Introduction: Within the Delusional Misidentification Syndrome (DMS) the Capgras syndrome and its variants show high prevalence. It is clinically characterized by the delusional belief that someone close, very well known, usually a close relative or a family member, has been replaced by an impostor with extreme physical appearance. Literature review: In Capgras syndrome are found morphological changes quite varied. In almost all cases there are bilateral cortical atrophy, predominantly in the right hemisphere, especially in the frontal, parietal and temporal. And some studies like Ramachandran et al. found the presence of enlargement of the ventricles, especially of the right ventricle. Ramachandran and Hirstein propose that the Capgras syndrome (CS) is given by the disconnect between the ventral visual processing pathways in the inferior temporal lobe and the limbic system, particularly the amygdala. In another words, the patient has the ability intact or even exaggerated to remember individual episodes, but cannot make a lasting connection, creating a common denominator between them. CS may also be associated - occur by - with overactive dopaminergic. Josephs (2007) describes the case of two patients who developed CS immediately after methamphetamine abuse, suggesting that this syndrome may be related to dopamine dysregulation - the drug causes loss the dopamine transporter. It would also explain the high association with CS and dementia with Lewy bodies, in which there is loss of dopamine transporters at the expense of neuronal loss. Conclusion: The presence of changes in brain morphology and physiology for the emergence of the syndrome is undeniable, which explains the high prevalence of the disorder in degenerative diseases. Since its first description by Capgras and Reboul-Lachaux in 1923, many theories have been overturned, and now more studies are needed to confirm what happens in the brain to its appearance.