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Neuroendocrine Cell Hyperplasia of Infancy: Diagnosis With High-Resolution CT

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Abstract

OBJECTIVE—Neuroendocrine cell hyperplasia of infancy is a form of childhood interstitial lung disease originally reported as persistent tachypnea of infancy. Reports of small series of cases and anecdotal experience have suggested that this disorder may have a consistent CT pattern. The purpose of this study was to review the CT findings in children with neuroendocrine cell hyperplasia of infancy to determine the findings at high-resolution CT, the diagnostic accuracy of CT compared with biopsy, and interrater reliability.

MATERIALS AND METHODS—Images from 23 CT examinations of children with biopsy-proven neuroendocrine cell hyperplasia of infancy and six CT examinations of children with other childhood interstitial lung diseases were reviewed by two pediatric radiologists with special expertise in thoracic imaging. Identifying digital data were removed, and images were reviewed without clinical data. A CT assessment form was completed for each patient.

RESULTS—Ground-glass opacification was the most common finding in patients with neuroendocrine cell hyperplasia of infancy. The right middle lobe and lingula were most commonly involved. Air trapping with a mosaic pattern was the second most common finding. Interrater reliability was very good with a kappa value of 0.93. The sensitivity and specificity of CT in the diagnosis of neuroendocrine cell hyperplasia of infancy were at least 78% and 100%.

CONCLUSION—Neuroendocrine cell hyperplasia of infancy can have a characteristic appearance on high-resolution CT scans, the imaging findings being useful in differentiating neuroendocrine cell hyperplasia of infancy from other types of childhood interstitial lung disease.

The appearance aids radiologists in suggesting a specific diagnosis but does not exclude this diagnosis; in 17–22% of cases, the readers in this study did not suggest the diagnosis of neuroendocrine cell hyperplasia of infancy when it was present.

Keywords

children; CT; ground-glass opacification; infants; interstitial lung disease; neuroendocrine cell hyperplasia of infancy; persistent tachypnea

Childhood interstitial lung disease (ILD) includes a diverse group of diseases with varying prognoses [1]. Childhood ILD is much less common than adult ILD, and unlike adult ILD, childhood ILD has no predominant type [2]. Children also have been found to have unique disorders not found in adults. Although lung biopsy continues to be considered the reference standard for diagnosis and advances in surgical technique have decreased complications and recovery time [3], biopsy is performed far less frequently on children than on adults. These factors make diagnosis and treatment frequently challenging in the care of children with ILD.

Neuroendocrine cell hyperplasia of infancy is a form of childhood ILD originally reported as persistent tachypnea of infancy [4]. The incidence is unknown, but cases have been identified at many institutions. Previous reports have included small numbers of patients from single institutions [4, 5]. A multi-center retrospective review of lung biopsy in children younger than 2 years included patients with neuroendocrine cell hyperplasia of infancy [1]. The report by Deterding et al. [4] detailed the pathologic criteria for this diagnosis and the clinical presentation. These reports and anecdotal experience have suggested that a consistent CT pattern may exist in children with neuroendocrine cell hyperplasia of infancy. To our knowledge, no systematic assessment of the imaging appearance has been reported.

Neuroendocrine cell hyperplasia of infancy has a prolonged course with eventual improvement. Corticosteroids have not been effective in improving lung status. Correct identification of this disorder thus has prognostic and therapeutic importance. Immunostaining for bombesin, a procedure not routinely performed, is necessary for pathologic confirmation of the diagnosis, therefore, CT findings suggesting the diagnosis can affect the pathologic evaluation. Improved understanding of the imaging appearance of neuroendocrine cell hyperplasia of infancy on thin-section CT scans should help determine the role of imaging in the evaluation of these children.

We reviewed the CT findings of 23 patients with histologically confirmed neuroendocrine cell hyperplasia of infancy and of six patients with other pathologically confirmed forms of childhood ILD with the intent of determining the high-resolution CT (HRCT) findings of neuroendocrine cell hyperplasia of infancy, the accuracy of CT findings of neuroendocrine cell hyperplasia of infancy compared with biopsy findings; and interrater reliability with use of a standardized CT assessment form in the diagnosis of neuroendocrine cell hyperplasia of infancy.

