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ANALYSIS OF CYTOCHEMICAL CHARACTERISTICS OF PERIPHERAL BLOOD MONOCYTES IN COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN UNDER ONE YEAR OF AGE WITH CONGENITAL HEART DEFECTS

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Abstract: Community-acquired pneumonia (CAP) remains a leading cause of morbidity in infants, particularly when complicated by comorbid conditions such as congenital heart defects (CHD). The hemodynamic disturbances associated with CHD create a unique pathological environment that alters the immune response at the cellular level. This article presents a study conducted jointly by Tashkent State Medical University and Andijan State Medical Institute. Using the IMRAD framework, the research investigates the metabolic activity of peripheral blood monocytes by analyzing key enzymes: succinate dehydrogenase (SDH), acid phosphatase (AP), and myeloperoxidase (MPO). The study involved 58 children aged 6 months to 3 years, stratified by the presence of CHD and the severity of pneumonia. The results demonstrate that children with CHD exhibit profound metabolic suppression in immune cells, characterized by significantly reduced SDH activity and elevated lysosomal enzyme activity (AP) during the acute phase. The study concludes that cytochemical monitoring of monocytes provides a sensitive marker for assessing the severity of metabolic disturbances and predicting the clinical course of pneumonia in children with cardiac anomalies.

Keywords: community-acquired pneumonia, congenital heart defects, monocytes, cytochemistry, succinate dehydrogenase, acid phosphatase, myeloperoxidase, infants.

АНАЛИЗ ЦИТОХИМИЧЕСКИХ ОСОБЕННОСТЕЙ МОНОЦИТОВ ПЕРИФЕРИЧЕСКОЙ КРОВИ ПРИ ВНЕБОЛЬНИЧНОЙ ПНЕВМОНИИ У ДЕТЕЙ В ВОЗРАСТЕ ДО ОДНОГО ГОДА НА ФОНЕ ВРОЖДЕННЫХ ПОРОКОВ СЕРДЦА

Аннотация: Внебольничная пневмония (ВП) остается одной из ведущих причин заболеваемости у детей грудного возраста, особенно при наличии сопутствующих заболеваний, таких как врожденные пороки сердца (ВПС). Гемодинамические нарушения, связанные с ВПС, создают уникальную патологическую среду, которая изменяет иммунный ответ на клеточном уровне. В данной статье представлено исследование, проведенное совместно Ташкентским государственным медицинским университетом и Андижанским государственным медицинским институтом. Используя структуру IMRAD, в работе исследуется метаболическая активность моноцитов периферической крови путем анализа ключевых ферментов: сукцинатдегидрогеназы (СДГ), кислой фосфатазы (КФ) и миелопероксидазы (МП). В исследовании приняли участие 58 детей в возрасте от 6 месяцев до 3 лет, стратифицированных по наличию ВПС и тяжести пневмонии. Результаты показывают, что у детей с ВПС наблюдается глубокое метаболическое подавление иммунных клеток, характеризующееся значительно сниженной активностью СДГ и повышенной активностью лизосомальных ферментов (КФ) в острой фазе. Исследование делает вывод, что цитохимический мониторинг моноцитов является

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чувствительным маркером для оценки тяжести метаболических нарушений и прогнозирования клинического течения пневмонии у детей с кардиальными аномалиями.

Ключевые слова: внебольничная пневмония, врожденные пороки сердца, моноциты, цитохимия, сукцинатдегидрогеназа, кислая фосфатаза, миелопероксидаза, дети раннего возраста.

TUG‘MA YURAK NUQSONLARI FONIDA BIR YOSHGACHA BO‘LGAN BOLALARDA KASALXONADAN TASHQARI PNEVMONIYADA PERIFERIK QON MONOTSITLARINING TSITOKIMYOVIY XUSUSIYATLARINI TAHLIL QILISH

Annotatsiya: Kasalxonadan tashqari pnevmoniya (KTP) go‘dak yoshidagi bolalar o‘rtasida kasallanishning asosiy sabablaridan biri bo‘lib qolmoqda, ayniqsa tug‘ma yurak nuqsonlari (TYN) kabi hamroh kasalliklar bilan kechganda. TYN bilan bog‘liq gemodinamik buzilishlar hujayra darajasida immun javobni o‘zgartiruvchi o‘ziga xos patologik muhitni yaratadi. Ushbu maqolada Toshkent davlat tibbiyot universiteti va Andijon davlat tibbiyot instituti hamkorligida o‘tkazilgan tadqiqot natijalari keltirilgan. IMRAD tuzilmasiga asoslangan ushbu ish suksinat degidrogenaza (SDG), nordon fosfataza (NF) va mieloperoksidaza (MPO) kabi asosiy fermentlarni tahlil qilish orqali periferik qon monotsitlarining metabolik faolligini o‘rganadi. Tadqiqotda 6 oylikdan 3 yoshgacha bo‘lgan 58 nafar bola ishtirok etdi va ular TYN mavjudligi hamda pnevmoniyaning og‘irlik darajasiga qarab guruhlarga ajratildi. Natijalar shuni ko‘rsatadiki, TYN bo‘lgan bolalarda immun hujayralarining chuqur metabolik susayishi kuzatiladi, bu o‘tkir davrda SDG faolligining sezilarli darajada pasayishi va lizosomal fermentlar (NF) faolligining oshishi bilan tavsiflanadi. Tadqiqot shunday xulosa qiladiki, monotsitlarning tsitokimyoviy monitoringi metabolik buzilishlarning og‘irligini baholash va yurak nuqsonlari bo‘lgan bolalarda pnevmoniyaning klinik kechishini prognoz qilish uchun sezgir marker hisoblanadi.

