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Chapter 1

EPIDEMIOLOGY, MICROBIOLOGY AND DIAGNOSIS OF HELICOBACTER PYLORI

Cundullah TORUN[1]

1. EPIDEMIOLOGY AND TRANSMISSION

Helicobacter pylori (H. pylori) was first cultured by Marshall and Warren in 1982. Despite references to spiral-shaped microorganisms in the gastric mucosa nearly a century before this discovery, they had not been isolated due to the lack of suitable culture conditions. Recognizing the curved, gram-negative rod shape similar to Campylobacter species, the researchers established a microaerophilic culture environment and observed growth after 5 days. Initially named Campylobacter-Like Organism (CLO) for its resemblance, it was later renamed Campylobacter pylori in 1984 and ultimately designated H. pylori in 1989 by Goodwin and colleagues (1).

It is estimated that approximately half of the world's population is infected with H. Pylori (2). The prevalence varies among countries and different socioeconomic groups. In recent years, a decline in H. pylori prevalence has been observed, particularly in countries where the infection is common (3). In Japan, a country with a high incidence of gastric cancer, the prevalence of H. pylori among those born before 1950 was 90%, whereas it decreased to 2% among those born after 2000 (4). This significant decrease cannot be solely explained by age-related exposure risk. Factors such as improved sanitation facilities, changes in family structure, and frequent use of antibiotics and proton pump inhibitors (PPIs) contribute to this trend.

Asia, Central and South America are regions where H. pylori infection is most widespread. Low socioeconomic status, underdeveloped sanitation facilities, and crowded family structures have been identified as risk factors (5). Consequently, developing countries exhibit a higher prevalence compared to developed countries. Once H. pylori colonizes the gastric mucosa, it persists throughout life

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Overall, understanding the epidemiology, microbiology, and pathogenicity of H. pylori is crucial for effective management and treatment strategies. The decline in prevalence in some regions suggests that public health measures and advancements in healthcare may contribute to controlling H. pylori-related diseases. Ongoing research and advancements in diagnostic methods will further enhance our ability to detect and manage H. pylori infections, ultimately improving patient outcomes and reducing the burden of associated diseases.

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Chapter 2

COMPARISON OF ATHEROSCLEROTIC RISK FACTORS IN PATIENTS WITH SECONDARY AMYLODOSIS AND NEPHROTIC SYNDROME

Osman CURE[1]
Kuddusi CENGIZ[2]

INTRODUCTION

Kidney diseases are an important public health problem. Proteinuria, which occurs as a result of primary and secondary diseases of the kidney, is closely associated with cardiovascular disease, cholesterol abnormalities and hypertension (1). Proteinuria increases the susceptibility to arteriosclerosis by causing deterioration in endothelial functions, inflammation, lipid abnormalities and coagulation. Nephrotic syndrome is a disease with severe proteinuria, low albumin, high lipid, edema and tendency to clot. There are many studies in the literature showing that the risk of cardiovascular disease increases in nephrotic syndrome (2,3). A second disease that causes nephrotic proteinuria by affecting the kidney is secondary amyloidosis (AA). In amyloidosis, the possibility of developing heart and vascular disease is accelerated due to amyloid deposition in vascular structures, inflammatory process and complications of nephrotic syndrome. The literature on cardiovascular diseases is mostly related to primary amyloidosis. It has been determined that the cardiovascular system is 50-75% in primary amyloidosis cases and 10% in secondary amyloidosis cases (4,5). There are quite a number of publications indicating the relationship between nephrotic syndrome and cardiovascular disease, among these two groups of diseases, very well (6-9). On the other hand, literature data on lipoprotein levels, which pose a cardiovascular risk, are less in patients with secondary amyloidosis with proteinuria (10).

