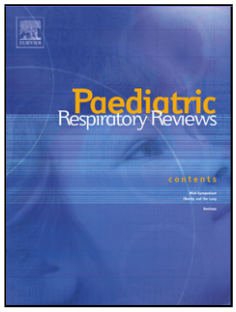

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Growth and development after oesophageal atresia surgery: Need for long-term multidisciplinary follow-up

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Abstract :

Survival rates in oesophageal atresia patients have reached over 90%. In long-term follow-up studies the focus has shifted from purely surgical or gastrointestinal evaluation to a multidisciplinary approach. We reviewed the literature on the long-term morbidity of these patients and discuss mainly issues of physical growth and neurodevelopment. We conclude that growth problems – both stunting and wasting – are frequently seen, but that sufficient longitudinal data are lacking. Therefore, it is unclear whether catch-up growth into adolescence and adulthood occurs. Data on determinants of growth retardation are also lacking in current literature. Studies on neurodevelopment beyond preschool age are scarce but oesophageal atresia patients seem at risk for academic problems and motor function delay. Many factors contribute to the susceptibility to growth and development problems and we propose a multidisciplinary follow-up schedule into adulthood future care which may help improve quality of life.

Keywords: oesophageal atresia, outcome, growth, motor function, neurodevelopment, cognition

Eductional aims:

After reading this review readers will be able to

- recognize risk factors for long-term morbidity in oesophageal atresia patients
- describe long-term problems with respect to growth and neurodevelopment in oesophageal atresia patients
- mention topics that should be addressed at various stages of life in long-term multidisciplinary follow-up of oesophageal atresia patients

Introduction

Oesophageal atresia (OA) is a rare congenital anatomical anomaly with a prevalence of 1 in 2,500 to 4,500 live births(1). Gross in 1953 already described the classification of the different types; the most common is type C with a distal tracheo-oesophageal fistula (TOF), which occurs in 85-90%(2). In the 1950s and 1960s the neonatal mortality rates were approximately 35%; severe bronchopneumonia was the main cause of death(3). Due to better surgical techniques and intensive care treatment, mortality has dropped to less than 10% today (4, 5), with severe associated chromosomal defects and complex cardiac defects as main causes of death. With decreasing mortality rates attention has shifted towards long-term morbidity.

The most common problems in OA-patients are: gastrointestinal morbidities (e.g. feeding difficulties, gastro-oesophageal reflux disease (GORD), dysphagia), respiratory problems (e.g. lower respiratory tract infections, restrictive lung function, impaired exercise tolerance), impaired physical growth, and neurodevelopmental delays. This review will focus on growth and neurodevelopment, and the importance of longitudinal multidisciplinary long-term follow up.

Physical growth

Long-term growth data

Growth has mainly been described in cross-sectional studies(6-11) and retrospective evaluations of medical charts(12-14). Gischler and co-workers evaluated longitudinal growth up till five years in a prospectively followed cohort of 23 OA-patients born from 1999 onwards; all but one patient had a type C-OA(15). Although 26% of the patients needed prophylactic antibiotics to prevent airway infections, both height and weight corrected for age increased between two and five years, but weight for age was still below the population norm at five years. The question is whether this trend of catch-up growth continues when the children get older. Meanwhile it has become known at 12 years of age growth had normalized in 22 of those 23 patients: mean (SD) standard deviation score (SDS) height was -0.10 (1.12), and mean (SD) SDS weight for height was -0.22 (1.04) (unpublished data).

Catch-up of height for age was also suggested by Andrassy and co-workers, who in 1983 published data on nutritional assessment in 53 cross-sectionally studied OA-patients – 83% with type C – aged 0.9 to 31 years. Chronic malnutrition – defined as height for age at least 2 SD below the norm – was significantly less prevalent in children 13 years and older than in those younger than 13 years (7.7 versus 22.5% of children, respectively)(6). This age relation was confirmed 10 years later – in 1993 - by Chetcuti and co-workers, who performed a cross-sectional study in 302 OA-patients aged 1 to 37 years (87% with type C; 164 of them were > 15 years). Adult OA-patients in that study had normal height and weight. Wasting, i.e. decreased weight for height, was reported in 32% of patients < 5 years, 19% of patients aged 5-10 years, and 13% of children aged 10-15 years(8).

In 2003, Little and co-workers published results of a chart review including 69 OA-patients – 77% with type C. At 5 years, height and weight were below the 5th percentile in 22 and 25%, respectively. Thirty-nine children were seen at the age of > 10 years; height was below the 5th percentile for 5 of those (12%) and weight was below the 5th percentile for 7 of those (17%)(12). Lacher and co-workers performed a retrospective chart review of 80 OA-patients (79% with type C); only 46 of them were evaluated at 10 years. Weight for age was below the 3rd percentile for 20, 28 and 17% at the age of one year, six years, and 10 years respectively (13). These data suggest that growth problems persist even beyond the age of 10 years.