Kalit so‘zlar: kasalxonadan tashqari pnevmoniya, tug‘ma yurak nuqsonlari, monotsitlar, tsitokimiya, suksinat degidrogenaza, nordon fosfataza, mieloperoksidaza, erta yoshdagi bolalar.

INTRODUCTION

Respiratory diseases continue to occupy a prominent position in the structure of pathology among young children, presenting a persistent challenge to pediatric healthcare. In recent years, significant data regarding the diagnosis and treatment of pneumonia in infants have been accumulated, allowing for new therapeutic approaches. However, numerous problems remain unresolved, necessitating further in-depth study [4, 6]. A primary factor complicating the clinical landscape is the anatomical and physiological peculiarities of the respiratory system in early childhood. Fundamentally, almost all biochemical processes in the body are mediated directly or indirectly by cellular membranes; therefore, alterations in membrane structure and function are intrinsically linked to the pathogenesis of many diseases [7].

In characterizing the immunological status of a child suffering from community-acquired pneumonia (CAP), the state of the macrophage system is of paramount importance. Consequently, the functions of mononuclear phagocytes and their enzymatic profiles have been, and remain, a subject of rigorous investigation. The degree of functional impairment in these cells determines the extent of pathological changes at the organismal, tissue, and cellular levels. Based on these premises, the cytochemical study of the enzymatic spectrum of blood cells, particularly monocytes, can be utilized to assess the depth of metabolic disturbances and the state of organism reactivity at the cellular level.

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Most indicative of these metabolic states are the enzymes myeloperoxidase (MPO), acid phosphatase (AP), and succinate dehydrogenase (SDH). It is, therefore, of significant clinical interest to study the activity of SDH, MPO, and AP relative to the stage of the disease. A particularly unfavorable background for the course of the pneumonic process in young children is the presence of comorbidities such as rickets, protein-energy malnutrition, anemia, and Congenital Heart Defects (CHD). These conditions often determine the recurrent nature of pneumonia, the duration of the illness, and the propensity for exacerbations and complications [5, 6]. Congenital heart defects are anatomical anomalies of the heart and great vessels leading to circulatory disorders. In defects with a pathological shunt in the pulmonary circulation, a disturbance in the ventilation-perfusion ratio develops in the lungs. This causes a reduction in gas exchange and arterial hypoxemia. These changes contribute to the formation of respiratory failure and significantly complicate the course of pneumonia.

Despite certain successes in treating pneumonia patients [4, 5], the search for new pathogenetically substantiated approaches to diagnosis and management continues. Of particular interest is the early diagnosis of the course and outcome of CAP against the background of CHD. The objective of this research is to determine the clinical and biochemical features of community-acquired pneumonia in young children with comorbid congenital heart defects to optimize the diagnosis and treatment of the disease.

METHODS

This observational study was conducted under the auspices of Tashkent State Medical University and Andijan State Medical Institute. The study population consisted of 58 children aged between 6 months and 3 years. The cohort was stratified to analyze the impact of congenital heart defects on the course of pneumonia. The participants included children diagnosed with community-acquired pneumonia against a background of CHD, including a subgroup of 22 patients with CAP complicated by cardiorespiratory, toxic, and circulatory manifestations, and 18 patients presenting with purulent-destructive pneumonia. A control group was comprised of 20 healthy children of the same age and demographic background.

In addition to standard general clinical and radiological examinations, all patients underwent specific cytochemical analysis of peripheral blood monocytes. These assessments were performed at three distinct time points: upon admission to the hospital, during the acute period of the disease, and during the period of clinical improvement and recovery. The enzymatic activity of monocytes was evaluated using established cytochemical methods. Myeloperoxidase (MPO) activity was determined according to the Quaglino method. Acid phosphatase (AP) activity was assessed using the Goldberg and Barka method, with the subsequent calculation of the mean cytochemical coefficient (MCC). Succinate dehydrogenase (SDH) activity was measured using the quantitative cytochemical method developed by R.P. Narcissov.

The clinical assessment focused on the duration of fever, the persistence of local physical signs (rales), and the development of complications. Specifically, symptoms of respiratory and cardiovascular distress were monitored, including tachypnea, dyspnea during feeding and physical exertion, and signs of physical development delay. The specific heart defects monitored included Ventricular Septal Defects (VSD) and Atrial Septal Defects (ASD), which are characterized by left-to-right shunting.

