

Discussion

The present study demonstrates that the mean serum TSA and LSA levels in patients with RA was significantly increased compared to normal, this results confirm those of studies that observed high plasma or serum TSA and LSA in patients with cardiovascular disease, inflammatory reaction and different types of cancer^[14-17].

TSA includes a small amount of free sialic acid as well as glycoprotein and glycolipid-bound sialic acid, these oligosaccharide chains, with sialic acid on the N-terminal position, are present on the cell surface. Sialic acid induces an electronegative charge^[12] because, as a relatively strong acid ($pK_a=2.6$), it is completely ionized at physiological pH^[18].

This ionization plays a major role because the distribution of cell surface dense anionic sites is correlated, in vitro, with abnormal cell aggregation^[19]. The mechanism of increased serum SA level in inflammatory conditions is unclear, but several explanations have been proposed these include: (a) spontaneous release of aberrant SA-containing cell surface glycoconjugates, (b) increased levels and /or glycosylation of normal serum glycoproteins^[16].

One of hypothesis of increase of plasma or serum TSA levels in patients with inflammatory state could be explained by an increased output of serum proteins from the liver due to some type of acute phase protein. This also requires an increase in the activity of sialidase, which will catalyze and remove the sialic residues from an acute proteins^[20].

References

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