# **Special Article - Anatomical Pathology**

# Lamina Papyracea Dehiscence CT Incidence in a Paediatric Population with Medial Orbital Wall Review

# Hendy K<sup>1\*</sup>, Taranath A<sup>2</sup>, Ghabriel M<sup>3</sup> and Nasserallah M<sup>4</sup>

<sup>1</sup>Radiology Consultant, Repat Health Precinct, 216 Daws Rd, Daw Park, SA 5041, Australia <sup>2</sup>Expert Paediatric Neuroradiologist, Women's and Children's Hospital, 72 King William Road, North Adelaide, SA 5006, Australia

<sup>3</sup>Associate Professor of Anatomy, Adelaide Medical School, University of Adelaide, 30 Frome Road, Adelaide, SA 5000, Australia

<sup>4</sup>ENT Surgical Registrar, Peninsula Health, 2 Hastings Road, Frankston VIC 3199, Australia

\*Corresponding author: Hendy K, Radiology Consultant, Repat Health Precinct, 216 Daws Rd, Daw Park, SA 5041, Australia

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### Abstract

**Background:** Congenital Lamina Papyracea Dehiscence (LPD) is a described anatomical variant, knowledge of which is important to avoid misdiagnosis of orbital fractures, to indentify this variant prior to sinonasal instrumentation including Functional Endoscopic Sinus Surgery (FESS) and to improve interpretation when evaluating orbital and sinus pathologies such as infection and tumour infiltration. We aim to quantify the prevalence of LPD in children presenting for Computerized Tomography Scan (CT) of the paranasal sinuses.

**Methods:** The database in the Radiology and Imaging System (RIS) for all CT scans of paranasal sinuses performed between 1/1/2019 and 31/1/2021 at the Women's and Children's Hospital (WCH), Adelaide, South Australia, were accessed for this study. Patients aged above 18 years were excluded. Subjects with repeat studies were only included once. Standardized CT imaging was performed on a GE machine. Images obtained were 0.6 mm thick and were subjected to reformatting during observer interpretations. Imaging review was performed by two readers who agreed on the findings. Prevalence was calculated by dividing the number of subjects with LPD by the total number of subjects included.

**Results:** Using the above criteria 90 subjects were included in the study, and among these, one patient was noted to have congenital LPD.

**Conclusion:** Congenital LPD is an uncommon anatomical variant and was found in 1.1% of children presenting for CT evaluation of the nasal sinuses in this study of a South Australian paediatric population.

**Keywords:** Lamina papyracea; Congenital lamina papyracea dehiscence; Radiology and imaging system

# Introduction

The orbital plate of the ethmoid forms a major part of the medial wall of the orbit. This plate is fragile and extremely thin, hence known as the Lamina Papyracea (LP). Congenital Lamina Papyracea Dehiscence (LPD) is a described anatomical variant [1]. Knowledge of this anatomical variant is important for several reasons [2]. These include avoiding misdiagnosis of orbital fractures, identification of variant anatomy prior to sinonasal instrumentation including Functional Endoscopic Sinus Surgery (FESS) and for improving accuracy of interpretation when evaluating orbital and sinus pathologies such as infection and tumour infiltration. Misdiagnosis of this entity predisposes patients to several risks including unnecessary medical treatment and perforation of the medial orbital wall during FESS. Other possible FESS related complications consequent upon failure to recognize this variant include injury to extraocular muscles, orbital hematoma and orbital infection [2,3].

To our knowledge, the CT prevalence of congenital LPD extracted specifically from a paediatric population has not been reported previously. It is important to note that the pathogenesis of congenital LPD is distinct from the acquired dehiscence secondary to extensive sinonasal polyps in which the ethmoid sinuses are not filled with orbital fat [3].

## Aim

To quantify the prevalence of LPD in children presenting for CT scans of the nasal sinuses.

## **Materials and Methods**

# **Patient cohort**

Our study was performed in accordance with ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments thereby meeting criteria for consent waiver by the local health network Human Research Ethics Committee.

The radiological database of South Australia's largest paediatric referral center, the Women's and Children's Hospital (WCH), was accessed for selecting includable patients. All patients who underwent a CT scan of the sinuses at WCH anytime in between 1/1/2019 and 31/1/2021 (25 months) were retrospectively screened for inclusion. Patients greater than eighteen years of age were excluded. Patients with repeat studies during this period were only included once.

