

Aplasia Cutis Congenita Associated with Duodenal Atresia: A Marker of Underlying Congenital Abnormalities: A Case Report

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ABSTRACT

Introduction: Aplasia cutis congenita (ACC) is an uncommon congenital skin defect characterized by the absence of skin, commonly in the scalp. ACC has been found to be accompanied by certain embryological malformations, so it may indicate underlying dysplasia. This case report emphasizes that ACC is not only a superficial finding but also serves as an indicator of underlying abnormalities. **Case:** A premature female neonate was born via spontaneous delivery with respiratory distress. There was an absence of the epidermis in the region of colli, bilateral antebrachia to dorsum manus, and bilateral distal femoral to dorsum pedis with firm borders, visible blood vessels, underlying muscles, and agenesis of the ears and nose. On the third day, the patient vomited blood, and the babygram examination showed a double bubble appearance. The patient was diagnosed with ACC and duodenal atresia. The patient was treated with D10% infusion, cefotaxime injection, parenteral amino acid, oral sucralfate, and gentamicin ointment. While preparing for referral, the patient's condition deteriorated, and the patient died on the 7th day of treatment. **Discussion:** ACC is characterized by the absence of skin and underlying tissues, and its classification is not always clearly defined. Developmental anomalies of the skin and subcutaneous tissue, such as ACC, can indicate serious underlying conditions. Prognosis depends on the severity of other genetic disorders. In this case, definitive surgical management of the duodenal atresia was not performed because the patient's clinical condition deteriorated before the intervention could be undertaken. **Conclusion:** This unusual combination highlights the multisystem nature of ACC and the challenges of applying Frieden's classification. Extensive or atypical ACC warrants thorough evaluation for associated congenital abnormalities to guide management and prognosis.

Keywords: Aplasia cutis congenita, case report, congenital skin abnormality, duodenal atresia, skin.



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INTRODUCTION

Aplasia cutis congenita (ACC) is a congenital skin defect that occurs in 3 out of 10,000 births, characterized by a confined absence of skin, commonly on the scalp.¹⁻⁶ ACC has no single cause and is thought to be the end result of different pathological processes, one of which is incomplete closure of the neural tube.^{1,2} ACC has been found overlying certain embryological malformations, so it may also signify underlying dysplasia.³ Developmental anomalies of the skin and subcutaneous tissues can indicate potentially serious underlying conditions. Underlying tissue involvement in ACC includes limb or skull defects and organ involvement, such as the central nervous system, cardiovascular system, and gastrointestinal system.⁷ In

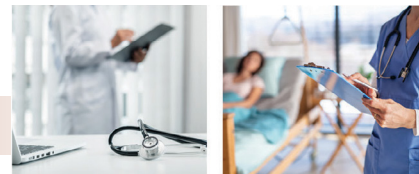
1986, Frieden devised an ACC classification system into nine types based on the location and the pattern of skin absence, underlying causes, presence of associated deformities, the association with other types of skin pathology, and mode of inheritance.^{2,5-13}

We report a newborn with extensive and multifocal ACC involving the neck, both forearms, and both lower extremities, accompanied by multiple congenital anomalies, including external ear and nasal agenesis and duodenal atresia. The combination of extensive, multifocal cutaneous defects with craniofacial and gastrointestinal malformations represents an unusual clinical presentation and may not be readily accommodated within a single

category of Frieden's classification. This case therefore highlights the potential complexity and multisystem nature of ACC, particularly when the lesions are extensive and occur at multiple anatomical sites.

The novelty of this case lies in the coexistence of extensive and multifocal ACC with both craniofacial agenesis and duodenal atresia, emphasizing that the extent and distribution of cutaneous defects may provide an important clue to associated developmental abnormalities. This case contributes to the clinical understanding of ACC by demonstrating the importance of considering underlying multisystem congenital anomalies in newborns presenting with extensive or atypical ACC. Recognition of ACC as a

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potential marker of developmental dysplasia may facilitate comprehensive evaluation and earlier identification of associated internal abnormalities.

