研究成果の刊行に関する一覧表

書籍

著者氏名	論文タイトル名	書籍全体の編集者名	書　籍　名	出版社名	出版地	出版年	ページ
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Ⅳ. 研究成果の刊行物・別刷

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ORIGINAL ARTICLE

The incidence and outcome of allied disorders of Hirschsprung's disease in Japan: Results from a nationwide survey

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allied disorders;
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pseudo-obstruction

Summary *Background:* Allied disorders of Hirschsprung's disease (ADHD) have been proposed to be the concept of the functional obstruction of the intestine with the presence of ganglion cells in the terminal rectum. They are classified into two categories based on pathology: (1) abnormal ganglia, including immaturity of ganglia, hypoganglionosis (HG), and intestinal neuronal dysplasia; (2) normal ganglia, including megacystis microcolon intestinal hypoperistalsis syndrome (MMIHS), segmental dilatation (SD), internal anal sphincter achalasia (IASA), and chronic idiopathic intestinal pseudo-obstruction (CIIP). Some of these show poor prognosis, therefore, the establishment of criteria and appropriate treatment strategies is required.
Methods: The questionnaires were sent to the 161 major institutes of pediatric surgery or gastroenterology in Japan, in order to collect the cases of ADHD during 10 years from 2001 and 2010.
Results: In total, 355 cases were collected. They included 28 immaturity of ganglia, 130 HG (121 congenital, 9 acquired), 18 intestinal neuronal dysplasia, 33 MMIHS, 43 SD, three IASA, and 100 CIIP. Of the 95 institutes, 69 (72.6%) had their own criteria for ADHD. Criteria were based on clinical symptoms and signs, and conventional pathological examinations. Prognosis was poor in congenital HG, MMIHS, and CIIP, while the others showed good survival rates.
Conclusion: Almost all Japanese cases of ADHD in the past 10 years were collected. Congenital HG and CIIP showed relatively high incidence, whereas acquired HG and IASA were extremely

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rare in Japan. The criteria of each disorder were also collected and summarized. Prognosis was poor in congenital HG, MMIHS, and CIIP.

1. Introduction

Allied disorders of Hirschsprung's disease (ADHD) have been understood as the conditions that clinically resemble Hirschsprung's disease (HD), despite the presence of ganglion cells in the terminal rectum.[1] Patients with Hirschsprung's disease generally present in the newborn period with delayed passage of meconium and abdominal distention or as a young child with severe chronic constipation. Patients with ADHD show similar symptoms and signs to HD, but they can be distinguished from HD by the pathological findings. The term *pseudo HD* was proposed by Ravitch in 1958.[2] They encountered patients referred for treatment of megacolon in whom the difficulty lay elsewhere rather than in the congenital absence of ganglion cells of the myenteric plexuses of a segment of the rectum or of the colon and rectum. Bentley et al[3] summarized *HD and allied disorders* in the *Seminar on Pseudo-Hirschsprung's Disease and Related Disorders*. The main thing to remember was that the various disease patterns were essentially determined by their underlying pathology, irrespective of what we choose to call them. ADHD was classified into two categories based on histology[3]: those with abnormality of ganglion cells and those without abnormality of ganglion cells (Table 1). Puri and Gosemann[4] called this group *variants of HD*, including four disorders: intestinal neuronal dysplasia (IND); isolated hypoganglionosis (HG); internal anal sphincter achalasia (IASA); and megacystis microcolon intestinal hypoperistalsis syndrome (MMIHS) in 2012.[4] They did not treat chronic idiopathic intestinal pseudo-obstruction (CIIP) as one of the variants of HD.

Okamoto and Toyosaka[5] used the term of *pseudo-Hirschsprung's disease* in the Japanese literature. It was defined as a congenital, nonmechanical obstruction of the intestine with presence of intramural ganglion cells in the terminal rectum. They classified them based on histology into two categories for six disorders: immaturity of ganglia (IG); HG; hypogenesis; IND; CIIP; and MMIHS.[5]

According to the literature and Okamoto and Toyosaka's[5] classification, ADHD was classified into two categories depending on the pathological findings (Table 2): (1) abnormal ganglia, including IG, HG, and IND; (2) normal ganglia, including MMIHS, segmental dilatation (SD), IASA, and CIIP. Some of them show poor prognosis; therefore, establishment of criteria, severity, and treatment strategy are required. In order to examine the incidence and criteria of ADHD, a preliminary nationwide survey was planned in Japan.

