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

Hanneke IJsselstijn *, Saskia J. Gischler, Leontien Toussaint, Marjolein Spoel, Monique H.M. van der Cammen-van Zijp, Dick Tibboel

Department of Intensive Care and Paediatric Surgery, Erasmus MC – Sophia Children's Hospital, Rotterdam, The Netherlands

EDUCATIONAL 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

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SUMMARY

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.
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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].

* Corresponding author. Erasmus MC – Sophia Children's Hospital, Wytemaweg 80, 3015 GD Rotterdam, The Netherlands. Tel.: +31 10 7036203.
E-mail address: h.ijsselstijn@erasmusmc.nl (H. IJsselstijn).

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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)

The numbers in brackets refer to the references of the studies.

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

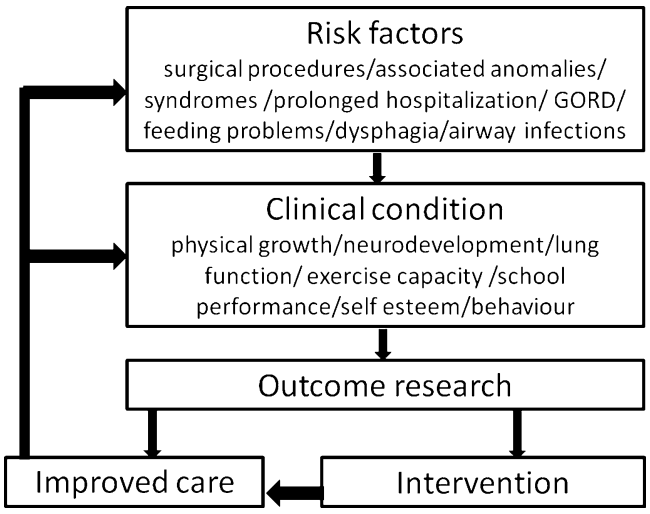

Figure 1. Standardized multidisciplinary approach to optimize care for oesophageal atresia patients. GORD = gastro-oesophageal reflux disease.

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