RESULTS

The clinical observations revealed a severe disease trajectory in children with CAP and comorbid CHD. In 32 percent of these patients, the disease was characterized by prolonged fever

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and toxicosis lasting more than 6 days. Local rales persisted for more than 12 days in 18.4 percent of cases. Furthermore, destruction of lung tissue was observed in 11.3 percent of patients as early as 3 days after the initiation of treatment. In these patients, despite active antibacterial therapy, non-intensive infiltrative shadows appeared in the lungs on days 3-5, increasing in volume and intensity over time. The pneumonia assumed an aggressive character, accompanied by progressive severe respiratory failure. The leading symptoms were disturbances of the respiratory and cardiovascular systems, including tachypnea and dyspnea during feeding.

The biochemical investigation revealed distinct enzymatic patterns correlating with the disease phase and severity. Succinate dehydrogenase (SDH) activity showed a significant depression during the acute phase of the illness compared to the control group ($P < 0.001$). The lowest levels of SDH were observed when the clinical picture was most severe, interpreted as a cellular response to metabolic shifts, specifically acidosis, which was present in the majority of these children. SDH activity only approached normal levels towards the moment of recovery, manifesting the highest activity during the convalescent phase.

Conversely, the activity of Acid Phosphatase (AP) changed in a diametrically opposite manner relative to the stage of the pneumonic process. The highest activity of this enzyme was detected during the acute period of the disease ($P < 0.001$). While it gradually decreased towards the moment of recovery, it remained elevated compared to healthy children. These results indicate a robust activation of blood cell lysosomes in response to the infectious agent.

The activity of Myeloperoxidase (MPO) was unstable and did not show a clear dependency on the period of CAP in the general CHD group. However, the level of this enzyme correlated clearly with the form of pneumonia and the premorbid background. This was particularly evident in patients with purulent-destructive pneumonia against the background of CHD. In these patients, MPO activity was significantly elevated during all periods of the disease. By the time of recovery, it exceeded the age-specific norm by 1.5 times ($P < 0.001$). The elevation of MPO likely reflects a high intracellular concentration of myeloperoxidase system components participating in phagocytosis. Similarly, in destructive pneumonia, AP activity also peaked in the acute period and remained elevated at recovery, while SDH showed marked depression initially with a rapid increase during the dynamic improvement of the pathological process.

DISCUSSION

The findings of this study underscore the complexity of managing community-acquired pneumonia in children with congenital heart defects. The presence of a left-to-right shunt (as seen in VSD and ASD) leads to pulmonary congestion and altered ventilation-perfusion ratios. This hemodynamic burden exacerbates the infectious process, leading to the prolonged clinical symptoms and higher rates of destructive complications observed in the study cohort. The delay in physical development and fatigue common in these infants further reduces their physiological reserve to fight infection.

The cytochemical analysis provides a window into the cellular mechanism of this susceptibility. The significant reduction in SDH activity during the acute phase indicates a failure in the Krebs cycle and mitochondrial energy production within the monocytes. This "energetic starvation" of immune cells likely impairs their ability to effectively combat pathogens, leading to the aggressive course of the disease. The acidosis resulting from hypoxia and infection further inhibits key metabolic enzymes.

On the other hand, the elevation of Acid Phosphatase and Myeloperoxidase confirms active mobilization of the lysosomal apparatus. The persistent elevation of these enzymes even during the recovery phase suggests a state of prolonged immune activation and potentially incomplete

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resolution of the inflammatory process at the cellular level, despite clinical improvement. This discrepancy highlights the value of cytochemical monitoring; it can reveal hidden metabolic strain that standard clinical exams might miss. The specific profile of enzyme activity—low SDH combined with high AP and MPO—can serve as a diagnostic marker for the severity of the inflammatory response in children with CHD.

CONCLUSION

Community-acquired pneumonia developing against the background of congenital heart defects is distinguished by a severe and protracted clinical course, characterized by prolonged toxicosis and a tendency towards lung tissue destruction. The study confirms that the presence of cardiac anomalies fundamentally alters the metabolic response of the immune system.

The cytochemical evaluation of peripheral blood monocytes reveals deep metabolic disturbances. Specifically, the acute phase is marked by a significant depression of succinate dehydrogenase activity, reflecting mitochondrial energy failure, and a compensatory increase in acid phosphatase activity, indicating lysosomal stress. The persistence of elevated MPO levels in destructive forms suggests an aggressive phagocytic response that may contribute to tissue damage.

These findings provide a basis for optimizing the management of these patients. Therapy should not only target the infectious agent but also include metabolic support to correct cellular energy deficits. Furthermore, the cytochemical profile of monocytes can be used as an additional criterion for assessing the efficacy of treatment and determining the true point of recovery. Future research should focus on developing targeted metabolic therapies to support immune cell function in this vulnerable pediatric population.

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