2. Patients and methods

As a nationwide retrospective cohort study, supported by Ministry of Health and Welfare, Japan, the preliminary questionnaires, requesting the number of cases of ADHD from January 2000 to December 2009 and the criteria of each institute, were sent to the 161 major institutes of pediatric surgery or pediatric gastroenterology representing the core members of the Japanese Society of Pediatric Surgeons, the Japanese Society of Pediatric Nutrition, Gastroenterology, and Hepatology, and the Japanese Study Group of Pediatric Constipation. Therefore almost all institutes that are treating ADHD are considered covered. The number of patients, including the definite and suspected cases, based on the classification of ADHD in Japan (Table 1) and the survival rate and clinical outcome were asked. The criteria of each institute were asked to be answered as free descriptions. The criteria for *definitive* or *suspected* were dependent on each institute.

This study was performed according to the Ethical Guidelines for Clinical Research published by the Ministry of Health, Labor, and Welfare of Japan on July 30, 2003. And this study was approved by the Ethical Committee for Clinical Research of Kyushu University Hospital, Fukuoka, Japan (No. 24-163).

3. Results

Replies were obtained from 157 of 161 institutes (98%). Out of 157 institutes, 95 (61%) had ADHD. In totally, 355 cases,

Table 1 Hirschsprung's disease and allied disorders (Ehrenpreis 1966).[3]

With abnormalities of ganglion cells
Hirschsprung's disease
Congenital megacolon
Congenital aganglionosis
Chagas' disease
Acquired megacolon
Aperistalsis
Hypoganglionossis
Immaturity of ganglion cells
Without abnormalities of ganglion cells
Aetiology obscure
Idiopathic megacolon
Functional megacolon
Psychogenic megacolon
Megarectum
Chronic constipation
Pseudo-hirschsprung
Segmental dilatation of the colon
Achalasia of distal rectal segment
Clear etiology
Symptomatic megacolon
Secondary megacolon (anal stricture, myxoedema, cerebral atrophy)

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Table 2 Classification for allied disorders of Hirschsprung's disease in Japanese survey.

(1) Abnormal ganglia (abnormal histology in hematoxylin–eosin or acetylcholinesterase staining)
Immaturity of ganglia (or immature ganglionosis)
Hypoganglionosis (or oligoganglionosis)
Congenital hypoganglionosis (or hypogenesis, hypoplasia)
Acquired hypoganglionosis intestinal neuronal dysplasia
(2) Normal ganglia (normal histology in hematoxylin–eosin or acetylcholinesterase staining)
Megacystis microcolon intestinal hypoperistalsis syndrome
Segmental dilatation of intestine
Internal anal sphincter achalasia
Chronic idiopathic intestinal pseudo-obstruction

including 287 definite cases and 68 suspected cases were collected between 2001 and 2010. More than half of the institutes (53 institutes) had three cases or fewer (Figure 1). The mean number of cases per institute was 3.7 cases. There were 165 of 355 cases (47%) treated in university hospitals, 93 (26%) in children's hospitals, and 97 (27%) in general hospitals. ADHD included 28 IG, 130 HG (121 congenital, 9 acquired), and 18 IND in abnormal ganglia; and 33 MMIHS, 42 SD, three IASA, and 100 CIIP in normal ganglia, and these numbers were compared with those of the previous study in Japan (Table 3).[2]

