Obstructive sleep apnea associated with Diabetes mellitus Type 2: a proteomic study

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INTRODUCTION

We previously showed that Obstructive sleep apnea (OSA), a common public health concern causing deleterious cardiometabolic dysfunction, induces alterations in red blood cell (RBC) proteome, including redox/oligomeric state of PRDX2 as putative biomarker for OSA severity and/or PAP (positive air pressure) therapy monitoring1-8 (Figure 1). Herein, we aimed to investigate whether OSA patients with Type 2 Diabetes Mellitus before and after positive airway pressure (PAP) treatment present similar changes in the RBC antioxidant protein PRDX2 to better understand the molecular basic mechanisms associated with OSA and OSA outcomes.

METHODS

RBC samples from Snorers (n=22 being 3 diabetics) and OSA patients before and after six month of PAP-treatment (n=29 being 8 diabetics) were analysed by non-reducing western blot using antibody against PRDX2 or PRDX2SO2 to measure the total and overoxidized levels of monomeric/dimeric/oligomeric forms of PRDX2. Groups were statistically compared and correlated with clinical/biochemical data and significance set up at 5% (p value< 0.05).

RESULTS

We confirmed our previously data showing higher overoxidation on monomeric forms of PRDX2 in OSA RBC that after PAP treatment decreased followed by an increase of multimeric-overoxidized forms associated with chaperone protective function (Figure 2B, Figure 3B). In contrast, in diabetic OSA RBC, although the level of monomeric PRDX2 was significantly higher abundant compared with OSA RBC without this comorbidity (Figure 2A), its levels of overoxidation was significantly lower (Figure 2B). After PAP treatment, the level of monomeric PRDX2 decreased but not its overoxidation level in diabetic OSA RBC cells (Figure 2A, 2B). The level of oxidized (S-S/S-S) and overoxidized (SO2) dimer forms were lower in diabetic OSA RBC compared with OSA RBC without this comorbidity (Figure 2C, 2E). After treatment, the level of overoxidation in these dimers decreased in OSA RBC without comorbidity but not in diabetic OSA RBC (Figure 2E).

CONCLUSIONS

The redox/oligomeric state of RBC PRDX2 regulated by overoxidation of the active cysteines were differentially modulated in diabetic OSA patients compared to OSA without this comorbidity. PAP-induced overoxidized oligo forms of PRDX2 associated with chaperone protective function showed decreased in OSA patients with diabetes.

The clinical impact of all these findings needs further investigation and validation.

Table

Correlation of redox/oligomeric state of PRDX2 to biochemical & metabolic variables under study

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To ICAT-2019 organizers for congress attendance

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