Materials and Methods

Images from 29 CT examinations of children with pathologically confirmed childhood ILD obtained from three sites participating in a childhood ILD research cooperative were reviewed retrospectively. Identifying information had been removed from the scans. Institutional review board approval was obtained at each site. Demographic information, including age at CT, was recorded. Twenty-three patients had biopsy-proved neuroendocrine cell hyperplasia of infancy. The pathologic findings were characterized by absence of any

other disease process, including clinically significant inflammation, reactive injury, architectural distortion, and fibrosis. At bombesin immunostaining, all biopsy specimens exhibited a marked increase in neuroendocrine cells within the bronchioles and increased size and number of neuroepithelial bodies in the lobules (Fig. 1).

All CT scans were obtained with high-resolution technique. Seven of the patients with neuroendocrine cell hyperplasia of infancy had inspiratory and expiratory images. In five of the seven cases, the slices were 1 or 1.3 mm with a 10-mm interval in the inspiratory phase and a 20-mm interval in the expiratory phase. For one patient the slice thickness was 1.3 mm with a 5-mm interval for the inspiratory phase and a 10-mm interval for the expiratory phase. For the seventh patient, the slice thickness was 1 mm with a 15-mm interval for both inspiratory and expiratory images. In the imaging studies without expiratory acquisition, 2-mm slices at 10-mm intervals were used for nine patients; 1- or 1.3-mm slices at 5-mm intervals for five patients; and 1- or 1.3-mm slices with a 15-mm interval for two patients.

Six patients had a pathologic diagnosis of childhood ILD other than neuroendocrine cell hyperplasia of infancy and had CT features of prominent ground-glass opacification (GGO). These cases were selected by an author who was not a reviewer. CT scans for clinical indications were obtained with a range of techniques. Most of the patients underwent scanning during quiet breathing. CT images were provided to the readers in the same manner as the neuroendocrine cell hyperplasia of infancy images. To produce a degree of diagnostic uncertainty and blinding, the readers were not informed about the total number of cases of neuroendocrine cell hyperplasia of infancy and of other childhood ILD in the study. The diagnoses in the six cases other than neuroendocrine cell hyperplasia of infancy were alveolar growth abnormality due to bronchopulmonary dysplasia, lymphangiectasia, surfactant protein abnormality due to defective adenosine triphosphate binding cassette A3, surfactant protein C abnormality, lymphocytic interstitial pneumonitis, and Niemann-Pick disease.

The digital image data from the CT scans were transferred to CDs with all patient- and hospital-identifying information removed. Each CT scan was reviewed independently by two pediatric radiologists with special expertise in thoracic imaging and childhood ILD. One reader had 18 years in practice, and the other had subspecialty certification in pediatric radiology and 8 years of post-fellowship experience. The readers completed an assessment form for each patient. Parenchymal, airway, and other abnormalities were identified. The lobe involved for each abnormality seen was rank ordered from most to least involved, and central or peripheral location of the abnormality within that lobe was recorded by each reviewer. When more than one abnormality was present on a CT scan, the severity of the abnormalities was recorded by labeling the most prominent abnormality 1, the second-most prominent abnormality 2, and continuing in ascending order for each additional abnormality. Because of difficulty in identifying air trapping on inspiratory images, particularly when GGO is present [6], only patients with expiratory images were evaluated for air trapping. The readers indicated whether the air trapping pattern was mosaic or diffuse. The readers also were asked to indicate on the form cases in which they would make an imaging diagnosis of neuroendocrine cell hyperplasia of infancy. The CT assessment form is shown in Appendix 1.

Descriptive statistics were calculated as mean and SD for continuous variables and percentage for categorical variables. Cohen kappa statistics were calculated for estimation of the level of agreement between the two readers. The lower bound of the 95% CI for the kappa statistic was estimated with the Garner equation as suggested by Blackman and Koval [7]. Sensitivity and specificity were determined for each reader separately.

