Personalized management of patients with pulmonary tuberculosis considering melatonin levels

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Conflict of interest: none

BACKGROUND. Infectious diseases, particularly those caused by Mycobacterium tuberculosis (TB), remain a major global health challenge and impose a substantial burden on society. Melatonin is an endogenous hormone with multiple biological properties. Accumulating evidence indicates that melatonin may play a significant role in the management of infectious diseases, including TB.
OBJECTIVE. To improve the effectiveness of pathogenetic treatment in patients with pulmonary TB during the intensive phase by incorporating melatonin, considering its therapeutic properties.
MATERIALS AND METHODS. A randomized clinical study enrolled 117 TB patients, including 60 individuals with pulmonary TB, who were divided into two groups of 30 patients each. Group 1 (main) included patients with drug-sensitive pulmonary TB who received standard anti-TB therapy combined with melatonin. Group 2 (control) consisted of patients who received anti-mycobacterial therapy alone.

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RESULTS AND DISCUSSION. In patients with pulmonary TB, the inclusion of melatonin as part of pathogenetic therapy alongside anti-mycobacterial treatment is accompanied by a significant increase in its serum concentration (from 33.2±1.28 to 41.96±3.95 pg/mL). Melatonin contributed to a statistically significant reduction in hepatic enzymes (ALT and AST), indicating its hepatoprotective and anti-inflammatory effects. The median level of C-reactive protein at the end of the intensive phase was substantially lower in the melatonin-supplemented group.

The administration of melatonin was associated with a statistically significant decrease in interleukin-6 level ($r=-0.52$; $p=3.19\times10\text{-}4$) and a tendency toward higher interleukin-10 level, reflecting its modulatory influence on the immune system. The pathogenetic use of melatonin in the complex management of patients with pulmonary TB accelerates radiological regression, which may be attributed to its capacity to modulate immune responses and promote reparative processes within the pulmonary tissue.

CONCLUSIONS. Enhanced pathogenetic therapy with adjunctive melatonin during the intensive treatment phase promotes activation of nonspecific immunity and reduces tissue damage resulting from the infectious process. Thus, melatonin increases and mediates the patient's resistance to mycobacterial infection while attenuating the harm caused by the host inflammatory response.

KEY WORDS: tuberculosis, melatonin, resistance, pathogenetic therapy, treatment.