CASE

A female neonate was delivered prematurely at 34 weeks of gestation via spontaneous vaginal delivery at a medical facility. The infant's birth weight was 1,660 grams, with a body length of 39 cm, head circumference of 30 cm, and chest circumference of 28 cm. The APGAR scores were recorded as 8–9–10. Initial vital signs included a heart rate of 128 bpm and a temperature of 36°C. The neonate exhibited signs of respiratory distress, with an oxygen saturation of 87%, accompanied by visible suprasternal and epigastric retractions. There is an absence of epidermis with well-defined borders in the colli region, bilateral antebrachia to dorsum manus, and bilateral distal femoral to dorsum pedis, with visible blood vessels and muscles underneath. The patient also had agenesis of the ears and nose (**Figure 1**).

The neonate was delivered to a 27-year-old P2A0 (*para 2, abortus 0*) mother. Tests for HIV,



*Photo documentation by Lidya Renaningtyas Setia Budi.

Figure 1. Clinical findings. Absence of epidermis in the colli region, bilateral antebrachia to dorsum manus, and bilateral distal femoral to dorsum pedis with clear boundaries, revealing the underlying blood vessels and muscles. The patient also had agenesis of the ears and nose.

syphilis, and HBsAg were negative. During pregnancy, the mother had no trauma or infectious diseases. Ultrasound examination was not done regularly; no ultrasound results were available. The drugs taken during pregnancy were ferrous sulfate, calcium lactate, and vitamin B6 obtained from the health center. Neither side of the family had experienced a similar illness.

Because of respiratory distress, the patient was sent to the high-risk perinatology unit and continuous positive airway pressure was initiated. On the 3rd day, the patient developed profuse hematemesis, which was subsequently evaluated with a babygram. Babygram results favor respiratory distress syndrome (dd/ hyaline membrane disease); prominent gastric air appears with a picture resembling double bubble (dd/ duodenal atresia), and necrotizing enterocolitis cannot be ruled out (**Figure 2**). Complete blood laboratory results were within normal limits, except for elevated total bilirubin of 15.03 mg/dL, with direct bilirubin of 0.42 mg/dL and indirect bilirubin of 14.61 mg/dL.

The patient was diagnosed with low birth



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Figure 2. Babygram result. Gas shadow shows a double-bubble sign, which is seen as a large radiolucent stomach and a smaller, more distal bubble to the right of the midline shows a dilated duodenum, indicating Duodenal Atresia.

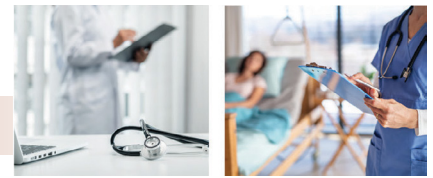
weight, premature neonate, respiratory distress, disseminated intravascular coagulation, necrotizing enterocolitis, duodenal atresia, and aplasia cutis congenita. The patient received D10% at 10 drops per minute via microdrip infusion, cefotaxime 3 x 75 mg, parenteral amino acids 50 mL/day, and gastric lavage with sucralfate 4 x 1 mL. A phototherapy plan to correct bilirubin was postponed due to the patient's poor general condition. For topical care, the patient received with NaCl 0.9% and gentamicin ointment three times a day.

Following the babygram findings suggestive of duodenal atresia on the 3rd day of hospitalization, the patient was promptly referred to a type A hospital through the Sistem Rujukan Terintegrasi for further management by a pediatric surgeon, as this specialist was not available at the referring hospital. While waiting for the type A hospital's approval, the patient's condition deteriorated. On day 6, the patient developed septic shock and progressed to terminal shock, resulting in cardiac arrest, after which spontaneous circulation was briefly restored. The parents declined further intensive care and resuscitation; the patient died on day 7 of treatment.