#### **CT** examinations

Studies were performed on a 64 slice multidetector GE CT scanner, which yielded volumetric data of the paranasal sinuses and orbits. Studies were performed without administration of intravenous

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#### Hendy K

contrast material. All patients were in supine position and received a cross section scan. Craniocaudal extent of scanning range was from the superior border of frontal sinus to the inferior border of maxillary alveolar process. Bone algorithm imaging (parameters: reformation slice thickness of 0.63mm, increment of 0.6mm and pitch of 0.562), and multiplanar reformation of multi-slice CT were used for continuous and dynamic observation of ethmoidal sinuses and their adjacent structures in axial, coronal and sagittal planes. The Multiplanar Reconstructions (MPR) were performed with Carestream PACS software programs, which provide a simultaneous display of axial, coronal, and sagittal planes in double oblique mode allowing orthogonal triangulation of anatomical structures in multiple planes and precise visualization of ethmoid foramina, basal lamella and bulla lamella.

Imaging review was performed on quality assured and calibrated diagnostic BARCO monitors utilized in routine diagnostic radiology interpretation at the WCH. Each study was scrutinized for defects in the medial orbital wall by a single observer (KH) under a double oblique mode on two separate reporting standard diagnostic BARCO monitors. Orthogonal triangulation was achieved by adjusting the image angles using the following landmarks: sagittal plane was viewed in line with the perpendicular plate of the ethmoid bone, coronal plane was perpendicular to the floor of the anterior cranial fossa and also viewed in line with the perpendicular plate of the ethmoid bone, axial plane was therefore aligned according to the aforementioned parameters. All three planes were interrogated for analysis. Any uncertainty in imaging interpretation prompted review by the second observer (AT) and an agreed determination on the findings was established.

Information extracted from each examination included: basic patient demographics, scan indication, the presence or absence of LPD, the content of dehiscence, characterization of the ingression location and dimensions, and any associated paranasal sinus pathology. The total number of patients with LPD were divided by the total number in the study group to determine congenital LPD prevalence.

## **Results**

A total of 106 paranasal sinuses CT scans were performed at WCH between 1/1/2019 and 31/1/2021. Five patients were aged above 18 years and therefore were excluded from our study population. One patient had scout images obtained but refused to lie still to acquire the CT and therefore assessment of the lamina papyracea was not possible. Five patients had multiple scans within this time period and therefore were only included once. Hence, a total of 90 subjects (180 laminae papyracea) were assessed in this study.

Of the included subjects, 53 were males. The ages ranged from 15 days to 17 years (average 11 years). The two most common indications for patients undergoing paranasal sinuses CT scans in our study were categorized as infection/inflammation (20%) and obstructive rhinitis (26%).

One of the 90 subjects (1.1%) had evidence of LPD. This was unilateral on the left side (Figure 1A and 1B). The defect measured 1.6 x 8.7 x 5.2 mm (transvers x anterior posterior x craniocaudal) and was located anterior the basal lamella. The defect harboured the left



Figure 1A: Left lamina Papyracea dehiscence in a 16-year-old female seen on CT of the paranasal sinuses. Bone window demonstrates a left medial orbital wall defect (short black arrow) located anterior to the left basal lamella (long black arrow). These features are compatible with congenital dehiscence. The bulla lamella is indicated with a white arrow. The remainder of the orbit is unremarkable and there is no scan evidence of prior trauma. The remainder of the paranasal sinuses and mastoid air cells are clear.



Figure 1B: Soft tissue window demonstrates the presence of fat herniation into the left lamina papyracea dehiscence, the anterior and posterior boundaries of which are indicated with short arrows. The medial boundary of the lamina Papyracea dehiscence is indicated with a long arrow. There is asymmetrical muscle belly thickness of the Left Medial Rectus (LMR) compared to its contralateral counterpart supporting muscle hypertrophy, which is a described association with lamina papyracea dehiscence. However, there is no displacement or bowing of the Left Medial Rectus muscle (LMR).

anterior ethmoidal foramen, which transmits the anterior ethmoidal nerve and vessels. There was accompanying extraconal orbital fat herniation into the defect. The ipsilateral medial rectus muscle belly was asymmetrically hypertrophied (Figure 1B) compared to its contralateral counterpart, but no significant bowing was observed. There was general background mild mucosal thickening of the maxillary and ethmoid sinuses in this patient without evidence of bony erosion elsewhere and the scan indication was for assessment of sinusitis.