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Secondary amyloidosis is characterized by the accumulation of serum amyloid A (SAA) protein in tissues as a result of ongoing inflammation due to rheumatological diseases such as Familial Mediterranean Fever and rheumatoid arthritis, chronic infections such as tuberculosis and inflammatory bowel diseases. SAA is an acute phase reactant. Serum amyloid A protein, whose level can increase with inflammatory stimuli, is synthesized mainly in the liver in association with HDL-C. However, studies conducted in recent years have shown that SAA protein is found in endothelium, atherosclerotic lesions, and smooth muscle cells. SAA protein found in atherosclerotic lesions is thought to play a role in lipid metabolism by causing uptake or excretion of lipids at the cellular level. In addition, it is thought that SAA may cause remodeling of vascular walls or plaques by increasing collagenase synthesis from smooth muscles. SAA protein also plays a role in thrombus formation by impairing the assembly and adhesion of platelets in the endothelium (25-27).

Limitations: Our study has several limitations. The small number of cases, the lack of randomized controlled studies, and the single-center design.

CONCLUSIONS

Atherosclerotic risk factors of patients with nephrotic syndrome presenting with primary glomerulonephritis and secondary amyloid with equal proteinuria were significantly higher than the control group. In addition, patients with secondary amyloidosis should be closely monitored for other atherosclerotic risk factors as well as amyloid accumulated in organs. In addition, we believe that primary disease treatment and atherosclerotic risk factors and proteinuria should be treated in diseases with proteinuria.

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Chapter 3

INVESTIGATION OF THE FREQUENCY AND CAUSES OF ANEMIA IN PATIENTS WITH HYPOTHYROIDISM IN THE EASTERN BLACKSEA REGION

Kadir ILKKILIC[1]
Ekrem ALGUN[2]

INTRODUCTION

Hypothyroidism is the clinical condition that occurs with decreased production of thyroid hormones. Although its prevalence varies by region, it has been reported as 2-5% worldwide (1,2). Hypothyroidism and anemia are common diseases in society, and anemia accompanying thyroid diseases is a clinical condition that should not be ignored. The incidence of anemia in hypothyroid patients varies between 20% and 60% (4,5). Although the most common type of anemia is normochromic normocytic anemia, it is also seen in hypochromic microcytic and macrocytic anemia (8). Thyroid hormones have an important role in the proliferation of erythroid precursors and contribute to erythropoiesis by increasing erythropoietin (Epo) levels (3). In hypothyroidism, metabolism slows down and the oxygen need of tissues decreases as an adaptation to the hypometabolic state. This situation leads to inadequate erythropoietin stimulation, causing normochromic normocytic anemia. Additionally, iron malabsorption due to thyroid hormone deficiency or hypochromic microcytic anemia due to iron loss may develop. In addition, macrocytic anemia may develop in hypothyroid patients due to vitamin B12 and folic acid malabsorption or lack of intake (6). Additionally, in patients with autoimmune thyroid disease, anemia may develop due to accompanying autoimmune diseases such as pernicious anemia, celiac disease, and atrophic gastritis (4,7).

In our study, we aimed to reveal the frequency and subtypes of anemia in patients diagnosed with hypothyroidism in the Eastern Blacksea region.

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Anemia seen in patients diagnosed with hypothyroidism may occur through a variety of mechanisms. In this study, we found that the frequency of anemia increases in newly diagnosed hypothyroidism patients and the most common anemia subtype is chronic disease anemia**.**

CONCLUSION

We found that the frequency of anemia increases in newly diagnosed hypothyroidism patients and the most common anemia subtype is chronic disease anemia. We think that evaluating patients diagnosed with hypothyroidism in terms of the presence and etiology of anemia will contribute positively to the treatment strategy of clinicians.

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Chapter 4

COMPLETE BLOOD CELL COUNT AND HEPATOKINES AS INFLAMMATORY MARKERS IN OBESITY

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INTRODUCTION

Obesity is a complex, multifactorial and treatable disease, which is characterized by low-grade inflammation. It contributes to various chronic metabolic disorders. Hepatokines along with adipokines and myokines, play regulatory roles in inflammation via various pathways. Obesity is a well-known risk factor for complications during adolescence including type 2 diabetes mellitus (T2DM), metabolic syndrome (MetS), cardiovascular diseases, cancer, dyslipidemia, hypertension, polycystic ovary syndrome (PCOS), and non-alcoholic fatty liver disease (NAFLD). Obesity is also a well-known contributor to insulin resistance (IR). Obesity-induced inflammation leads to IR. There are also some mechanisms pointing out the association between IR and liver dysfunction (1-7).

