Obesity in Children and Adolescents
– a New Challenge in Orthodontic Practice

Otyłość u dzieci i młodzieży – nowe wyzwanie w praktyce ortodontycznej

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A – research concept and design; B – collection and/or assembly of data; C – data analysis and interpretation; D – writing the article; E – critical revision of the article; F – final approval of article

Abstract

Obesity is one of the most common chronic disorders affecting the health and well-being of children and adolescents and its prevalence is increasing steadily and dramatically all over the world. Because of the growing number of overweight or obese children and adolescents in orthodontic practice, the aim of the study was to discuss a variety of medical and psychosocial issues associated with childhood obesity which may have an impact on orthodontic therapy. Overweight and obesity are usually associated with the early onset of puberty and earlier pubertal growth spurt. Obesity has been hypothesized to impact craniofacial growth and lead to more precocious skeletal maturation of the maxilla and the mandible, which has a fundamental significance in dentofacial orthopaedics. An acceleration of dental development in obese children may alter the diagnosis and timing of orthodontic treatment. In fact, when incorporating orthodontic therapies such as growth modification or serial extractions, the timing of intervention may require recalculation to consider also the weight status of the patient. Based on available literature, obesity seems to impact bone metabolism through several mechanisms, which may considerably affect the orthodontic tooth movement. Obesity in childhood and adolescence is associated with significant psychosocial problems, which are probably more common than medical consequences. Most typical complications are related to psychosocial dysfunction and social isolation. Obesity in childhood and adolescence should be considered as a chronic medical condition associated with a multitude of medical and psychosocial consequences, which may influence orthodontic therapy (Dent. Med. Probl. 2015, 52, 2, 131–136).

Key words: obesity, children, overweight, orthodontic treatment, adolescents.

Słowa kluczowe: dzieci, młodzież, otyłość, nadwaga, leczenie ortodontyczne.
bled from 7.5% to 15.2% in boys and from 6.5% to 11.8% in girls. The data obtained in 2009 pointed to the further increase in the percentage of overweight and obese children reaching 35% in boys and almost 20% in girls [6, 7]. According to WHO, the definition of overweight and obesity is an abnormal and excessive fat accumulation which has a negative effect on our health. Body mass index (BMI) is an index of weight-for-height that is routinely used to define overweight and obesity in adults. It accounts for a person’s weight in kilograms divided by the square of his height in meters (kg/m²). A BMI greater or equal to 25 points to overweight and a BMI greater and equal than 30 points to obesity [1]. BMI values assessed in children vary depending on sex and age of the examined individuals but the correlation is not the same as in adults. Thus it is recommended to use International Obesity Task Force cut-off points applied for children and adolescents that correspond with their age and sex. In accordance with them, obesity is defined as greater than or equal to the 95th percentile of body mass index (BMI) and overweight is considered greater than the 85th percentile of BMI [8–10].

As the prevalence and severity of childhood obesity increase, concern about adverse health outcomes in childhood and adolescence is rising. Obese children are likely to develop a wide range of health problems as adults, like cardiovascular disease, insulin resistance, musculoskeletal disorders (especially osteoarthritis), some cancers (endometrial, breast, colon). Childhood obesity is strongly related to a higher risk of obesity, premature death and disability in adulthood. But apart from increased future risks, obese children experience breathing difficulties, higher risk of fractures, hypertension, early markers of cardiovascular disease, insulin resistance and psychological effects [1, 3, 8].

Because of the growing number of overweight or obese children and adolescents in orthodontic practice, the aim of the study was to discuss a variety of medical and psychosocial issues associated with childhood obesity which may have an impact on orthodontic therapy.

**Timing of Puberty**

Nutrition is an important regulator of the tempo of human growth. Overweight and obesity are usually associated with the early onset of puberty and earlier pubertal growth spurt [11–14]. Nevertheless, there still remain many unanswered questions concerning the relationship between childhood obesity and childhood growth and matura-

tion. The idea that weight can affect the timing of puberty was initially introduced by Frisch and Revelle [15] in 1970 as the critical mass theory. In accordance with this, it was thought that the critical weight of 22% body fat had to be reached to let puberty progress. Although there was some controversy about that hypothesis, the previously presented evidence lends some credibility to the critical mass theory. Leptin was discovered in 1994 by Zhang et al. [16] and is a candidate for a hormone that may have a regulatory function for body fat levels. Leptin is secreted by adipocytes and regulates appetite and metabolism through hypothalamic mediators. Research in rodents led to the theory that leptin might be involved in the timing of puberty, thus providing the missing link in the critical mass theory. Leptin was reported to slowly rise before puberty and may play a permissive role for the onset of puberty [17]. In addition to leptin, it has become increasingly evident that adipose tissue is a source of a variety of other secreted signals, such as adiponectin, resistin and several adipocytokines and is an active site for the conversion of cortisone to the more active cortisol and of androgens to estrogen, which contribute to the earlier activation of the hypothalamic-pituitary-gonadal axis and thereby to the earlier onset of puberty in the obese [11].