In contrast, in two cross-sectional studies from different centres in Finland normal physical growth was reported in children with a mean age of 12 years studied several decades ago(7, 9), and Legrand and co-workers recently even found overweight/obesity in 9% of 57 type C OA-patients studied at a mean age of 13 years(10).

The most important results per age category are summarized in Table 1. Since most studies have a cross-sectional or retrospective study design, conclusions on longitudinal growth cannot be drawn.

Prospective, longitudinal data collection is needed to describe growth profiles in OA-patients. Still, on the basis of the currently available literature it can be concluded that OA-patients are at risk for physical growth problems, especially within the first years of life, but that problems may persist even at older age. None of

the published long-term studies provides data on deviation from target height SDS. The target height is the expected adult height given the heights of the biological parents and corrected for secular trend(16).

Factors that influence long-term growth in OA

Children with OA have many problems that may affect long-term growth: recurrent surgical interventions, feeding difficulties, gastrointestinal problems, respiratory infections, associated congenital malformations, and genetic syndromes, among other things (Figure 1). Moreover, many are prematurely born or are small for gestational age (SGA)(4, 17). The current literature is not conclusive regarding the specific contributions of these factors to growth. Many studies do not provide data on associated anomalies and proportions of prematurity and SGA. The presence of a long-gap OA is a risk factor for feeding problems and growth impairment(13, 18). In the study of Lacher and co-workers, weight of 6 of 14 long-gap OA-patients (43%) was below the 3rd percentile at 10 years of age(13). A recent paper described persistent growth failure in 16 long-gap OA patients up till the age of 7 years(19). Puntis and co-workers administered a standardized questionnaire regarding feeding difficulties to 230 members of the British parent support group. The response rate was 54%, data were obtained on patients with a median age of 4 to 5 years (depending on type of OA). In 74% a primary anastomosis was performed and in 26% a delayed oesophageal substitution procedure. The median questionnaire scores did not differ between those two groups and the scores did not correlate with SDS for weight and height(18). In the study of Legrand and co-workers in type C OA-patients only a past history of GORD was a significant predictor of lower weight for SDS height(10); factors such as prematurity, SGA, actual GORD, fundoplication, or current respiratory problems did not affect long-term growth.

Neurodevelopmental outcome

The scarce data on neurodevelopmental outcome in OA-patients are summarized for different age categories in Table 1. Worldwide, three centres have reported on prospective evaluation of neurodevelopment within the first years of life(20-23). They used different versions of the Bayley Scales of

Infant Development and different reference groups. In all three studies the mental and psychomotor domain scores for non-syndromal OA-patients generally were within the normal ranges(20-22). In the study by Walker and co-workers, however, the mean score on language expression at one year was significantly below the norm (21). In that study, all other mean domain scores of OA-patients were slightly lower than the control scores although not significantly different(21). In the other two studies, too, the mean scores of the OA-patients never reached the population norm score of 100(20, 22). The meaning of this observation is not clear. Mazer and co-workers studied whether any factors within the first two years of life predicted cognition at five years of age in a cohort of 105 children with major non-cardiac congenital anomalies, including 15 OA-patients. Significant predictors were the number of congenital anomalies and the mental developmental scores obtained within the first two years of life(24). Mazer and co-workers also concluded that psychomotor developmental scores in the first years of life were the only significant predictors of motor function at five years of age(24).

Thus far, only one published study addressed motor function in OA-patients beyond toddler age. Van der Cammen and co-workers studied motor function performance in a prospective cohort of 29 OA-patients without severe retardation or neurological impairments at the age of five years(23). Seven of these children (24%) received physical therapy at home. Motor function was normal in 19/29 children (66%), which is significantly less than the 85% expected from the Dutch reference data. Most problems were encountered with gross motor function (ball skills and balance skills) whereas manual dexterity was normal in 93% (versus 85% expected) of them(23).

Only three cross-sectional studies on cognition in OA-patients at school age are available(7, 25, 26). In 1984, Lindahl reported normal intelligence at an age of 8.8 to 16.5 years (mean age 12.7 years) in a cohort of 33 OA-patients born between 1966 and 1973. Compared with healthy Finnish children these patients had normal body-image but more anxiety problems(7).

