Background and objective: Hepatotoxicity induced by hepatotoxins has been regarded as one of the most serious health problems. The present work aimed to investigate the possible protective effects of carvedilol against paracetamol-induced hepatotoxicity. **Methodology:** Thirty two male rats were randomly divided into four groups as follows: vehicle control, hepatotoxicity control, N-acetyl cysteine (300 mg/kg; p.o.) and carvedilol (30 mg/kg; p.o.). Seven days after initiation of treatments, hepatotoxicity was induced by a single oral administration of paracetamol (1 g/kg). At the end of the experimental period, blood samples were collected for estimation of serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH) and gamma-glutamyl transferase (GGT) activities as well as serum total protein (TP) level as markers of hepatic dysfunction. In addition, hepatic malondialdehyde (MDA), glutathione (GSH) and nitric oxide (NO) contents were assessed as oxidative and nitrosative stress markers. Serum tumor necrosis factor alpha (TNF-α) and interleukins-1beta (IL-1β) levels were also determined as inflammatory markers. Moreover, histopathological and immunohistochemical studies were performed. **Results:** Paracetamol administration resulted in a significant elevation of ALT, AST, LDH and GGT activities , MDA and NO contents, as well as TNF-α and IL-1β levels coupled by significant reduction of TP level and GSH content. Pretreatment with carvedilol mitigated paracetamol-induced biochemical, histological and immunohistochemical changes. **Conclusion:** It has been concluded that carvedilol could alleviate hepatotoxicity induced by paracetamol, most probably through its antioxidant and anti-inflammatory properties. It may be of therapeutic value in treatment of paracetamol-induced hepatotoxicity