# Discussion

Our findings indicate that congenital LPD is not commonly encountered. It is also noted to be an under-recognized entity evidenced by not being reported in our patient. Its under recognition and misinterpretation may have negative impacts on patients'



Figure 2: Image of the left orbit.

The medial wall of the orbit extends from the anterior lacrimal crest (1) which is located on the frontal process of the maxilla (yellow) to the optic canal (6) located in the sphenoid bone. The lacrimal bone (green) carries the posterior lacrimal crest (3). The fossa for the lacrimal sac (2) is formed by the frontal process of the maxilla (yellow) and lacrimal bone (green). The orbital plate (lamina papyracea, 4, pink) of the ethmoid bone forms a large part of the medial orbital wall. The posterior (7) and anterior (8) ethmoidal foramina are seen along the suture between the frontal bone (F) and the ethmoid bone (pink). Maxilla (M); Nasal bone (N); Zygomatic bone (Z). (Obtained from a skull housed in the Anatomy Museum, University of Adelaide).

outcomes especially those undergoing FESS or patients undertaking cross sectional examination to assess for facial bone trauma and or paranasal sinus disease infiltration. It is therefore important that radiologists are aware of this anatomical variant and make mention of its presence or absence in their reports. Hence, a review of radiological observations and classification of LPD in addition to review of the medial orbital wall anatomy and surgical implications of LPD are discussed.

Lamina papyracea ingression refers to lamina papyracea dehiscence with herniated orbital content [5,6]. The LPD refers to the osseous discontinuity in the medial orbital wall [8]. This can be arc or slit-shaped in CT imaging [7-9]. The medial orbital ingression always contains fat [3]. Some authors have categorized the degree of ingression by measuring the depth of herniation relative to the normal distance between the perpendicular plate of the ethmoid and the expected location of the normal medial orbital wall [10]. Accordingly, the ingression has been classified into three grades. Ingressions encroaching less than one third of the distance from the expected medial orbital wall to nasal septum being labelled grade 1 ingression, those between one and two thirds as grade 2 ingression, and those encroaching greater than two thirds the distance to the nasal septum were classified as grade 3 [10]. On this basis, the ingression in our subject could be classified as grade 1.

Several authors have described the dehiscence of the LP as most commonly occurring anterior to the basal lamella and frequently unilateral [1,6,7,9,11]. This is consistent with our results, as is the previously described association with extraconal orbital fat herniation into the dehiscence [1]. Furthermore, the previously described association with ipsilateral medial rectus hypertrophy [9] is also demonstrated in our subject. Unlike other studies [9], our findings did not demonstrate obvious bowing of the medial rectus muscle in association with the LPD. Surgical studies examining LPD have described a dehiscence in the periorbita along the medial orbital wall with herniation of extraconal fat through this defect but with intact connective tissue separating the ethmoid sinus from the orbital

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#### Figure 3: Image of the left orbit.

The medial wall of the orbit showing anterior (1), middle (2) and posterior (3) ethmoidal foramina seen just above the suture between the orbital plate of the frontal bone (OPF) and the lamina papyracea of the ethmoid bone (LPE). (Obtained from a skull housed in the Anatomy Museum, University of Adelaide).

#### compartment [4].

The medial orbital wall is the most complex and most fragile of the orbital walls. It extends from the anterior lacrimal crest anteriorly to the optic canal posteriorly. The medial wall is approximately five cm in length [12]. It is formed by four bones from anterior to posterior, these are the frontal process of the maxilla, the lacrimal bone, orbital plate of the ethmoid bone (lamina papyracea, LP) and the body of the sphenoid bone (Figure 2). By far the largest contribution to the medial wall is made by the LP. The medial wall separates the orbit from the nasal cavity and the ethmoidal air sinuses. It slopes downwards and laterally as it joins the floor of the orbit, while superiorly it articulates with the roof of the orbit formed predominantly by the orbital plate of the frontal bone (Figure 2).

