Metrifonate (trichlorfon) is an inhibitor of acetylcholinesterase (AChE) used as pesticide and Alzheimer’s disease (AD) drug. The primary aim of the experiment was to judge implication of oxidative stress after metrifonate administration and compares it with AChE inhibition level. Wistar rats were subcutaneously exposed to either 25 or 50 % of metrifonate LD_{50} and compared to the controls treated with saline only. Cerebral cortex and livers were collected from animals 40 minutes after exposure. Activity of AChE, glutathione reductase, glutathione-S-transferase, caspase 3, total protein level, thiobarbituric acid reactive substances (TBARS), reduced glutathione level, ferric reducing antioxidant power (FRAP) were assayed in the tissue samples. Though activity of liver AChE was decreased, there were not found significant alterations in the followed stress markers. Contrary to it, cerebral cortex tissue had not only decreased AChE activity due to metrifonate, but also increased activity of caspase 3 and glutathione reductase. FRAP level and total proteins were also increased in cerebral cortex due to metrifonate. Increased FRAP level even exceeded the AChE inhibitory impact. Perceptiveness of metrifonate to modulate oxidative stress in AD suffered brain is discussed. It is estimated that metrifonate can be helpful in AD patients not only due to expected inhibition of AChE but also due to the mentioned pro-antioxidant effect.