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Journal

Hormone and Metabolic Research, 48(8)

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Publication Date

2016-08-01

DOI

10.1055/s-0042-106725

Peer reviewed

HHS Public Access

Author manuscript

Horm Metab Res. Author manuscript; available in PMC 2020 August 21.

Published in final edited form as:

Horm Metab Res. 2016 August ; 48(8): 509–513. doi:10.1055/s-0042-106725.

Attention Deficit Hyperactivity Disorder in Pediatric Patients with Pheochromocytoma and Paraganglioma

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Abstract

The aim of the study is to evaluate if there is an association between attention deficit hyperactivity disorder (ADHD) and the diagnosis of pheochromocytoma/paraganglioma (PHEO/PGL) in pediatric patients. A case series study of 43 patients under the age of 18 with PHEO/PGL tumors who were evaluated at the National Institute of Health between January 2006 and May 2014 is reported. Prior diagnosis of ADHD and treatment course with stimulant medications was recorded. Patient symptoms, catecholamine and metanephrine levels, tumor characteristics, and genetic analyses for syndromes associated with PHEO/PGL were evaluated. A chi-squared test was used to assess the prevalence of ADHD in the PHEO/PGL patients compared to the general population. Nine out of 43 (21%) of patients diagnosed with PHEO/PGL had been diagnosed with ADHD prior to tumor identification. Four of the 9 patients had been treated with amphetamine, dextroamphetamine, and/or methylphenidate, potentially exacerbating an adrenergic crisis. In addition, 4 patients exhibited hypertension at the initial diagnosis of their PHEO/PGL. Three patients had resolution of their ADHD symptoms after successful surgical removal of PHEO/PGL. Our study found a prevalence of ADHD in 21% of our PHEO/PGL patients, significantly higher than 7.2% seen in the general pediatric population. Symptoms of anxiety and difficulty in concentration in these patients may have been related to their underlying PHEO/PGL and were not recognized as part of the constellation of symptoms in a child with PHEO/PGL. In pediatric

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Conflict of Interest

The authors declare no conflict of interest.

patients with hypertension and ADHD symptomatology, an evaluation to rule out PHEO/PGL is warranted prior to treatment with stimulant medications.

Keywords

catecholamines; neuroendocrine tumor; epinephrine

Introduction

Pheochromocytomas (PHEOs) are rare neuroendocrine tumors that originate from chromaffin cells in the adrenal medulla and may secrete excess catecholamines [1]. Paragangliomas (PGLs) arise in extra-adrenal paraganglia of the sympathetic and parasympathetic systems [2]. The incidence of PHEO/PGLs in the U.S. is about 1 in 2500–6500 [1,3]. PHEO/PGL typically occurs around the fourth and fifth decade, however 10% are seen in children, usually in the setting of an inherited or sporadic genetic syndrome. These mainly include familial PGL due to succinate dehydrogenase subunit (SDHx) mutations, multiple endocrine neoplasia type 2A or B (MEN2A, MEN2B), neurofibromatosis type 1 (NF-1), and von Hippel-Lindau syndrome (VHL) [1,3,4]. Some of the most common clinical presentations of PHEO/PGL include paroxysmal hypertension, headaches, excessive sweating, palpitations, dyspnea, and weight loss [5]. In children, 1–2% of cases of hypertension can be attributed to underlying PHEO/PGL [3,6]. Psychiatric symptoms including anxiety or panic disorder may also be part of the presenting symptoms, occurring with a frequency of 20–40% in adults, while the exact frequency in children is unknown [7].

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder with a prevalence of 7.2% in the age group 4–18, and is higher in males, with a 2.3: 1 male to female ratio [8,9]. The Diagnostic and Statistical Manual of Mental Disorders V (DSM-5) and the American Academy of Pediatrics (AAP) provide guidelines for the diagnosis of ADHD that specify that it is a diagnosis of exclusion that cannot be attributed to external stimuli, medications, or disorders [10]. ADHD is characterized by a pattern of behavior that causes performance issues in multiple settings with symptoms being divided into 2 categories of inattention and hyperactivity/impulsivity. Symptoms include being easily distracted, fidgeting, excessive talking, bursts of energy and activity as well as anxiety and nervousness [10]. These symptoms may overlap with those seen in children with metabolically active PHEO/PGL.

