BRIEF COMMUNICATIONS

Atresia Ani and Multiple Vertebral Anomalies in a Squirrel Monkey (Saimiri sciureus)

R. L. Harris, M. G. Bond and B. C. Bullock

Atresia ani, rectovaginal fistula and several vertebral anomalies were found at necropsy in a 10-day-old infant Brazilian squirrel monkey (Saimiri sciureus-Letecia, Colombia). Death resulted from intestinal obstruction. Atresia ani is thought to be hereditary in several species, including man. The cause of the anomalies was not determined. There are no congenital malformations in known relatives of this monkey.

At necropsy the monkey weighed 99 grams. The umbilicus was healed. The monkey had no anal opening (fig. 1). It had a short, crooked tail, 4 centimeters long (fig. 2) and abdominal distension. The genitalia were normal; intestines were distended with ingesta (fig. 1). The mucosa of the descending colon and rectum was hyperemic. The submucosa contained multiple foci of acute inflammatory cells and the muscularis externa was edematous. The inner circular layer of the muscularis externa had degenerative changes. A small rectovaginal fistula was demonstrated when dye was injected into the terminal colon. The small rectovaginal fistula was not seen grossly or microscopically.

The distended gastrointestinal tract elevated the diaphragm bilaterally, and parts of both lungs were atelectatic. There was no evidence of vomition or aspiration of gastric contents. The abnormally short tail had two obvious deformities. Radiographic findings showed 13 ribs on the left side; each rib articulated with the thoracic vertebra of the same number. There were 14 ribs on the right side. The right sixth and tenth ribs articulated with extraveebral ossification centers (fig. 2). Thus, the 13th thoracic vertebra had no attached right rib. There were eight lumbar vertebrae instead of the normal seven. The fifth lumbar vertebra was wedge shaped, with a separate, extraossification center caudal to the vertebral body. There were only eight caudal vertebrae. The fifth and eighth caudal vertebrae had angular deformities. The normal Saimiri vertebral formula is C7, T13, L5, S3, Cd6 [7].

Anal atresia is the result of faulty migration of the urorectal septum [1]. Fistulas from the rectum to various parts of the genitourinary tract, such as rectovaginal fistulas, are also the result of abnormal urorectal septum migration and are often associated with atresia ani. Various classifications of human anorectal abnormalities have been proposed [16]. The “low” rectovaginal fistula occurs when the bowel passes through the puborectalis sling. A “high” type occurs when the bowel does not penetrate the structure. A puborectalis muscle was not seen in our monkey, but the deformity seemed to approximate a “high” lesion of the human type—the rectum terminated within the pelvic canal.

In man, the incidence of atresia ani is 1 in 5000 live births [18]. A recessive mode of inheritance is suggested by the distribution of anal anomalies in eight of 11 families with more than one affected sibling [20]. Most anorectal anomalies of man are considered chance occurrences. Environmental influences have been incriminated [6, 9, 11]. Colonic atresia and
Fig. 1: Perineum at necropsy. Absence of anal opening. Opened abdomen and abdominal wall. Distended colon and tail deformities.

Fig. 2: Radiograph of infant squirrel monkey skeleton. Axial skeletal deformities. Thirteen ribs on left, 14 on right. Sixth and tenth ribs articulate with extraossification centers (arrows).
absence of a tail have been reported in a male rhesus monkey (Macaca mulatta) fetus whose mother received the anti-convulsant drug trimethadione [17].

The mode of inheritance in swine is uncertain, but seems to involve more than one pair of genes. A frequency of atresia ani as high as 35 in 553 births has been reported [13]. A similar high incidence (64/4417) has been reported in lambs [2].

Anorectal anomalies appear spontaneously in mice with the “disorganization” mutation [8] and in mice with the “fused” gene [3]. The occurrence of atresia ani in an inbred herd of bison (Bison bison) is suggestive of a genetic cause in this species [12]. Atresia ani has been described in dogs but heritability has not been investigated [15].

The association of atresia ani with other anomalies is common. Various authors report different frequencies, but in surveys of autopsied cases, all human infants with atresia ani have additional anomalies [19]. Atresia ani is a component of several recognized human syndromes, including the syndrome of imperforate anus, vertebral anomalies and polydactyly [4, 10], the VATER syndrome [14], and the “cat-eye” syndrome [5]. Imperforate syndrome occurs in mice with the “fused” mutation, a syndrome of vertebral anomalies [3].

The vertebral anomalies in our monkey probably would not have caused death. The thoracolumbar lesions would have gone unnoticed had caudal vertebral deformities not been present. Although the tail of the squirrel monkey is not prehensile, the newborn squirrel monkey can support its body weight with its tail, and the tail is used to hold on to the mother. Intestinal obstruction due to the atretic anus is the probable cause of death. From the information available it is impossible to rule out genetic causes, environmental causes or chance occurrence.

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**References**

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Request reprints from Robert L. Harris, DVM, Department of Comparative Medicine, Bowman Gray School of Medicine, 300 S. Hawthorne Road, Winston-Salem, NC 27103 (USA).