The cause why apoptotic sperm have poor morphology profile as assessed by the sperm deformity index (SDI): A prospective study

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Objective: Externalization of phosphatidylserine (PS) to the sperm outer membrane leaflet is considered to mark terminal apoptosis. Magnetic cell sorting (MACS) using paramagnetic annexin V-conjugated microbeads eliminates spermatozoa with externalized PS. The relationship between apoptosis and the sperm morphology has not been well characterized. The SDI is a novel quantitative expression of sperm morphological quality that has an enhanced predictive power and reproducibility. The aim of this study was to chart the shift in sperm morphology profile between the non-apoptotic and apoptotic sperm fractions.

Design: Prospective study.

Materials and Methods: Semen specimens collected from 50 healthy donors were prepared by density gradient centrifugation (DGC) followed by MACS using annexin V-conjugated microbeads. The procedure delivers 2 sperm fractions: annexin-negative (non-apoptotic) and annexin-positive (apoptotic). Sperm morphology in the DGC preparations and the non-apoptotic and apoptotic sperm fractions was assessed using the Tygerberg’s strict criteria. A multiple entry technique was utilized to calculate the SDI scores and to assess the frequency of different sperm deformities in different sperm fractions. The SDI score was calculated by dividing the total number of deformities observed by the number of sperm randomly selected and evaluated irrespective of their morphological normality. Pair-wise comparisons were made using Wilcoxon’s signed ranked test.

Results: The non-apoptotic sperm fractions had significantly lower median SDI score compared to the apoptotic sperm fractions (p<0.0001) and to DGC preparations (p<0.0001). The non-apoptotic sperm fractions had significantly lower median percentage sperm with acrosomal defects and cytoplasmic droplets compared to the apoptotic sperm fractions (p<0.0001, p<0.03, respectively) and to DGC preparations (p<0.0001, p=0.0002, respectively). On the other hand, the median percentage of sperm with midpiece defects was significantly higher in the non-apoptotic and apoptotic sperm fractions compared to the DGC preparations (p=0.03, p<0.0001, respectively). The non-apoptotic sperm fractions and the DGC preparations had similar median percentages of sperm with tail defects but the percentage was significantly higher in the apoptotic sperm fractions. The median percentages of sperm with normal morphology using Tygerberg’s strict criteria were similar in the non-apoptotic sperm fractions and the DGC preparations, but significantly higher in the non-apoptotic sperm fractions compared to the apoptotic sperm fractions.

Conclusion: The poor sperm morphology profile seen in the apoptotic sperm fractions may be partly due to the isolation of sperm with acrosomal damage and/or cytoplasmic droplet. It may also be partly due to
a technique-induced damage to sperm midpiece and tail. The damage to the midpiece was significant even in the non-apoptotic sperm fractions. Under these circumstances the SDI is a more sensitive research tool than percentage of normal morphology in evaluating the shift in sperm morphology profile. Financial Support: None.

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