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Respiratory Failure after Hematopoetic Stem Cell Transplant

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Introduction

It might be difficult to diagnose and manage pulmonary problems following allogeneic hematopoietic stem cell transplantation (HSCT). A baseline pre-transplant respiratory assessment with pulmonary function testing (PFT) and a computed tomography (CT) scan of the chest is required, especially for older patients and smokers, to enable the interpretation of post-HSCT explorations. The whole list of factors that contribute to the increased survival seen in HSCT patients admitted to the ICU is not entirely known. The presence or absence of a malignant process, which may predispose to more intensive chemotherapy or radiation prior to transplant, and renal replacement therapy, which may predispose to fluid overload and worsening respiratory status, poor wound healing, and increased work of bleeding, are variables noted to have a significant impact on the outcome [1].

Description

Through the detection of patterns in gene polymorphisms or through other methods, it may be possible to prospectively screen for patients who are at high risk. This would enable prophylaxis to be tailored for individual individuals in order to reduce cytokine release during the first few days following a bone marrow transplant. This would stop the cascade that can lead to respiratory failure and acute respiratory distress syndrome. Even in very small patients, renal replacement therapy, continuous veno-venous hemofiltration, or dialysis were available for early intervention. This may have prevented massive fluid overload, pulmonary edoema, and the requirement for high ventilator settings to achieve adequate gas exchange due to poor pulmonary compliance. Although it is still unknown how these factors affect specific patients, ventilation is a viable option for the majority of kids who experience respiratory failure after receiving a bone marrow transplant and can increase their chances of long-term survival [2].

A baseline pre-transplant respiratory assessment with pulmonary function testing (PFT) and a computed tomography (CT) scan of the chest is required, especially for older patients and smokers, to enable the interpretation of post-HSCT explorations. The diagnostic strategy then calls for thorough clinical evaluation of the patient, accurate interpretation of lung CT scans, and knowledge of the various lung entities that can complicate the follow-up of HSCT. The respiratory clinical symptoms are unfortunately not very specific. These problems are diagnosed in various ways. Based on a chest CT scan or PFT conducted sequentially during HSCT follow-up, or during the investigation of respiratory symptoms or isolated fever, it might be fortuitous. Post-HSCT noninfectious lung problems such impaired lung function, bronchiolitis obliterans syndrome, or idiopathic pneumonia syndrome may also be linked to respiratory viral infections after HSCT. Due to alveolar rupture that causes an air leak into the pulmonary interstitium with retrograde dissection through the perivascular sheath into the hilum and into the mediastinum, patients report with severe chest discomfort and impairment of respiratory function [3].

Respiratory distress, severe tachypnea, laboured breathing, and activation of accessory respiratory muscles are the symptoms of severe acute respiratory failure. The most typical pulmonary infiltration pattern, diffuse bilateral infiltrates, may be seen. Most studies of acute respiratory failure featured patients receiving less than 6 L/min of conventional oxygen, and patients with acute respiratory failure need oxygen therapy.

Mesenchymal stem cells (MSCs) have numerous remarkable immunomodulatory and extracellular matrix remodelling effects through secretion of various types of cytokines, growth factors, and tissue mediators. As one of their therapeutic effects in restoring the pulmonary microenvironment, safeguarding alveolar epithelial cells, preventing pulmonary fibrosis, and treating lung dysfunction and COVID-19 pneumonia, they modulate the cytokine storm or balance immune responses [4,5].

Conclusion

Due to many factors, these individuals rapidly acquire severe pneumonia, moderate-to-severe ARDS, and septic shock. One of the main reasons of death is the emergence of refractory hypoxemia, and the systemic inflammatory response syndrome (SIRS) may be the pathogenesis's primary deadly element. Our observations show that from 1 to 12 months after normal therapy and combination therapy with MSC transplantation, the majority of clinical complaints improved. Before MSC applications may be used in clinical practise, a few typical side effects still need to be addressed. Even though MSC transplantation has produced a number of encouraging results, the issue of long-term safety is still up for question, especially given how challenging it is to manage long-term follow-up for all patients. The second issue is that MSC can create new blood vessels, which may encourage tumour development and metastasis in addition to having the ability to suppress tumour immune responses.

Acknowledgement

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Conflict of interest

None declared.

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