In 1999, Bouman and co-workers reported a mean intelligence quotient (IQ) of 90.2 in 36 OA-patients aged 8 to 12 years (mean age 10.2 years) ; this was significantly lower than that of the reference population. Thirty

percent of the study group had an IQ < 85. Their overall feelings of self-worth were normal. Neither their parents nor their teachers reported more behavioural problems compared to healthy peers(25).

Even less favourable data on long-term cognitive development have more recently been published by Kubota and co-workers: Mental retardation with IQ < 70 was observed in 25% of 20 OA-patients aged 6 to 17 years. Furthermore, parents-reported behavioural problems within the clinical range for 35% of OA-patients. Unfortunately, the authors do not explain how these patients were recruited and therefore the possibility of selection bias cannot be excluded(26).

Only three studies report on school performance. In the study of Bouman and co-workers, 22% of 36 OA-patients received special education(25); this percentage was even higher in a study by Faugli and co-workers, i.e. 7 of 21 adolescents with OA (33%) (11). Lastly, Lacher and co-workers reported that 6 of 60 OA-patients (10%) who had reached school age received special education due to mental retardation (13).

Implications and future perspectives

As survival rates for OA have increased, more of these children will grow up into adulthood. Parents and care providers will therefore need to pay attention to signs of long-term morbidity. A multidisciplinary approach with smooth transition of care from the paediatric to the adult care setting is essential in this context, as most of the problems of OA-patients are multifactorial and interact with each other.

With respect to growth the outcomes have hardly changed over the past 30 years. Still, we report growth failure especially in the first years of life without knowing its most important determinants. Findings from cross-sectional studies suggest that catch-up growth occurs in adolescence and adulthood, but confirmatory longitudinal data are lacking. The current literature suggests that both height and weight are below normal. So, the question arises why this should be so. In the studies that have been published thus far, data on target height and growth deviation of target height SDS are not available. Are these children undernourished? Standardized studies on energy expenditure have – as far as we know – not yet been performed in OA-

patients. For future studies it may be worthwhile to include data on parental heights, making use of population-specific reference data.

The relationship between chronic malnutrition and cognition has been studied extensively during the past years and Corbett and Drewett performed a meta-analysis of these studies(27). They concluded that evidence from reasonably well-controlled studies indicates that failure to thrive in infancy is associated with adverse intellectual outcomes sufficiently large to be of importance at a population level(27). Some authors have suggested that the critical period of weight gain is within the first 8 weeks of life, and that weight faltering during that period predicts IQ at the age of 8 years(28). Although there is little evidence to recommend this for OA-patients, we still propose to start early and standardized nutritional assessment with dietary management within the first weeks of life, i.e. during the initial hospitalization. The next question that arises is whether nutritional interventions at older age are beneficial in OA-patients – taking into account the window of opportunity in early infancy and the fact that the available studies suggest catch-up growth in adolescence. Considering the differential timing of growth of body systems in humans, nutritional interventions at older age seem to be recommendable(29). We have to realize that most data on stunting in infancy and childhood and the critical windows for its treatment with nutritional intervention have been obtained in underdeveloped countries(29). Nevertheless, the pubertal growth phase is considered as an additional window of opportunity for nutritional intervention(29). Dysphagia complaints have been reported in 44-61% of OA-patients during puberty(10, 30). Offering nutritional intervention to OA-patients in that phase of life may be challenging because puberty nowadays is characterized by increasing autonomy and importance of peer groups, and leaving well-controlled primary educational settings. Long-term follow up of growth and feeding difficulties in OA-patients is important; and timely interventions should be offered also if problems arise at later age. Actively asking about dysphagia, GORD, diet, and feeding habits may be helpful in this respect. OA-patients may have altered perception of GORD, for example, because they have grown up with these symptoms(31).

It seems that despite normal mental and psychomotor development in the first years of life(20-22), the need for special education among OA-patients is higher than in the normal population(11, 13, 25). The currently

available literature on mental health and development in OA-patients does not allow for explanations of this observation. Some issues that could be worthwhile to address in future care for OA-patients are speculated on here: Although OA-patients usually do not suffer from severe malnutrition, we should be aware that moderate malnutrition in infancy may lead to lower IQ, impaired academic achievement, or (subtle) neuropsychological problems even into adulthood(32, 33). Disturbed mother-child interaction, which has been described in OA-patients(34), may lead to insecure attachment with early relational trauma(35). This – in its turn – could lead to maldevelopment of the right hemisphere(36). Studies in critically ill neonates have shown that eight-year-old survivors concentration and school performance than peers, despite normal intelligence(37, 38). When children get older tasks get more complex and demanding. For those with more subtle developmental delays "growing into their deficits" is a realistic phenomenon(39) and may explain why – also in OA patients – developmental evaluations within the first years of life show scores within normal ranges. Ongoing prospective longitudinal follow-up programs should be continued with a focus on neurodevelopmental evaluations at school age and beyond. These evaluations should not only assess cognition and academic achievement, but also executive functions, attention, language, sensorimotor functions, visuospatial processes, memory, and behaviour(40). Standardization and use of appropriate reference data or control groups is mandatory to draw conclusions and provide directions for improvement of care and intervention studies (Figure 1). As OA is a rare disease and case series are small, collaborative studies should be encouraged, also to evaluate the long-term effects of new surgical techniques on growth and development(41, 42).