DISCUSSION

Aplasia cutis congenita (ACC) is an uncommon congenital skin defect characterized by a confined absence of skin accompanied by scarring alopecia. Unlike other neonatal blistering disorders, ACC affects the epidermis, dermis, and subcutaneous tissue. The scalp is the most commonly affected area, but there is potential for involvement of other areas.^{2–5,12}

ACC is not attributable to a solitary underlying cause, but rather seems to be the culmination of a series of discrete pathological processes. It simply represents a physical finding that reflects an abnormality in intrauterine skin development. The following theories have been posited to explain its development: neural tube defect; skin vascular insufficiency resulting from placental insufficiency; presence of infections within the uterus; usage of teratogenic agents; amniotic bands; and presence of chromosomal abnormalities.^{1,3,8,12,14} When examining



patients with ACC, it is crucial to inquire about the presence of ACC or limb defects in their family members.³

Compression of growing tissue, such as that resulting from oligohydramnios, multiple gestations, or macrosomia, may also lead to the development of ACC. An alternative hypothesis posits that premature rupture of the amniotic membranes may cause the amniotic membranes and/or the placenta to adhere to the fetus, thereby detaching the normal skin. These bands may disrupt the tissue's normal growth process. ACC has been found to overlie certain embryological malformations. It may also indicate underlying dysplasia, such as non-ossified bones, changes to the normal brain structure, or the central nervous system in cases involving the scalp.³

The number of cases reported in the literature to date is about 500 cases.^{6,10,15} The incidence of ACC is estimated to be approximately 3 out of 10,000 births.^{5,6,9,15} The mortality risk is significant, with a mortality rate as high as 18%.⁹ This condition is frequently underreported, particularly in cases where mild lesions are present in newborns.^{5,15}

Clinical manifestations are the primary basis for diagnosing ACC.⁹ Most cases of ACC are identified and treated during the newborn period.² The hallmark of ACC is the complete or partial absence of epidermis, dermis, and, occasionally, underlying tissues at the time of birth.^{6–8} The underlying tissue involvement may also be associated with underlying malformations including limb or skull defects, lumbosacral sinus tract, faun tail nevus and spinal cord malformation, myelomeningocele, porencephaly, spinal dysraphism, gastroschisis, omphalocele, glaucoma, ear malformations, cleft lip and palate, absence of the nasal septum, laryngomalacia, choanal atresia, congenital heart diseases, umbilical hernia, renal anomalies, ambiguous genitalia and cryptorchidism.^{2,7,10} Developmental anomalies of the skin and subcutaneous tissues, such as congenital aplasia cutis, can indicate potentially serious underlying conditions.¹

ACC most commonly presents as an oval, clearly defined margins, atrophic macule on the midline of the posterior scalp. They range in

size from tiny patches to covering around 90% of the body. The initial lesion may be raw and oozing. Eventually, it will evolve into erosion and ulceration with either a granulating base or a thin, shiny covering.^{1,11,16} Aplasia cutis is consistently hairless. Most lesions are known to re-epithelized spontaneously over several months, leaving hypertrophic or atrophic scars.^{1,5,12} ACC lesions most commonly occur on the scalp (70%), but they can also appear on any part of the body, including the face, trunk, and limbs.^{6,8} Multiple skin involvement in ACC is rare. One study reported three cases of multiple skin lesions out of 90 cases, while another reported none out of 22. Most ACC cases present as isolated skin lesions.⁷ ACC may be an isolated anomaly or part of a syndrome, and its occurrence may be familial or sporadic.^{2,7,16,17} In our case, there was an absence of epidermis in the colli region, bilateral antebrachia to dorsum manus, and bilateral distal femoral to dorsum pedis with clear boundaries, revealing the underlying blood vessels and muscles. The patient also had agenesis of the ears and nose.

An important role is played by the skin of a newborn in the transition from the intrauterine to the extrauterine environment. The skin of a newborn is more susceptible to injury due to trauma and infection than the skin of an adult, so it requires appropriate care. In newborns, the functions of the skin include thermoregulation, providing a barrier against toxins and infections, excreting water and electrolytes, storing fat and perceiving tactile sensation.¹⁸

After birth, a clinical diagnosis of ACC is made. To identify any potential anomalies, it is essential to perform a series of diagnostic procedures, including standard radiographs, ultrasonography, echocardiography, and chromosome analysis. Prenatal ultrasonography can detect ACC.² The histological characteristics of ACC exhibit variability depending on the depth of the lesions. A dramatic decrease in collagen fibers is also seen, and skin structures like hair follicles, sebaceous glands, and sweat glands are lost. During the healing process, the ulcers are flattened by the proliferation of fibroblasts in the connective tissue stroma. The absence of dermal appendages persists even when ACC is fully resolved.¹⁵ A biopsy

of a midline lesion should only be performed if cranial imaging has been used to rule out an intracranial connection.¹ In our case, Babygram examination revealed a double-bubble image, a classic finding in abdominal radiography indicating duodenal atresia and an enlarged stomach and duodenum.