Results

Patient Demographics

The 23 patients with neuroendocrine cell hyperplasia of infancy were 19 boys and four girls. The mean age at first evaluation by a pulmonologist was 7.4 ± 4.5 months (range, 3–19 months). CT was performed at a mean age of 11.7 ± 8.2 months (range, 3–40 months). Lung biopsy was performed at a mean age of 13.8 ± 9.1 months (range, 4–43 months). The mean time between CT and biopsy was 2.6 ± 3.2 months (range, 0–15 months). Among the six patients without neuroendocrine cell hyperplasia of infancy, CT was performed at a mean age of 33.5 ± 43.0 months (range, 2–108 months).

CT Appearance

Table 1 is an overview of the readers' responses on the CT assessment forms while evaluating the images of patients with a confirmed diagnosis of neuroendocrine cell hyperplasia of infancy. The findings include abnormalities and severity scores by abnormality and the rank order of lobes involved. GGO was the most common finding and had the highest severity ranking. Reader 1 rated GGO the most prominent finding of neuroendocrine cell hyperplasia of infancy in 22 of 23 cases (96%), and reader 2 rated GGO highest in 100% of cases. Reader 1 identified the extent of GGO as greatest in the right middle lobe in 14 cases (61%). The lingula was the next most involved in 10 of these 14 cases. The right lower lobe was the most involved lobe in only one case. Reader 2 identified the extent of GGO as greatest in the right middle lobe in 16 cases (70%). In these 16 cases the lingula was the next most involved in 15 cases. The lower lobes were not the most involved or second most involved in any case.

GGO affected zero to six lobes, the lingula being considered a lobe. Four or more lobes were considered affected in 22 patients (96%) by both readers. In the other patient, GGO was found by reader 1 and not by reader 2. Both readers indicated GGO was geographic in distribution, the central and peripheral portions of the lungs being affected.

Expiratory images were available for assessment of air trapping for seven patients with neuroendocrine cell hyperplasia of infancy and three patients with other diagnoses. Air trapping was identified in six of seven and seven of seven patients with neuroendocrine cell hyperplasia of infancy by readers 1 and 2 and second in severity in six of six and four of seven patients. Both readers identified air trapping as having mosaic attenuation in all seven patients. No air trapping was identified on the expiratory images of the three patients with diagnoses other than neuroendocrine cell hyperplasia of infancy.

Other abnormalities reported by the readers included consolidation, bronchial wall thickening, bronchiectasis, linear and reticular opacity, nodules, and honeycombing (Table 1). Consolidation was identified in four patients by reader 1 and five patients by reader 2, bronchial wall thickening in zero and five patients, and linear and reticular opacification in zero and two patients. Other findings were seen in one patient each.

Interrater Reliability for Imaging Diagnosis of Neuroendocrine Cell Hyperplasia of Infancy

The readers had almost perfect agreement in their diagnoses of neuroendocrine cell hyperplasia of infancy (Table 2). Lack of agreement occurred in one case. The kappa value describing the strength of this agreement was 0.93 with a 0.74 lower bound of the 95% CI, which is considered very good reproducibility [8, 9]. A kappa value of 1 reflects perfect agreement.

Imaging Diagnosis of Neuroendocrine Cell Hyperplasia of Infancy

Compared with the reference standard, lung biopsy and immunostaining, CT was fairly accurate in the diagnosis of neuroendocrine cell hyperplasia of infancy. The sensitivity was 78% and the specificity 100% for reader 1, and the sensitivity was 83% and the specificity 100% for reader 2. Reader 1 considered neuroendocrine cell hyperplasia of infancy present in 18 of the 23 cases and misclassified it in five cases. Reader 2 considered neuroendocrine cell hyperplasia of infancy present in 19 of the 23 cases and misclassified it in four cases (Table 3). Neither reader diagnosed neuroendocrine cell hyperplasia of infancy in patients with other diagnoses. In all cases in which neuroendocrine cell hyperplasia of infancy was absent, the absence was correctly identified.