In conclusion, this review suggests that children born with OA are at risk for growth failure, neurodevelopmental delay, and school problems. There is every reason therefore to include them in long-term multidisciplinary follow-up programs addressing a wide range of topics, many of which may interact. Outcome research should focus on collaborative multicentre projects with uniformity in assessment protocols and use of standardized instruments and data management.

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Table 1: Overview of long-term growth and (neuro)development outcomes

	Infancy (< 2 yrs)	Preschool age (2-5 yrs)	School age (6-12 yrs)	Adolescence (>12 yrs)
Growth	impaired weight, impaired height(13-15, 22, 42)	impaired weight, impaired height(8, 15, 18)	weight improves > 10 yrs(8, 12, 13), height improves > 10 yrs(12), normal growth(7, 9)	weight impaired(8, 11), height impaired(11), normal growth(6, 7, 10)
(Neuro)developmental outcome				
Motor function	normal(20-22)	abnormal in 34% at 5 years(23)	unknown	unknown
Cognition	normal(20, 22), language expression scores low(21)	unknown	normal(7), mild to moderate delay(25, 26)	unknown
Neuropsychological tests	not applicable	unknown	unknown	unknown
School performance	not applicable	not applicable	special education 10-22%(13, 25)	special education 33%(11)
Behaviour	unknown	unknown	anxiety problems, behaviour in clinical range 23%(26)	normal, anxiety problems, behaviour in clinical range 23%(26)

Table 2: Topics to be addressed at the various ages

	Specific topics	Relevance/intervention
Infancy	Growth	Hyperalimentation
	Feeding difficulties/oral aversion	Referral preverbal speech-language pathologist
	Psychosocial wellbeing	Psychological support
	Gastro-oesophageal reflux	Drug therapy or anti-reflux surgery
	Dysphagia	Dilatation in case of stenosis
	Airway infections	Antibiotic prophylactic therapy if indicated
	Neurological impairment	Early recognition, rehabilitation, genetic counseling
	Mental development	Early recognition, rehabilitation, genetic counseling
	Motor development	Referral physical therapist
	Associated anomalies	Organ-specific intervention if indicated
Toddler/preschool age	Growth	Hyperalimentation
	Feeding difficulties/oral aversion	Referral preverbal speech-language pathologist
	Psychosocial wellbeing	Psychological support
	Gastro-oesophageal reflux	Drug therapy or anti-reflux surgery
	Dysphagia	Early evaluation, management based on cause
	Airway infections	Antibiotic prophylactic therapy if indicated
	Neurological impairment	Rehabilitation, genetic counseling
	Language development	Referral speech-language pathologist
	Mental development	Early recognition, rehabilitation, genetic counseling
	Motor function development	Referral physical therapist
	Associated anomalies	Organ-specific intervention if indicated
School age	Growth	Hyperalimentation, dietary advice
	Feeding difficulties/dysphagia	Management based on cause
	Gastro-oesophageal reflux	Drug therapy or anti-reflux surgery
	Motor function development	Referral physical therapist, sports participation
	Neuropsychological assessment	Early school support
	Self esteem	Early intervention, support
	Airway infections	Antibiotic prophylactic therapy if indicated
	Lung function assessment	Evaluate reversibility of airflow obstruction
	Exercise capacity	Exercise training, sports participation
	Associated anomalies	Organ-specific intervention if indicated
Adolescence into adulthood	Growth	Hyperalimentation, dietary advice
	Feeding difficulties/dysphagia	Management based on cause
	Gastro-oesophageal reflux	Drug therapy or anti-reflux surgery
	Neuropsychological assessment	School support, choice of profession/career
	Self esteem	Psychological support
	Exercise capacity	Exercise training, sports participation
	Associated anomalies	Organ-specific intervention and transition of care if indicated
	Transition to adult care	Gastroenterology (endoscopic surveillance); clinical genetics (counseling)

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Figure 1: Standardized multidisciplinary approach to optimize care for oesophageal atresia patients
GORD = gastro-oesophageal reflux disease

Figure 1.