Pediatric obesity is a serious health problem, which may lead to obesity in adulthood. With the increasing prevalence of obesity in children, NAFLD has become one of the most common causes of chronic liver disease in children (8-11). Noninvasive biomarkers related to the matter are gaining importance (12).

The effects of PCOS are amplified by obesity, therefore, PCOS is an obesity-related condition (1,13). However, PCOS is difficult to diagnose during adolescence because normal pubertal development overlaps with the characteristic features of the syndrome (14).

Studies on the member profiles of complete blood cell count (CBC) analysis in obesity were performed. Increases in total white blood cell (WBC) count as well as in platelet (PLT) count were observed during inflammation in obese individuals. High blood pressure was associated with high hemoglobin (Hgb) and hematocrit (Hct). Increased body mass index (BMI) values were associated with increased RBC, WBC, PLT, Hgb and Hct. These observations pointed out that alterations

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Chapter 5

EFFECTS OF INSULIN AND THYROID HORMONES ON ANTIOXIDANT ENZYMES AND LIPID PEROXIDATION PRODUCTS IN DIABETES MELLITUS

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INTRODUCTION

Diabetes Mellitus (DM) is an endocrine and metabolic disease characterized by complete or partial insufficiency of insulin secretion by the pancreas or insufficient insulin effect, manifested by hyperglycemia and characterized by carbohydrate, lipid, and protein metabolism disorders (1). Studies have shown that free oxygen radicals and lipid peroxidation are significantly increased in rats with experimentally induced diabetes and in diabetic patients, and oxidative stress has been reported to play a role in the etiology and progression of diabetes (2).

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increased SOD activity in diabetic rat kidney (26). Contrary to most studies, the increased SOD activity in this study is attributed to the stimulation of enzyme activity by the increased production of superanion radicals.

Volkovova et al. In their study on diabetic rat kidneys, they found that SOD activity did not change, and GPx activity increased (30). Similarly, Elmalı et al. They also found that SOD activity did not change, and GPx activity increased (31). In the results of our study, it was observed that SOD activity did not change significantly in the diabetic (DM) group compared to the control group (C), while GPx activity increased especially in the diabetic (DM) group compared to the control group (C). Even though the changes in GPx enzyme activity are statistically significant, there are no significant differences between the groups.

CONCLUSION

Therefore, the results suggest that the possible role of thyroid hormones in insulin regulation of impaired antioxidant enzyme activity in diabetes is not seen in the kidney (unlike in the liver and heart) and may be most likely related to the duration of diabetes.

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Chapter 6

GOUT DISEASE AND CURRENT TREATMENT

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INTRODUCTION

Gout is a chronic inflammatory disease that develops due to the accumulation of monosodium urate (MSU) crystals in the joints and periarticular tissues. It is the most common cause of inflammatory arthritis (1). Age, male sex, dietary habits, genetic, and environmental factors play a role in its development. High serum uric acid (>6.8 mg/dL) is the most important risk factor for gout disease. Recurrent monoarthritis attacks are typically observed. In patients who do not receive appropriate treatment and have been diagnosed for a long time, polyarticular involvement, joint damage, and tophi may be seen (2). Changes in dietary habits are made to lower uric acid levels, and urate-lowering medications are used in patients as needed.

EPIDEMIOLOGY AND RISK FACTORS

The prevalence in developed countries is higher than in developing countries (3). In Europe and North America, the prevalence ranges from 1% to 4%. Its frequency increases with age (4). In Turkey, patients are most commonly diagnosed in the fifth and sixth decades. It is four times more common in males than in females. It is more commonly seen in older ages in females (5).