Five cases of neuroendocrine cell hyperplasia of infancy were misclassified by at least one reader. The abnormalities that contributed to these misclassifications are shown in Table 4. Key findings that appeared to mislead readers included the presence of abnormalities other than GGO and air trapping and a pattern of GGO that did not include the right middle lobe or lingula among the areas most severely affected (Fig. 2).

In summary, neuroendocrine cell hyperplasia of infancy can be best described by the most severe abnormality being GGO involving the right middle lobe and lingula with involvement of at least four lobes, and the second most severe abnormality being mosaic attenuation air trapping (Fig. 3). Other abnormalities occasionally seen in association with GGO and air trapping can lead to misdiagnosis, but these abnormalities are not as severe as the GGO and air trapping.

Discussion

The results of this multicenter review of imaging findings in a group of children with pathologically confirmed neuroendocrine cell hyperplasia of infancy suggest the presence of a constellation of consistent features in children with this disease. Interrater reliability for two experienced pediatric radiologists was high for the diagnosis of neuroendocrine cell hyperplasia of infancy. In at least 78% of cases, neuroendocrine cell hyperplasia of infancy was correctly diagnosed by a radiologist familiar with the radiographic appearance of the disorder. Neither of the readers suggested the diagnosis of neuroendocrine cell hyperplasia of infancy present on the CT scans of any of the six children with other diagnoses. Use of the CT image assessment form also appeared to be a valid method of scoring neuroendocrine cell hyperplasia of infancy. The most prominent features were GGO and air trapping. GGO was characteristically geographic in appearance and widely distributed but was most marked in the lingula and right middle lobe (Fig. 3). In 57–65% of cases, depending on the reader, no other abnormalities were identified. In all cases in which other abnormalities were seen, they were less prominent than GGO and air trapping. In the group without neuroendocrine cell hyperplasia of infancy, GGO also was the most prominent feature, but air trapping was absent in all three patients who had expiratory images.

Our findings suggest that imaging should have an important role in the evaluation of children with neuroendocrine cell hyperplasia of infancy. Infant pulmonary function results showing airflow obstruction and hyperinflation in a small number of patients with neuroendocrine cell hyperplasia of infancy also have been reported [10]. No other biomarkers or genetic tests for this disorder have been identified, although this is an area of active research. Further investigation of the relation between air trapping on HRCT images and hyperinflation at pulmonary function testing of infants is needed. Determining whether confirmatory biopsy is necessary for children with classic clinical features and the imaging characteristics of neuroendocrine cell hyperplasia of infancy found in this study also is important.

Our data confirm previous suggestions that neuroendocrine cell hyperplasia of infancy can have a characteristic imaging appearance at HRCT and that these imaging findings are useful in differentiating neuroendocrine cell hyperplasia of infancy from other types of pediatric ILD. Unlike many forms of childhood ILD, the imaging appearance of neuroendocrine cell hyperplasia of infancy can lead the radiologist to suggest a specific diagnosis. Imaging findings, however, cannot be used to exclude the diagnosis. In this study, the readers did not suggest the diagnosis of neuroendocrine cell hyperplasia of infancy in four and five of 23 cases (17% and 22%) of children. Patients' conditions frequently are misclassified when other abnormalities are present and the distribution pattern of GGO differs from the classic finding of severe right middle lobe and lingular involvement.

The appearance of neuroendocrine cell hyperplasia of infancy differs from that of other forms of childhood ILD characterized by air trapping and GGO. Bronchiectasis is a primary finding in bronchiolitis obliterans. Among the 23 patients with neuroendocrine cell hyperplasia of infancy in this study, bronchiectasis was found in only two patients as a less prominent finding. Air trapping is a prominent finding in asthma, but GGO is much less prominent and does not preferentially affect the right middle lobe and lingula. GGO is a major feature of surfactant protein abnormality but is more diffuse than in neuroendocrine cell hyperplasia of infancy and is associated with septal thickening, which was not seen in any of our patients.