At the junction of the medial wall and roof of the orbit, several foramina are found along the suture between the ethmoid and frontal bones. The anterior and posterior ethmoidal foramina are the most commonly reported and transmit respectively named nerves and vessels. The anterior and posterior ethmoidal foramina are approximately 24mm and 36mm respectively posterior to the anterior lacrimal crest [12]. A study of 44 South Indian dry skulls found that the anterior ethmoidal foramen was 24-30 mm posterior to the anterior lacrimal crest, while the posterior ethmoidal foramen was 9-13 mm further posterior [13]. The optic canal was located 5-13 mm further behind the posterior ethmoidal foramen [13]. Occasionally a middle ethmoidal foramen may be present (Figure 3). The middle ethmoidal foramen was reported bilaterally in 27% of the skulls examined in a skeletal study [13].

The labyrinth of the ethmoid bone contains multiple air-filled cavities (ethmoidal air cells) which are divided by lamellae into anterior (average 11), middle (average 3) and posterior (average 6) ethmoidal air cells [14]. The most consistent and complete one of these lamellae is the basal (third) lamella [15]. The Lamina Papyracea (LP) forms the lateral wall of the middle and posterior ethmoidal sinuses, while the anterior ethmoidal air cells are completed laterally by the lacrimal bone and frontal process of the maxilla [14]. Since the basal lamella is the only complete lamella that connects the nasal plate to the orbital plate of the ethmoid [15], current practice considers all the air cells anterior to the basal lamella as the anterior group while those posterior to the basal lamella as the posterior group [15].

# **Clinical Relevance**

Lamina papyracea dehiscence may be a congenital or acquired defect. Acquired LPD may be secondary to facial trauma, infection, malignancy or iatrogenic injury during FESS surgery [5]. Furthermore, several case reports have demonstrated that LPD (acquired or congenital) can be an underlying risk factors for orbital cellulitis or even isolated ophthalmological symptoms, such as diplopia and blurred vision [5,16,17]. Therefore, awareness of this uncommon variant will aid clinicians in the diagnosis and management of patients with LPD that present with orbital or sinonasal pathology.

Computerized tomography of the sinuses is an essential step prior to performing (FESS). The CT aids otolaryngologists and radiologists to discern between different sinonasal pathologies, such as chronic rhinosinusitis, allergic fungal rhinosinusitis and unilateral nasal mass, and also to determine the extent of disease. Most importantly, a preoperative CT facilitates more confident preoperative planning and provides radiologists and surgeons with the opportunity to prospectively identify anatomic variants that may predispose patients to major surgical complications. The key structures identified preoperatively, using the CT scan, are commonly considered using the mnemonic CLOSE: Cribriform plate, Lamina papyracea, Onodi cells, Sphenoid sinus pneumatization and (anterior) Ethmoidal artery. These structures are examined carefully as intraoperative injury during FESS can result in devastating complications such as CSF leak, major bleeding or vision loss. When LPD is present, the periosteum of the LP of the medial orbital wall and mucoperiosteum lining the ethmoid sinus are displaced medially into the ethmoid sinus, along with extraconal orbital fat and occasionally the medial rectus muscle. Soft tissue herniation into the ethmoid sinus can be misinterpreted as an ethmoid air cell or accessory sinus and, if not recognized, places the lamina papyracea and orbital structures at risk for intraoperative penetration during ethmoidectomy. Inadvertent violation of intra-orbital structures can lead to haematoma and may result in temporary or permanent visual loss. Furthermore, penetration of the LP establishes a direct communication between the orbit and potentially infected sinus, which may predispose to the development of orbital infections and its sequelae. Therefore, awareness of LPD and its anatomical variants provides critical information to surgeons prior to FESS in order to reduce the risk of major intraoperative complications [18].

Other studies have described an increasing incidence of LPD with age and an overall prevalence rate much higher [11] compared to the paediatric population we examined. The possibility of acquired factors predisposing to this entity has therefore been raised and discussed [11]. Given our study was confined to the paediatric population (aimed at assessing congenital frequency) this may partly explain why our incidence is lower than those published from other studies.

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