The most important risk factor is hyperuricemia. However, not every patient with hyperuricemia develops gout (asymptomatic hyperuricemia). Even in people with severe hyperuricemia (≥10 mg/dL), the rate of patients developing gout at 15-year follow-up is less than 50% (6). Various diseases, obesity, nutritional habits and various medications increase the risk of hyperuricemia and gout. Diseases associated with increased cell turnover (hematologic malignancies and psoriasis) lead to increased uric acid production, while metabolic syndrome and chronic kidney disease lead to decreased uric acid excretion, resulting in hyperuricemia

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periarticular tissues. Flares of gout develop due to the immune response against these crystals. NSAIDs, colchicine, and glucocorticoids are primarily used in the treatment of flares. In patients unresponsive to these treatments, anakinra and canakinumab are used. Lifestyle changes, dietary adjustments, and hypouricemic drugs such as allopurinol, febuxostat, and probenecid are used to lower serum uric acid levels. In patients diagnosed early and receiving appropriate treatment, the disease can be controlled, and long-term complications associated with the disease can be prevented. However, in patients without appropriate treatment, long-term joint damage occurs, and tophi develop. Therefore, early diagnosis and treatment are crucial.

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Chapter 7

PHYSIOLOGICAL CHANGES IN THE ELDERLY

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INTRODUCTION

As we age, our body's ability to respond to stress decreases, and there is an increased risk of age-related diseases due to a homeostatic imbalance. This natural process begins at birth and continues until death. Although there is no fixed age limit for old age, people over 65 are defined as elderly by the World Health Organization (WHO). Demographically, age is divided into 3 periods. Those aged 65-74 are referred to as "young old", those aged 75-84 as "middle-aged" and those aged 85 and over as "advanced old" (1). Aging is physiological and begins at a very early age. Marital age is not a reflection of a person's physiological age. A decline in organ function can occur at the age of 30 or even earlier. It can be accelerated by our lifestyle and chronic diseases (2). Mechanistically, the lifelong accumulation of a variety of molecular and cellular disorders leads to the aging of the organism. Several factors affect aging, such as oxidative stress, programmed cell death, mutation and accumulation of abnormal proteins, cell membrane abnormalities, mitochondrial differentiation, and telomere dysfunction.

The aging population poses a serious medical problem, as it places a burden on healthcare systems and society in general, and age-related diseases and disorders are widespread (3). The number of older people has increased rapidly in recent years, leading to profound demographic changes worldwide (4). Turkish Statistical Institute (TUIK) data shows that the number of Turkish citizens aged 65 or over was 6,895,385 in 2017. This number is expected to increase to 8,451,669 in 2022. However, based on community-based population studies, it is estimated that the proportion of the elderly population in our society will be 12.9% of the total population in 2030 (5). By 2050, it is estimated that a fifth of the world's population will be 60 or older (6).

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CONCLUSION

Nowadays, the elderly population is increasing in parallel with the increase in average life expectancy, and physical changes and medical problems increase with aging. The heterogeneous changes associated with advancing age lead to a decline in functional capacity. A better understanding of the biological processes of aging can lead to new preventive care perspectives that enable independent aging by preventing and/or delaying the onset of chronic diseases and disabilities.

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Chapter 8

FACTORS AFFECTING MORTALITY AND MORBIDITY IN NON-VARICOSE ACUTE UPPER GASTROINTESTINAL SYSTEM BLEEDING

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INTRODUCTION AND PURPOSE

Acute upper GI (Gastrointestinal) tract bleeding is an important source of mortality and morbidity. The annual hospitalization rate is approximately 102 per 100,000. It is twice as common in men than in women. Its incidence increases with age (1).

Despite advances in intensive care treatment, diagnostic and therapeutic procedures, and the availability of powerful antisecretory drugs, the mortality rate of acute upper GI bleeding varies between 2.3% and 14% (2).

Most of the deaths occur in elderly patients over 60 years of age and those with additional severe diseases such as serious heart disease, cancer, kidney failure. While mortality was found to be 40% in patients who were hospitalized for another reason, only 0.6% of patients under 60 years of age who did not have any other serious disease or malignancy (3).

Bleeding is the most common complication of ulcers. About one-third of peptic ulcers are symptom-free and can occur with direct bleeding.

Most bleeding attacks stop spontaneously. However, since the mortality rate of patients with ongoing or recurrent bleeding is high, invasive interventional techniques are needed.