Detection of air trapping and GGO is relatively insensitive to substantial image noise [11]. This phenomenon suggests that reducing radiation exposure with very-low-dose protocols may be appropriate in the evaluation of children with neuroendocrine cell hyperplasia of infancy. However, when the first CT examination is requested, the differential diagnosis probably includes causes of childhood ILD in addition to neuroendocrine cell hyperplasia of infancy. If the CT findings do not suggest the presence of neuroendocrine cell hyperplasia of infancy, the images should be of sufficient quality to allow adequate diagnosis of other forms of childhood ILD. A high-resolution protocol with both inspiratory and expiratory imaging would facilitate evaluation of the lung parenchyma. If evaluation for other abnormalities, such as those of the mediastinum and central airways, is needed, a protocol of contiguous images at inspiration and high-resolution images at expiration can be performed with a low radiation dose. For example, high-resolution CT performed with a 16-MDCT scanner at 10-mm intervals at 100 kVp, 50 mAs, and 1.25-mm collimation has an effective dose of 0.8 mSv. Expiratory images obtained at 20-mm intervals with otherwise the same technique add 0.4 mSv, for a total dose of 1.2 mSv. This dose is equivalent to the average background radiation received by persons in the United States every 4–5 months.

This study had several limitations. The total number of patients included was relatively small, but for a rare lung disease, 23 biopsy-confirmed cases is a significant number. The children with other forms of ILD were older, but the age difference was not apparent at review of the CT images. Expiratory images were not available for most of the patients. The sample was limited to 23 children with a pathologic diagnosis of neuroendocrine cell hyperplasia of infancy and six children with other forms of childhood ILD. Considering the selection of disease groups within this retrospective design, the reported false-positive rate may not reflect that which would be observed in a prospective study. In addition, some pulmonologists care for children with typical imaging findings, infant pulmonary function, and clinical findings of neuroendocrine cell hyperplasia of infancy without requesting lung biopsy. The sample in this study therefore also might have been biased toward patients with atypical clinical and imaging features.

Neuroendocrine cell hyperplasia of infancy has a typical HRCT appearance that differs from the appearance of other forms of childhood ILD. When this appearance is seen, the specific

diagnosis of neuroendocrine cell hyperplasia of infancy should be considered. Future studies are needed to determine whether HRCT can replace lung biopsy as a definitive diagnostic test in the evaluation of this disorder.

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APPENDIX 1

CT Assessment

Number _____ Reader _____

Neuroendocrine cell hyperplasia of infancy present _____ (yes/no)

Circle all abnormalities seen on each CT scan. If more than one parenchymal or airway abnormality is present, list abnormalities in order of severity, 1 indicating most marked abnormality. For parenchymal and airway abnormalities, list sites of involvement by lobe in order of severity. For example, bronchiectasis involving only the right lung, greatest in the apex and least in the right middle lobe would be entered follows: Bronchiectasis RUL, RLL, RML. Also indicate C for central or perihilar and P for peripheral.

Parenchymal Abnormality

Interlobular septal thickening

Linear or reticular opacity

Nodules

Honeycombing

Consolidation

Ground-glass opacification

Cysts

Air trapping, diffuse

Air trapping, mosaic

Airway Abnormality

Bronchiectasis

Bronchial wall thickening

Mucous plugging

Tree-in-bud or centrilobular nodules

Other Abnormalities

Cardiomegaly

Increased vascularity

Pleural effusion

Pleural thickening

Thickened fissures

Pneumothorax

Adenopathy

Calcification

Fig. 1. 6-month-old boy with neuroendocrine cell hyperplasia of infancy. Photomicrograph obtained after immunostaining with bombesin shows increased number of neuroendocrine cells within bronchiole and large neuroepithelial bodies (*inset*). (bombesin, $\times 200$; inset, bombesin, $\times 400$)

Fig. 2.