Haws et al. reported 2 cases of PHEO patients that were initially treated and diagnosed for ADHD until subsequent diagnosis of PHEO was made. One of these patients underwent treatment with dextroamphetamine/amphetamine for 4 months, and over this time frame developed new onset headaches, palpitations, excessive sweating, and hypertension. Norepinephrine was elevated over 10 times the upper limit of normal (ULN), dopamine 4 times the ULN, and metanephrine 2.5 times the ULN. Hypertension and ADHD resolved after the tumors were surgically resected [11]. With the large PHEO/PGL pediatric patient cohort seen at the National Institutes of Health (NIH) we had the unique ability to assess

whether there is a relationship between ADHD diagnosis and PHEO/PGL development as well as to explore how subsequent treatment of PHEO/PGLs affects the course of ADHD symptoms.

Patients and Methods

Patients with PHEO/PGL under 18 years of age were evaluated at the NIH Clinical Center between January 2006 and May 2014 (n=43, 18F, 25M, median age 14 years, range 6–17). Patients were seen through the protocol 00-CH-0093, Diagnosis, Pathophysiology, and Molecular Biology of Pheochromocytoma and Paraganglioma. Study evaluations were approved by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development Institutional Review Board, and informed consent was obtained from the legal guardians of participants. On evaluation at the NIH, patients underwent biochemical assessment for plasma catecholamines and metanephrines. Blood samples were collected from a forearm venous line after laying supine for 20 min after catheter insertion before sampling as previously described. For patients in whom surgical resection of their PHEO/PGL was performed prior to their initial evaluation at the NIH, historical data was collected from their referring institution, and values normalized to the relevant reference range. Genetic testing was performed for mutations or large deletions in *RET, VHL, SDHA, SDHB, SDHC*, and *SDHD*. All children were found to be euthyroid on measurement of TSH and free T4. Imaging was used to identify the location, type, and metabolic activity of the tumors in the patients.

Surgical history and anatomic pathology were reviewed. Patients with an initial diagnosis of ADHD made by the child's pediatrician prior to surgery (n = 9) were re-evaluated an average of 2±0.6 years after their surgery (range 1.3–3.2) to assess their PHEO/PGL and to elucidate the status of their ADHD symptoms. A chi-squared test compared the observed prevalence of ADHD in the PHEO/PGL cohort to what would be expected based on population estimates.

Results

Of the 43 patients diagnosed with PHEO/PGL, that were seen at NIH, 9 (21%) carried a diagnosis of ADHD compared to 7.2% in the general population (p = 0.0328). Characteristics of the patients are presented in Table 1. Among the individuals with ADHD, there were 7 males and 2 females, 7 Caucasians, 1 African American, and 1 patient of multiple races. Out of the 9 patients 4 (44%) were found to have hypertension as a presenting symptom. The age of diagnosis of ADHD was at a mean of 8 ± 3 years. The average age when their first symptom attributed to PHEO was identified was 11±3 years, while the average age of tumor identification was 12±3 years. In 1 of the 9 patients, tumor metabolites were not measured prior to their PHEO/PGL resection. All 8 patients with ADHD and PHEO/PGL with preoperative biochemical testing had at least one catecholamine or metanephrine level at or above the upper limit of normal in urine and/or plasma (Table 2). The breakdown of individuals with elevated pre-operative metabolites is as follows: norepinephrine (n = 7), dopamine (n = 3), epinephrine (n = l), metanephrine (n

= 5), and normetanephrine (n = 7). In 4 out of 5 patients with preoperative chromogranin A measurement, elevated levels were detected.

Two patients presented with a solitary PHEO, 1 had bilateral PHEOs in the setting of MEN2A, 3 had a single PGL, and 3 had multiple PGLs. Five patients harbored germline mutations in *SDHB*, 1 patient was found to have a mutation in *RET* consistent with MEN2A, 1 patient had familial *VHL*, 1 patient had a mutation in *SDHA* (Table 3). At the most recent post-operative follow-up visits between 1.3–3.2 years after surgery, 6 of the 9 patients (66%) had no signs of PHEO/PGL recurrence; one patient is still being monitored due to suspicion of the presence of a small PHEO/PGL and the remaining 2 patients have metastatic disease (Table 4). After surgical resection of their tumors, 3 out of the 9 patients (33%) experienced resolution of their symptoms that had been attributed to ADHD. Among the patients that had resolution of their ADHD after surgical resection of their PHEO/PGL, 2 have shown no clinical signs of PHEO/PGL recurrence with complete normalization of their catecholamines and metanephrines while one is under evaluation for a small pelvic lesion, with a drop in her norepinephrine levels from 38 times the ULN down to 1.2 times the ULN. Of the patients that had a persistence of ADHD symptoms post-operatively, 2 have metastatic PGL with persistent elevation of tumor metabolites. Two patients continue on stimulant medication (Table 4).

