OBJECTIVE:
To establish a model of hematopoietic stem and progenitor cells with iron overload derived from umbilical cord blood (UCB) cells and explore the effects of reactive oxygen species (ROS) on the hematopoiesis of hematopoietic stem and progenitor cells with iron overload.

METHODS:
The model was established by adding different concentrations (50, 100, 200, 400 µmol/L) of ferric citrate (FAC) into mononuclear cells from UCB and culturing for different times (6, 12, 24 h). The UCB cells were divided into 4 groups: control group, group FAC, group FAC+N-acetyl-L-cysteine (NAC) and group FAC+ L-Glutathione (GSH). Then the changes of ROS, labile iron pool (LIP), apoptosis, the capacity of hematopoietic colony forming (CFU-E, BFU-E, CFU-GM, CFU-mix) and the percentage and the numbers of CD34(+), CD33(+), GlyA(+) cells were detected. And the changes of these indices were tested after the treatment of iron overload UCB with antioxidants (NAC and GSH).

RESULTS:
UCB cells were cultured with the addition of FAC at different concentrations for different times. The level of total ROS increased in time and concentration-dependent manners. The intracellular level of ROS peaked when cultured at 200 µmol/L of FAC for 24 hours. Cells were treated with antioxidants NAC or GSH after cultured with 200 µmol/L FAC for 24 hours. Then the ROS levels of total cells, myeloid cells and erythroid cells decreased markedly versus normal controls. The LIP of total cells, myeloid cells and erythroid cells increased markedly when cells were cultured at 200 µmol/L of FAC for 24 hours versus normal controls (P < 0.05). NAC and GSH had no effect on the level of LIP. The apoptotic rates of FAC-treated cells [(20.90 ± 3.45)%] increased significantly versus normal controls [(9.20 ± 1.29)%] (P < 0.05). The capacity of hematopoietic colony forming in FAC treated cells decreased markedly versus normal controls. The percentage and numbers of CD34(+), CD33(+), GlyA(+) cells of FAC-treated cells also decreased significantly versus normal controls (P < 0.05). And these changes could be recovered by the addition of NAC or GSH.

CONCLUSION:
Oxidative stress plays an important role in the injuries of hematopoiesis of hematopoietic stem and progenitor cells with iron overload by inducing the generation of ROS. These findings may help us find a specific target and improve the therapeutic efficacy of ineffective hematopoiesis in patients with iron overload.