**Skeletal Growth**

The relationship between obesity and precocious skeletal maturation is a controversial subject in the literature. It has been suggested that leptin produced by the adipose tissue can stimulate skeletal growth by the activation of different mediators, such as insulin-like growth factor 1 and sex hormones. Alternatively, leptin might show a direct action on the skeletal growth centers. Leptin receptors have been found in the cartilaginous growth centers that are involved in skeletal maturation. Therefore, an obese subject probably has a mechanism of central resistance to leptin and an increased sensitivity to leptin at a peripheral level, leading to increased differentiation and proliferation of chondrocytes and resulting in precocious skeletal maturation [11, 12–14, 17].

In addition, it has been reported that early onset obesity can cause increases in vertebral bone density and bone size and an acceleration of skeletal growth. In particular, obesity has been hypothesized to impact craniofacial growth and leads to more precocious skeletal maturation of the maxilla and the mandible, which has a fundamental significance in dentofacial orthopaedics [18].

There is an evidence suggesting that obesity
may influence the timing of puberty and growth patterns. Higher BMI in childhood may result in an earlier pubertal growth spurt and subsequent earlier cessation of growth. Obese girls and boys present an earlier onset of puberty and completion of puberty with a shorter duration of puberty compared to the normal-weight peers. They tend to be taller during pre-puberty but lose this growth advantage during puberty and have a similar adult height compared to the normal-weight children [19].

In the study of Giuca et al. [20] obese individuals presented a greater mean discrepancy between skeletal age and chronologic age according to the carpal analysis and showed a significantly higher mean cervical vertebrae maturation score in comparison with the normal weight individuals. Therefore, in the obese individuals with skeletal discrepancies, it may be necessary to perform an examination and dentofacial treatment earlier than in the normal-weight patients [20].

It is obvious that growth and development can be influenced by an obese body mass, but to what level this occurs is currently unknown. The bones and soft tissues seem to grow differently in obese patients, and the differences between obese and normal-weight subjects have just begun to be examined. In growing patients, dental development and skeletal maturation are widely used to determine the timing of orthodontic treatment and the selection of treatment modalities [21–23]. Growth and development issues should be examined carefully in obese and overweight patients. Obese patients tend to go through puberty earlier than non-obese individuals. Treatment planning for obese patients should assume that they have less time until their pubertal growth spurt than normal weight subjects, but not to such a significant level that craniofacial growth modification should not be attempted [18].

**Dentition Development**

Orthodontists rely on dental age rather than on chronologic age as an indicator of when to initiate the treatment. Accelerated dentition development may affect orthodontic treatment timing and the selection of treatment options [21, 22]. Few authors studied the dentition development in overweight or obese children and adolescents. Hilgers et al. [24] conducted a study to determine if increased body mass index (BMI) is associated with accelerated dental development in children. The study revealed that dental development was significantly accelerated with an increased BMI. The mean dental age acceleration for overweight and obese subjects was 1.31 ± 1.22 years and 1.53 ± 1.28 years, respectively. Those findings were in agreement with Eid et al. [25], who also found a significant correlation between dental maturity and BMI (0.68 years for males and 0.62 for females).

Mack et al. [26] found that for every 1 percentile of increase in BMI percentile for age, there was a 0.005-year increase in dental age. The study conducted by Mack et al. [26] revealed a significant relationship between weight status defined by BMI percentile and dental age and cervical vertebral maturation stage. An increase in BMI percentile was thought to be associated with an increase in both dental and skeletal maturation [26].

An acceleration of dental development in obese children may alter the diagnosis and orthodontic treatment timing. In fact, when incorporating orthodontic therapies like serial extractions or growth modification, the timing of intervention may require recalculation to consider not only sex or race, but also the weight status of the patient.

**Craniofacial Morphology**

Craniofacial growth is highly dependent on interactions between genes, hormones, nutrients and epigenetic factors. Disturbances of any of those mechanisms may lead to an aberrant growth pattern resulting in a deviant craniofacial morphology.

In the study of Öhrn et al. [27] a majority of angular and linear measurements were increased in obese adolescents in comparison to the healthy controls and the differences between obese and non-obese individuals were more significant in females than males. The most significant difference was found in the length of the mandible (Cd-Pgn), which was 6 mm and 8.7 mm greater in obese females and males respectively compared to the non-obese individuals. The obese adolescents showed increased maxillary and mandibular prognathism, as well as anterior cranial base length. The findings of the study suggest that obese adolescents show more intense craniofacial growth activity than normal weight individuals.