Emergency esophagogastroduodenoscopy (EGD) is the first diagnostic option for upper GI bleeding that affects vital signs or requires blood transfusion. EGD not only allows the detection and treatment of a bleeding lesion, but also provides

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Chapter 9

THE DISTRIBUTION OF SEROPREVALENCE OF VIRAL HEPATITIS IN DIYARBAKIR PROVINCE

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INTRODUCTION

Viral hepatitis is a liver inflammation caused by viruses and is one of the most important public health problems worldwide. They can occur in an acute form with a relatively rapid onset or in a chronic form. Even though "epidemic jaundice" has existed since the beginning of civilization, the viral etiologies of hepatitis have just recently been discovered. The most common causes of viral hepatitis are hepatitis A, B, C, D, and E, and some unrelated hepatotropic (prone to settle in the liver) viruses (Table 1). Less commonly, other viruses such as cytomegalovirus, Epstein-Barr virus, herpes simplex virus, and yellow fever can also cause liver inflammation (1). It can cause significant morbidity and mortality by progressing to cirrhosis, liver failure, and liver cancer, and therefore closely concerns the country's economy.

VIRAL HEPATITIS

Table 1: Etiological, epidemiological, and some other features of hepatitis A, B, C, D and E

Type of Hepatitis	Family/Genome	Transmission	Mortality rate (%)	Prevalance of Turkiye*	Presence of vaccine
A	Picornaviridae/ RNA	Faecal-oral route	<0.5	+++	Available
B	Hepadnaviridae/ DNA	Perinatal, horizontal	1-2	+++	Available
C	Flaviridae/RNA	Bloodborne transmission	4	++	None

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necessary to diagnose HBV and HCV infection in the pregnant patient group. As healthcare personnel, we should not neglect vaccinations.

The World Health Organization has published 'Global health sector strategies on, respectively, HIV, viral hepatitis and sexually transmitted infections for the period 2022-2030'. The strategies target individuals most impacted and at risk by each disease and suggest national measures that are common and disease-specific. At the same time, the goal is to increase health insurance and primary healthcare services and contribute to achieving the goals of the 2030 Sustainable Development Agenda (15).

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Chapter 10

APPLICATIONS OF ARTIFICIAL INTELLIGENCE IN HEALTH SCIENCES

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INTRODUCTION

Artificial intelligence (AI) is a system of technologies that allows machines to perform complex processes such as learning, logical thinking, solving issues, and making decisions that normally require human intelligence (1). AI is being used to speed up, increase efficiency and reduce costs in various sectors, including banking, automotive, defense and healthcare institutions and organizations. AI has revolutionized the healthcare industry by changing the way of treat, diagnose, and keep track of patients (2). With the ability to provide more individualized treatments and more accurate diagnoses, this technology is significantly enhancing healthcare research and results. The application of AI in healthcare enables medical professionals to quickly identify disease markers and trends that they might otherwise miss by analyzing large volumes of clinical documentation. AI can be used in hospital and clinic settings to make healthcare systems faster, smarter, and more effective in treating millions of patients globally. With the increase in the amount of data and accessibility of data in the health sector, it has been possible to use of AI successfully in recent applications. Because AI technology may uncover clinical information buried behind large, complicated data sets, it can have a significant impact on physicians' judgment and decision-making processes. It is found that AI has a broad range of potential applications when studies utilizing it in the health sector are reviewed, from scanning radiological images for early diagnosis to treatment, monitoring, predicting results from electronic health records, classifying diseases and identifying high-risk conditions (3-12). Furthermore, successful applications have been made in fields like health management, robotic surgery, medical education, and the manufacture of drugs, devices, and vaccines (13-18). In addition to assisting medical professionals

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and other medical professionals are expected to increasingly adopt and use new technologies because they are responsible for providing healthcare. Consequently, a lot of benefits are expected to come from AI in the health domain; while in some areas it works better with doctors, in the long run it will benefit doctors more than it will replace them. Effective use of AI by doctors allows them to professionally manage their influence on patient care, the healthcare system, and society at large.

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