Discussion

Our findings show that in our cohort of pediatric PHEO/PGL patients, 21% had a prior diagnosis of ADHD compared to the 7.2% seen in the general population. Due to the overlapping symptoms between ADHD and PHEO/PGL, the diagnosis may present a challenge. Both the DSM-5 and the AAP specify that ADHD must be a diagnosis of exclusion using observations from multiple parties to verify whether the symptomology matches the diagnosis. The risk of misdiagnosis of ADHD in cases of PHEO/PGL is concerning in part because the stimulants used to treat ADHD may exacerbate the symptoms of the PHEO/PGL and potentially lead to a hypertensive crisis, as previously described in one published case [11]. Amphetamines, the most widely used ADHD medication class, lead to release of stored catecholamines from vesicles, block re-uptake of norepinephrine and dopamine, and block catecholamine degradation [12]. The use of stimulants on top of underlying elevated norepinephrine and dopamine in patients with PHEO/PGL may potentiate their sympathomimetic effect. The prevalence of sustained hypertension in children with PHEO/PGL is 60–90% with 14.5% experiencing intraoperative hypertension due to catecholamine secretion [13]. The clinical course of pheochromocytoma may be adversely affected by drug administration, and severe crises have been induced in adults by the stimulants ergotamine, caffeine, pseudoephederine, and sympathomimetic amines [14–16].

In our patient cohort, we had 4 patients who exhibited documented hypertension in conjunction with their ADHD symptoms. Because hypertension is rare in the pediatric population, practitioners should perform a thorough workup of organic causes. Over recent years, the diagnosis of ADHD and use of medication for treatment has increased [17].

If PHEO is suspected, recommendations include the use of biochemical assays as well as imaging modalities [18]. It is imperative that a thorough evaluation for PHEO/PGL is performed in a child with ADHD-symptoms and hypertension, especially if there is a family history of neuroendocrine disorders. Recently, concerns about the cardiovascular safety of stimulant medications have led to practice guidelines by the American Heart Association (AHA) and the AAP [19]. Warnings on the labels of stimulant medications themselves specifically indicate that they should not be used in children with cardiac problems that put them at high risk of sympathomimetic effects: “Caution is indicated in treating patients whose underlying medical conditions might be compromised by increases in blood pressure or heart rate, for example, those with pre-existing hypertension” (Prescribing information, Focalin XR, Novartis 2015). AHA guidelines recommend that in patients with heart rate or blood pressure more than 2 standard deviations above the mean, discontinuation of the medication, and further cardiovascular testing is indicated [19]. The AAP recommends that physicians carefully assess children being considered for ADHD therapy by getting a careful history of cardiac disease, palpitations, syncope, family history for cardiac disease, and a cardiac examination, prior to initiating medical treatment [20].

Evidence supports that the dysfunction in ADHD is caused by disturbances of catecholamine neurotransmission at the level of the prefrontal cortex [21]. While catecholamine dysregulation in ADHD is characteristically thought to be due to depleted levels of dopamine and norepinephrine, function of these receptors can be impaired by either deficient or excess stimulation. Extremely high concentrations of norepinephrine and/or dopamine in PHEO/PGL may be detrimental to the function of the prefrontal cortex, similar to the elevation seen during stress [22]. The levels of dopamine and norepinephrine in PHEO/PGL may be outside of the optimum range for normal physiologic functioning, and could potentially contribute to the overlapping symptomatology [23]. Importantly, in all 7 of our patients in whom we have preoperative plasma or urinary norepinephrine levels, all patients have values that exceed the upper limit of normal, as highlighted in Table 2.

One important limitation of our study is the lack of a control group consisting of patients with solid tumors that do not release catecholamines, as the presence of a tumor itself may make a child more vulnerable to developing anxiety. A larger study incorporating prospective screening for ADHD children with PHEO/PGL as compared to children with other types of solid tumors would be important as a future investigation.

Conclusion

This study showed that within the 43 PHEO/PGL patients, 21% had a prior diagnosis of ADHD compared to the 7.2% seen in the general population. Out of these predominantly male patients, 33% had resolution of their ADHD symptoms after surgical resection and were able to stop treatment with stimulant medications. In addition to these findings, 44% of our patients exhibited hypertension at initial diagnosis. These observations point to a need for pediatric patients with ADHD and hypertension to undergo a thorough evaluation to rule out the presence of a PHEO/PGL prior to initiation on stimulant medication.