5-month-old boy with biopsy-proven neuroendocrine cell hyperplasia of infancy. Neither reader considered neuroendocrine cell hyperplasia of infancy present. **A–C**, Inspiratory high-resolution CT scans show abnormalities are most marked in upper (**A**) and middle (**B**) portions of lungs, sparing lower parts (**C**). Ground-glass opacification (*asterisk*, **B**) is evident, as are areas of volume loss (*arrows*, **B**) and architectural distortion (*arrow*, **A**). **D–F**, Expiratory high-resolution CT scans show hyperlucent areas indicating air trapping is most marked in upper portion of lungs (**A**, **E**) with no air trapping in lung bases.

Fig. 3.
40-month-old girl with typical appearance of neuroendocrine cell hyperplasia of infancy.
A–C, Inspiratory high-resolution CT scans show sharply defined areas of ground-glass opacification along mediastinal borders, peripherally, and most prominently in right middle lobe and lingula (*asterisks, C*).
D–F, Expiratory high-resolution CT scans show only mild increase in attenuation in all areas of lung, consistent with air trapping involving both areas with ground-glass opacification and areas with normal attenuation.

TABLE 1

CT Findings in All Patients With Neuroendocrine Cell Hyperplasia of Infancy

Abnormality	Reader 1			Reader 2		
	No.	Severity	Lobes in Rank Order	No.	Severity	Lobes in Rank Order
Ground-glass opacification	22	1.0	RML, lingula, RUL, LUL, RLL, LLL	23	1.0	RML, lingula, RUL, LUL, RLL, LLL
Air trapping	6	2.0		7	2.6	
Consolidation	4	2.7	RLL, RML, LLL, RUL, LUL	4	2.8	RML, RLL, lingula
Linear or reticular opacification	1	3.0	RUL, LUL, LLL	2	2.0	LLL, LUL, RLL, RUL, lingula
Bronchiectasis	1	3.0		1	4.0	Lingula
Bronchial wall thickening	0			6	3.0	RLL, LLL, LUL, RUL
Nodules	1	3.0	RLL			
Honeycombing	0			1	3.0	LUL, RUL
Interlobular septal thickening	0			1	4.0	RUL

Note—RML = right middle lobe, RUL = right upper lobe, LUL = left upper lobe, RLL = right lower lobe, LLL = left lower lobe.

TABLE 2

Reader Agreement on Diagnosis of Neuroendocrine Cell Hyperplasia of Infancy

Reader 2	Reader 1		
	Absent	Present	Total
Absent	10	0	10
Present	1	18	19
Total	11	18	29

Note—Values are numbers of cases.

TABLE 3

Ability of Readers to Diagnose Neuroendocrine Cell Hyperplasia With CT Compared With Biopsy Findings

CT Finding	Biopsy Finding		
	Absent	Present	Total
Reader 1			
Absent	6	5	11
Present	0	18	18
Total	6	23	29
Reader 2			
Absent	6	4	10
Present	0	19	19
Total	6	23	29

Note—Values are numbers of cases.

TABLE 4

Misclassified CT Findings in Patients With Neuroendocrine Cell Hyperplasia of Infancy

Patient No.	Reader 1		Reader 2	
	Abnormality by Severity	Lobes in Rank Order for GGO	Abnormality by Severity	Lobes in Rank Order for GGO
2	GGO Air trapping Consolidation	RLL, LLL, RML, RUL, LUL	GGO Linear or reticular opacity Consolidation Air trapping	RML, RLL, LLL, lingula, RUL, LUL
21	GGO Consolidation	RUL, LUL, LLL	GGO Air trapping Consolidation Bronchial wall thickening	RUL, LUL
22	No abnormality		GGO	All lobes
23	GGO Air trapping Linear or reticular opacity	LUL, RUL, RLL, LLL	GGO Linear or reticular opacity Honeycombing Air trapping	LUL, RUL, RLL, lingula, RML, LLL
25	GGO	LUL, RUL, RML, RLL	Correct diagnosis	Correct diagnosis

Note—Air trapping was evaluated for only seven patients with or without neuroendocrine cell hyperplasia of infancy who had expiratory images.

GGO = ground-glass opacification, RLL = right lower lobe, LLL = left lower lobe, RML = right middle lobe, RUL = right upper lobe, LUL = left upper lobe.