Frieden proposed a classification of ACC that was based on a clinical review he conducted in 1986. ACC is classified into nine groups based on the location and the pattern of skin absence, underlying causes, presence of associated deformities, the association with other types of skin pathology and mode of inheritance.^{2,5–13} Frieden's classification is widely used, which can be seen in **Table 1**. Evers classified ACC etiologically which is less useful clinically; it includes chromosomal, monogenic exogenous or uncertain etiologies.¹⁶

Based on its location, which includes the pretibial and dorsal areas of the feet and hands, as well as the extensor surfaces of the wrists, this case is classified as type VII. However, based on the presence of other congenital abnormalities, such as duodenal atresia and ear and nose abnormalities, it is classified as type VI. Some authors also reported other cases that don't fit into the classifications. Effendi¹¹ reported a case of type V ACC with an extensive and symmetrical absence of the skin on the scalp, trunk, and limbs without *fetus papyraceus* and placental infarction. Boente reported a similar case,¹¹ and Kaur reported two more in 2016.¹¹ Mesrati reported type V ACC that was asymmetrical and had an underlying bone defect, with no history of *fetus papyraceus*. Mesrati has also reported type II ACC (Adams-Oliver syndrome), in which the lower limbs were more affected than the upper ones.² Frieden himself also implied that some cases of type VII ACC have had EB.¹⁶

ACC is a physical finding that may result from any intrauterine event disrupting skin development. Because of this, not all cases can be neatly categorized.¹⁴ We suggest maintaining Frieden's classification system with modifications and incorporating subtypes where genes have been identified. It remains valid and will continue to evolve.¹⁶

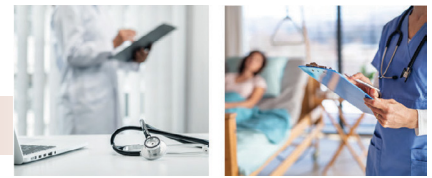


Table 1. Aplasia cutis congenita classification.^{2,6,11,14–16}

Type	Characteristics
I	Scalp ACC without multiple abnormalities. Autosomal dominant or sporadic inheritance
II	Scalp ACC associated with terminal transverse limb abnormalities (Adams-Oliver syndrome), cutis marmorata telangiectatica congenita, cardiac malformations, CNS abnormalities. Autosomal dominant inheritance.
III	Scalp ACC associated with epidermal and organoid nevi, ophthalmic abnormalities, CNS abnormalities. Sporadic inheritance.
IV	ACC overlying embryologic malformations such as cranial malformations, spinal malformations, thoracic malformations, abdominal malformations, and multiple malformations associated with generalized, lethal ACC. This ACC affects scalp, lumbosacral, chest and abdomen. Variable inheritance.
V	ACC associated with fetus papyraceus or placental infarcts or other ischemic events like duodenal, ileal, or biliary atresias with intestinal infraction. Affects any site, mostly symmetrical and linear. Sporadic inheritance.
VI	ACC associated with epidermolysis bullosa, usually on the lower extremities (Bart Syndrome) or large areas of the trunk and extremities. This type is characterized by blisters accompanied by abnormalities of the nails, ears, and nose, as well as pyloric or duodenal atresia and urethral stenosis. The inheritance is autosomal dominant or recessive, depends on the type of epidermolysis bullosa.
VII	ACC localized to the extremities without blistering and without associated abnormalities. This group affects pretibial areas, extensor forearms, dorsal hands and feet. This type is a convenient category for ACC lesions on trunk or limbs which do not fit any other group and where localized intrauterine ischemia or trauma is suspected. The inheritance is autosomal dominant or recessive.
VIII	ACC caused by specific teratogens, maternal conditions or intrauterine infections. This ACC appears mostly in the scalp region, or any site with congenital infections. Not inherited.
IX	ACC associated with malformation syndromes such as trisomy 13 or ectodermal dysplasia, Patau syndrome (Trisomy 13), Wolf-Hirschhorn (4p deletion), Setleis syndrome, Johanson-Blizzard syndrome, Goltz syndrome, ADAM complex, Kabuki syndrome, Delleman syndrome, Finlay-Mark syndrome, XY gonadal dysgenesis. Variable inheritance.