Acknowledgements

This work was supported by the Division of Intramural Research of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development.

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Table 1

Clinical Characteristics of the patients.

	Pheochromocytoma/paraganglioma No ADHD n = 34	Pheochromocytoma/paragaganglioma with ADHD n = 9	p-Value
Age at the diagnosis of PHEO/PGL? (years), mean±SD	12.2±2.5	11.9±3.0	0.71
Gender	18M (53%)	7M (78%)	0.26
	16F	2F	
Hypertension	14 (41.18%)	4 (44.44%)	1.00
Anxiety	1 (2.94%)	3 (33.3%)	0.02
SDHB+	20 (50.82%)	5 (55.56%)	1.00
Plasma (Pl) norepinephrine (80–498 pg/ml)	3641±6255	5193±7399	0.12
Pl dopamine (3–46 pg/ml)	53±104	1184±1048	0.83
P epinephrine (4–83 pg/ml)	31±25	62±55	0.13
Fractionated Pl metanephrine (12–61 pg/ml)	44±44	120±190	0.60
Fractionated Pl normetanephrine (18–112 pg/ml)	1132±1388	1238±1821	0.90
Chromogranin A (< 225 ng/ml)	887±1 950	709±567	0.16

Horm Metab Res. Author manuscript; available in PMC 2020 August 21.

Table 2

Fold above the upper limit of normal for biochemical studies (urine or plasma) at the time of diagnosis of pheochromocytoma/paraganglioma.

Patient	Norepinephrine	Dopamine	Epinephrine	Metanephrine	Normetanephrine	Chromogranin A
1	38.4	51.3	0.5	0.6	42.9	7.0
2	nd	0.8	0.3	nd	37.5	nd
3	1.1	0.2	0.5	1.0	2.0	1.4
4	15.9	nd	2.1	nd	nd	nd
5	3.8	0.8	nd	8.2	5.3	nd
6	1.1	0.2	0.4	1.7	3.0	0.4
7	nd	nd	nd	nd	nd	nd
8	5.0	1.1	0.2	10.9	15.2	3.5
9	1.0	1.0	0.2	2.7	3.2	3.4

nd: No data

Table 3

Genetic results.

Patient number	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
Mutation	*SDHB*	Negative	*VHL*	*SDHB*	*SDHB*	*RET*	*SDHB*	*SDHB*	*SDHA*
Mutation Type	Exon 1 Deletion			Exon 4 (c.418G>T, p.Val140Phe)	Exon 3 (c.268C>T)	C.1900T>C, p.Cys634Arg	Exon 4 (c.418G>T, p.Val140Phe)	Exon 7 (c.725G>A, p.Arg242His)	*c.91C>T, p.Arg31X.*

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Table 4

Follow-up data.

Patient number	1	2	3	4	5	6	7	8	9
Type of surgery	**Multiple pelvic tumors resected**	**Right adrenalectomy**	**Removal of retroperitoneal PGL**	**Right extra adrenal PGL**	**Aortocaval PGL**	**Bilateral adrenalectomy**	**Left adrenalectomy**	**Multiple Para aortic PGLs removed**	**Multiple PGLs in thorax removed**
Time elapsed from surgery to most recent follow-up (years)	3.2	2.0	2.8	2.0	2.0	2.4	1.5	1.6	1.3
Catecholamine and metanephrines values at most recent follow-up	NMN 2.2 X ULN NE 1.2 X ULN MN 1.8×ULN	WNL	WNL	WNL	WNL	Not done	WNL	NE1.6×ULN D 2×ULN NMN 2.8×ULN MN 2.3×ULN	D 1.2×ULN
Pheo/Para status at follow-up? (Remission, Metastatic, etc.)	Small metastatic pelvic lesion	Remission	Remission	Remission	Remission	Remission	Remission	Widespread skeletal disease +metastatic lymph nodes	Retroperitoneal mass skeletal metastases
Symptoms of inattention, hyperactivity, impulsivity	Resolution of ADHD	Persistence of symptoms of inattention and aggression	Persistence of ADHD	Resolution of ADHD	Resolution of ADHD	Improvement of ADHD	Continues to have panic attacks and anxiety	Persistence of ADHD and coexisting neuro-developmental delay	Persistence of ADHD
On ADHD meds at time of post-surgical follow-up?	No	No	Amphetamine salts, Guanfacine	No	No	No	No	No	Atomoxetine