuppressive drugs, or non steroidal anti-inflammatory drugs except acetylsalicylic acid.
5. Previous CABG, or Coronary angiography with or without PCI within the previous month.
6. Marked renal or hepatic impairment.
7. CRP level > 3mg/L prior to coronary angiography .

Many variable were considered in the analysis of patients data including basic characters (age and sex), risk factors (hypertension, diabetes, dyslipidemia, smoking), echocardiographic finding (EF%, WMSI), angiographic findings and interventional procedure details including (number, type, and size of stents, length and width of deployed stents, lesion type and TIMI flow).

Inflammatory markers including hs-CRP measured preprocedural, 24 h post procedural and after 6 month before follow up coronary angiography, as well as IL-6 was done preprocdural and after 6 month before follow up coronary angiography. All patients were followed closely during hospital stay.

Patients were classified into two group according the type of deployed stents DES group and BMS stent group, and based on the results of the

follow up coronary angiography each group subdivided into two subgroups instent restenosis group and patent stent group.

All patients were followed closely during hospital stay. After patients discharge the follow-up protocol included a telephone interview and out patient clinic at 30 days, 3 months and 6 months after the procedure with follow up cardiac examination, ECG and echocardiography and by review of the hospital medical records for outpatient clinic or emergency room visits. The major adverse cardiac events were reported including cardiac related death, nonfatal reinfarction, and recurrent ischemic pain requiring hospitalization.

Preprocedural inflammatory markers hs-CRP and IL-6 were not predictive of instent restenosis detected by follow up coronary angiography after 6 month or major adverse cardiovascular events (MACEs), and there was no statistical difference between BMS group and DES group with or without restenosis as regarding preprocedural inflammatory markers hs-CRP and IL-6.

Although the inflammatory response, measured by post procedural hs-CRP was significantly more intense ( $P < 0.0001$ ) among patients with deployed BMS than those with deployed DES, however even in patients in whom only DES were implanted, there was significant difference between preprocedural and post procedural hs-CRP ( $P < 0.0001$ ).

We followed our patients clinically for the occurrence of MACEs in the form cardiac related death, myocardial infarction and recurrent ischemia, as well as angiographic follow up was done after 6 month for detection of instent restenosis.

In-stent restenosis was more common in patients with BMS than those with deployed DES (  $P = 0.047$ ). 24 h post-procedural hs-CRP was higher in patients with restenosis than those without among patients with DES ( $P = 0.02$ ) and all of them were in 24h post procedural hs-CRP tertile III , as well as for BMS group 24h post-procedural hs-CRP was higher in patients with restenosis than those without ( $P=0.02$ ) and six patients were in tertile III and one was in tertile II .

After 6 month serum hs-CRP was higher in patients with restenosis than those without for patients with DES ( $P < 0.0001$ ) and for BMS group ( $P < 0.0001$ ), as well as 6 month follow up of serum IL-6 was higher in patients with restenosis than those without for DES group ( $P < 0.0001$ ), while for BMS group ( $P < 0.0001$ ).

More over we found that in patients with MACEs had higher post procedural hs-CRP than those without among DES group ( $P=0.039$ ), six patients were in tertile III and three patients were in tertile II, the same observation was found for BMS group with ( $P=0.01$ ), nine patients were in tertile III and two patients were in tertile II.