Decisions of ACC treatment must be individualized according to the width, depth, location, presence or absence of underlying cranial defects, and associated complications.^{6–9,19} A lack of a lipid layer in the skin's structure can cause increased water loss through the skin, poor temperature regulation, and electrolyte imbalances, most notably hypernatraemia.²⁰ A conservative approach is recommended, allowing for the gradual re-epithelization of the skin and the formation of a hairless, atrophic scar over the course of several weeks. Large or multiple defects may require surgical closure to prevent the risk of fatal complications.^{8,19} Skin grafting is a crucial procedure for patients with extensive skin defects. However, it can exacerbate the patient's discomfort and suffering.⁹

Conservative treatments are recommended if the defect is limited to the skin level, no bone damage has occurred, and the area is less than 3 cm².⁹ Conservative management included regular wound cleansing, local therapy, application of an occlusive dressing, and systemic antibiotics.^{6,19} This includes physiological saline, continuous saline

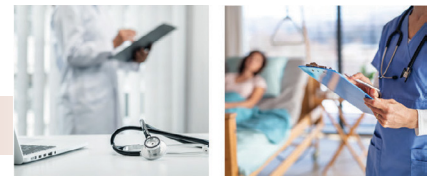
drips, betadine solution, bacitracin ointment, moist exposed burn ointment (MEBO), and silver sulfadiazine 1%, nitrofurazone 0.2%, which are employed to conserve moisture, avert desiccation, and enable spontaneous epithelialization to occur until secondary-intention healing is fully accomplished.^{6,7,9,19}

Surgical treatment is necessary when critical organs or tissues are involved and the affected area exceeds 3 cm².^{7,9,19} There were several approaches used for surgical management, including excision and primary wound closure, split-thickness or full-thickness skin grafts, acellular dermal matrices, autologous epithelial transplantation, local scalp flaps, free flaps, muscle flaps, and cranial vault reconstruction using bone grafts.^{6,7,9,19}

Nevertheless, in most cases of ACC, closure using a single large scalp flap is considered sufficient and effective.⁶ In conclusion, the selection of an appropriate surgical technique should be individualized, guided by the characteristics of the lesion, condition of the patient, and the surgeon's expertise and preference.⁶ Surgery, regardless of the chosen method, the fundamental objective

remains the closure of the skin defect. As with any operative procedure, surgery for ACC carries the risk of complications, most notably intraoperative hemorrhage and postoperative infection.⁶ In our case, the patient received gentamicin ointment. This was preceded by washing the area with 0.9% NaCl gauze. Due to the absence of a pediatric surgeon, the patient was referred to a type A hospital through the Sistem Rujukan Terintegrasi for treatment of duodenal atresia.

One of the most common congenital anomalies in newborn babies is intestinal obstruction. Causes of small bowel obstruction in newborn babies include atresia, malrotation, Hirschsprung's disease, and other rare causes such as congenital bands and obstructed hernias. Although neonatal intestinal obstruction is relatively uncommon, it accounts for most neonatal surgeries. Intestinal atresia is one of the most common causes of intestinal obstruction in newborn babies. Duodenal atresia is the most common type of atresia, followed by jejunoileal atresia.²¹ Duodenal atresia is the most prevalent congenital gastrointestinal obstruction.²² It has been linked to in utero



polyhydramnios and stands as one of the most prevalent etiologies of fetal bowel obstruction.²³ Duodenal atresia occurs in 1 of 5,000–10,000 live births.^{22,23} Duodenal atresia is more likely to be caused by developmental defects in intestinal organogenesis that result from the disruption of various signaling pathways, such as the fibroblast growth factor, bone morphogenic protein, and β -catenin pathways.¹⁷ The primary mechanism leading to duodenal atresia occurs during the eighth to tenth week of embryonic development, when abnormal duodenal recanalization occurs. This is characterized by a complete blockage of the duodenal lumen.²³

