Diabetes is one of the most important causes of endstage renal failure. The main reason for renal damage in diabetes is metabolic and hemodynamic changes induced by hyperglycemia(1). However, increasing evidence shows that more complex mechanisms play a role in diabetic nephropaty. Oxidative damage is one of these mechanisms. Hyperglycemia increases the production of reactive oxygen species in the kidney (2). Silibinin is a potent flavanoid antioxidant derived from a plant called Silybum Marianum. Previous studies have indicated the antioxidant effects of silibinin on different pathologies and organs(3). The aim of this experimental study was to investigate the effects of silibinin on the kidney tissue of Streptozotocin (STZ)-induced diabetic rats through histochemical methods. There were five groups in our study as follows: Control group, Diabetic Group, Treatment Group 1 (diabetic group treated with 100 mg/kg silibinin), Treatment Group 2 (diabetic group treated with 200 mg/kg silibinin), and Silibinin Group (no diabetes but 5 rats treated with 100mg/kg and 5 rats treated with 200 mg/kg silibinin). STZ was administered at a dose of 65mg/kg by intraperitoneal injection and Silibinin was administered by gastric gavage for 4 weeks. In contrast to some previous studies, we could not find any significant restorative effect of silibinin on renal damage. The diabetic group and the treatment groups demonstrated similar histological findings. These conflicting results can be related to the differences between the experimental designs. The role of oxidative stress in diabetic rat kidney damage is clear, but silibinin has no effect on these molecular mechanisms.