Sadeghianrizi et al. [28] studied the development of craniofacial complex in obese adolescents and found that obesity was related to bimaxillary prognathism and relatively greater craniofacial dimensions such as maxillary length (Pm-A), mandibular length (Cd-Pgn), posterior facial height (S-Go) and lower anterior facial height (ANS-Gn) despite decreased level of growth hormone. The general impression of the study was that obese ad-
adolescents presented a more advanced craniofacial development in comparison to the normal weight individuals of a corresponding age. Although the craniofacial dimensions were greater in the obese subjects, their facial proportions did not present any significant deviation from the norm.

Cephalometric and facial analyses should be altered in the examination of obese or overweight patients. Normal values for this group should be viewed with the knowledge that they were originally established on normal weight individuals and therefore might not be appropriate for obese and overweight subjects. Increased tissue thickness can actually allow for greater leeway when correcting dental and skeletal discrepancies. An area for future research should aim to examine the soft-tissue changes observed in obese patients during orthodontic therapy. Although clinical changes are expected in the soft-tissue profile as a result of bone and tooth movements, it seems reasonable to assume that the same movements in obese patients will result in less dramatic changes in the profile because of the increased thickness of the soft-tissues [29].

**Bone Metabolism**

In accordance with available literature, obesity seems to impact bone metabolism through several mechanisms, which may considerably affect the orthodontic tooth movement. Obesity is thought to decrease bone formation when increasing adipogenesis, as adipocytes and osteoblasts derive from a common multi-potential mesenchymal stem cell. For instance, mechanical loading promotes osteoblast differentiation and inhibits adipogenesis by down-regulating peroxisome proliferator-activated receptor gamma (PPARγ) or through stimulation of a durable beta-catenin signal. Activation of PPARγ decreases differentiation of osteoblasts, bone mineral density and trabecular bone mass simultaneously with the increase of differentiation of adipocytes and bone marrow adipose tissue volume.

Obesity may also stimulate bone resorption through upregulating proinflammatory cytokines such as IL-6 and TNF-α. These proinflammatory cytokines are able to enhance osteoclast activity through the regulation of the RANKL/RANK/OPG pathway.

Obesity is considered to influence bone metabolism directly or indirectly through adipocyte-derived cytokines such as leptin and adiponectin. Obesity results in a significant increase in serum leptin and decrease in adiponectin. Overproduction of leptin may have a negative impact on bone metabolism. Adiponectin is another cytokine secreted by adipocytes and shows anti-inflammatory effect. In animal model, adiponectin has been showed to inhibit osteoclastogenesis, decrease bone resorption and increase bone mass. Obese individuals present low serum adiponectin concentration in comparison with the normal weight subjects.

Finally, a high-fat diet that leads to obesity has been reported to impair intestinal calcium absorption. Free fatty acids are able to form unabsorbable insoluble calcium soaps resulting in decreased calcium absorption [30–33].

**Psychosocial Problems**

Obesity in childhood and adolescence is associated with significant psychosocial problems, which are probably more common than medical consequences. Most typical complications are related to psychosocial dysfunction and social isolation. Childhood obesity has a significant impact on the emotional development of a child or adolescent, who suffers discrimination and stigmatization. Individuals who were obese in childhood are more likely to have poor body image and low self-esteem and confidence. Overweight children and adolescents commonly report lower health-related quality of life in physical, emotional and social aspects. Adolescent girls have a tendency to experience body dissatisfaction, a drive for thinness, and a tendency to succumb to eating disorders. There is evidence that obese children and adolescents develop lower self-esteem than their nonobese peers. Importantly, girls are at a greater risk of self-esteem problems and can develop lower self-esteem during puberty. Adolescents and children might discount their condition as a coping mechanism. They may consider their appearance as unimportant, which can be problematic in orthodontic treatment. They could also use distortion as a defense mechanism and underestimate the severity of their body image or weight. Depression and anxiety are more common in obese patients.

An overweight patient might be unwilling or unable to regard the orthodontic problem as important, because of the defense mechanisms and disregard for body appearance. The attitude could also swing into hyperrealization of appearance with the patient developing unrealistic expectations. An orthodontist should have a high index of suspicion for psychosocial problems associated with childhood obesity to recognize them and deal with them at the right time before they become problematic for the orthodontic therapy [34–37].
Conclusions

Obesity in childhood and adolescence should be considered as a chronic medical condition associated with a multitude of medical and psychosocial consequences, which may also have a significant impact on orthodontic therapy. Due to the increasing number of overweight or obese children and adolescents in orthodontic practice, orthodontists should be aware of potential health issues associated with childhood obesity and as health care providers should promote health and actively refer or counsel overweight patients.

References


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