Two key clinical findings of duodenal atresia are abdominal distention and bilious vomiting within 24 to 38 hours of life after feeding. If this intestinal obstruction is found, urgent operative intervention may be needed.^{23,24} The use of prenatal ultrasound and MRI will show a double bubble and can predict the diagnosis of duodenal atresia earlier.^{21–25} Duodenal atresia has been linked to trisomy 21 (Down syndrome) and cardiac malformations, annular pancreas, malrotation, anorectal atresia, and genitourinary anomalies. Therefore, if a prenatal diagnosis of Duodenal atresia is made, a detailed anatomic scan must be performed to identify any associated anomalies.^{23,25} The initial postnatal radiographic evaluation for the diagnosis of duodenal atresia is a plain abdominal x-ray. A plain abdominal x-ray may reveal the gas shadow as a double-bubble sign, which is seen as a large radiolucent (air-filled) stomach and a smaller, more distal bubble to the right of the midline, which represents a dilated duodenum.^{23,24} Duodenal atresia patients require urgent treatment due to a closed obstruction of the stomach and proximal duodenum.¹⁷ If the patient is stable, a staged approach should be used to reduce morbidities and improve survival.^{17,23,26}

Treatment consists of decompression of the stomach that can be done by orogastric or percutaneous radiologic gastrostomy placement, and the definitive repair of the obstruction lesion can then be performed at a later point in time.^{17,23,26} The decompression of the stomach is necessary to obtain gastric detention and respiratory stabilization, at this stage intravenous fluid resuscitation is also performed.^{23,26} Duodenoduodenostomy is the customary surgical intervention, which can be executed either through an open or laparoscopic approach.^{23,24} The mortality rates are not directly related to the surgery itself, but rather to the presence of other organ anomalies, such as complex congenital cardiac defects.²³

The prognosis of ACC varies depending on the underlying genetic abnormality.¹³ Simple defects without complications make up about 85% of ACC cases, and they have an excellent prognosis.^{8,9,19} However, if ACC is associated with other abnormalities, the mortality rate can reach up to 50%.^{8,13,19} Mortality is correlated with larger lesions and underlying defects. Thus, treatment should focus on preventing these complications.^{8,9} In our case, the patient's duodenal atresia could not be treated. This resulted in sepsis, septic shock, and respiratory failure, which ultimately resulted in the patient's death on the seventh day of treatment.

CONCLUSION

Aplasia cutis congenita is a rare and clinically heterogeneous congenital disorder that may be associated with significant underlying developmental abnormalities. In this case, extensive, multifocal ACC together with external ear and nasal agenesis and duodenal atresia illustrates the potential for multisystem congenital involvement. The unusual combination of cutaneous, craniofacial, and gastrointestinal abnormalities also highlights the challenges in applying Frieden's

classification to complex ACC presentations. Therefore, newborns presenting with extensive, multifocal, or atypical ACC warrant thorough evaluation for associated congenital abnormalities to facilitate appropriate management, prognostic assessment, and multidisciplinary care.

DECLARATIONS

Consent for Publication

Not applicable. This case report does not contain institutional names, personal data, or identifiable images of the patient. Therefore, consent for publication was not required.

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Author Contributions

LRSB, MPSB, and JPRFN contributed to the conception and design of the case report, data collection, literature review, and manuscript drafting. ES and JSB contributed to the clinical management, interpretation of findings, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of Interest

The authors declare no financial or non-financial conflicts of interest related to this case report.

Declaration on the Use of Artificial Intelligence (AI)

AI assisted in preparing this manuscript for language editing, wording refinement, and drafting. The authors reviewed, verified, and approved the entire manuscript and remain fully responsible for its accuracy, originality, and final content.

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