Of the 95 institutes who experienced ADHD, 69 (73%) had their own criteria. The percentages of institutes that had criteria for each disorder were between 28% and 83% (Table 4). More than 80% of institutes had criteria for congenital HG and CIIP, while only ≤ 30% institutes had criteria for acquired HG and IASA. Criteria of each disorder were based on clinical symptoms and signs, examinations including radiography findings, manometric study, and conventional pathological examinations including hematoxylin–eosin (HE; Figure 2) and acetylchoinesterase (AchE). According to answers of the questionnaires, the major criteria listed in each disorder are follows. IG: small ganglion cells, 37/46 (80%); number and distribution of ganglion cells are normal, 19/46 (41%); chronological improvement of clinical symptoms, 8/46 (17%); intestinal obstruction on neonatal onset, 6/46 (13%); normal AchE staining, 3/46 (7%); abdominal distention, 2/46 (4%); and microcolon, 2/46 (4%). Congenital HG: few ganglion cells, 41/55 (75%); few small ganglion cells, 14/55 (25%); intestinal obstruction on neonatal onset, 11/55 (20%); hypoplasia of plexus, 4/55 (7%); normal AchE staining, 4/55 (7%); negative rectospincteric reflex, 4/55 (7%); and delayed meconium pass, 2/55 (4%). Acquired HG: ganglion cells decrease in number after some time, 6/19 (32%); few ganglion cells, 4/19 (21%); normal at birth and symptoms occur after some time, 2/19 (11%); no congenital factors, 2/19 (11%); chronic constipation and persistent bowel dilatation, 2/19 (11%); and normal AchE staining 1 (5%). IND: increased AchE positive fibers in the lamina propria, 17/34 (50%); ectopic ganglion cells, 14/34 (41%); giant ganglia (> 5 ganglion cells per plexus), 13/34 (38%); severe constipation or rectal dysmotility, 9/34 (26%); hyperganglionosis, 6/34 (18%); and dilatation of bowel, 2/34 (6%). MMIHS: megacystis, 39/47 (83%); permanent severe symptoms of intestinal obstruction, 35/47 (74%); microcolon 27/47 (57%); normal histology of intestinal neurons and muscles, 25/47 (53%); neonatal onset, 16/47 (34%); normal AchE staining, 5/47 (11%); and positive rectospincteric reflex 4/47 (9%). SD: persistent segmental dilatation, 36/42 (86%); normal histology of intestinal ganglion cells, 24/42 (57%); no mechanical obstruction distal to dilatation, 13/42 (31%); signs of intestinal obstruction in radiography, 7/42 (17%); complete curability after resection of dilated bowel, 5/42 (12%); abrupt caliber change to the normal intestine, 3/42 (7%); thick or thin muscle layer, 2/42 (5%); and positive rectospincteric reflex, 2/42 (5%). IASA: negative rectospincteric reflex, 9/21 (43%); normal AchE staining, 9/21 (43%); severe constipation since birth, 7/21 (33%); and absence of narrow segment, 4/21 (19%). CIIP: symptoms of intestinal obstruction without mechanical cause, 57/57 (100%); normal histology of intestinal ganglion cells,

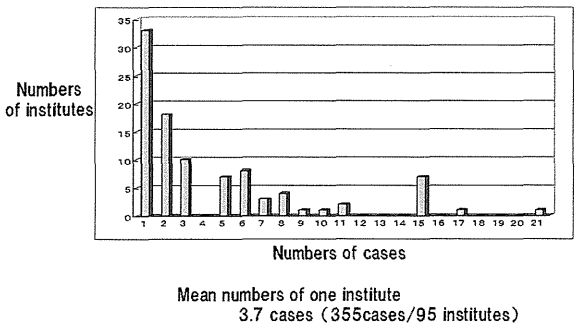

Figure 1 Number of cases in each institute.

Table 3 Numbers of patients in each disorder.

	Definitive	Suspected	Total	Okamoto and Toyosaka[2]
Abnormal ganglia				
IG	22	6	28 (7.9)	26 (24.1)
HG	112	18	30 (36.6)	44 (40.8)
Congenital	104	17	121 (34.1)	
Acquired	8	1	9 (2.5)	
IND	8	10	18 (5.1)	5 (4.6)
Normal ganglia				
MMIHS	27	6	33 (9.3)	9 (8.3)
SD	33	10	43 (12.1)	NE
IASA	1	2	3 (0.8)	NE
CIIP	84	16	100 (28.2)	24 (22.2)
Total	287	68	355 (100)	108 (100)

CIIP = chronic idiopathic intestinal pseudo-obstruction; HG = hypoganglionosis; IASA = internal anal sphincter achalasia; IG = immaturity of ganglia; IND = intestinal neuronal dysplasia; MMIHS = megacystis microcolon intestinal hypoperistalsis syndrome; NE = not examined; SD = segmental dilatation.

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