SUBTLE BUT DEVASTATING CAUSES OF FAILURE TO THRIVE

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ABSTRACT

'Failure to thrive' is a common presenting problem. It may affect any age and any species but is seen more often in the young. The common causes of this condition are often recognised and managed accordingly. There are occasions when, despite the correction of diet, stress, parasites and other causes, the problem persists and may be difficult to diagnose. This paper presents a number of case studies that illustrate some of the less common causes of 'failure to thrive'. We must be aware that such uncommon conditions exist.

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FAILURE TO THRIVE

This condition implies that the patient has not been growing at the expected and predicted rate consistent with their age, species, gender and other parameters. It is a commonly seen condition that often responds to simple alterations in management. The common causes of failure to thrive are,

Inadequate dietary input most nutrients
Stress acute and/or chronic
Parasitism internal and/or external
Chronic illnesses diarrhoea, renal disease
Congenital deformities such as cardiac conditions,
gastrointestinal malformations
metabolic aberrations
renal abnormalities
competition with colleagues/other species
toxicities

A thorough history and some basic investigations will often provide a diagnosis and consequent therapeutic change in management. If the problem persists following appropriate therapy and opportunity for slowly resolving problems to resolve then further investigation may be necessary.

This paper outlines a number of cases in which patients continued not to thrive and their management required further interference.

CASE 1 EGK JOEY 'B'

B was orphaned when mother was killed by a motor vehicle collision. She suffered several limb fractures at the time and these were managed appropriately. Initially she responded in the expected manner of eventually starting to accept oral bottle feeding and seemed to make reasonable progress. Body weight increased from 650 gm to 1.1 kg. The very adept carer felt that over the following few weeks there was less progress than was expected and that the joey became less enthusiastic about feeding, and weight gain was slower than usual. There was no definite abnormality detectable on clinical examination. Cardinal signs were normal, mucosal colour was normal, heart and lungs were clear, abdomen was non tender with normal bowel sounds and faeces that was microscopically negative for ova,

cysts and parasites. Urinalysis was normal. Various changes were made to diet included some nutritional supplements. The joey remained bright and alert until several weeks later when the situation started to deteriorate. Diarrhoea occurred and that was corrected by conservative management. She suffered urinary incontinence in her pouch. She was treated with antibiotics for a urinary tract infection and the incontinence settled. The incontinence became recurrent and she then suffered a small prolapse of the bowel. That was reduced successfully. On examination at that time she was 1.7 kg. The only other abnormality was a vague discomfort on abdominal palpation. In the absence of being able to obtain any further investigations it was decided to perform an exploratory laparotomy.

Standard general anaesthesia was given with endotracheal intubation, spontaneous ventilation with supplemental IPPV, isoflurane plus oxygen, intravenous fluid therapy and monitoring of BP, SpO2 (oxygen saturation), ventilation rate and temperature.

A midline laparotomy was performed which allowed visualization of the following. The gall bladder was grossly enlarged with bile. High in the dorsal abdomen there were adhesions that constricted the common bile duct, pylorus and small intestine. The pancreas was scarred and was attached by adhesions to the other organs. Some adhesions were divided to allow decompression of the intestinal lumen and the common bile duct. The gall bladder was drained and a drainage tube placed to exit the abdominal wall lateral to the primary incision. Closure was routine. Fluid therapy, antibiotics and analgesia were given with postoperative monitoring and temperature control.

B did not survive and post mortem examination confirmed the above intraoperative findings that were obviously not of recent origin. The interpretation was that the initial motor vehicle trauma had incited the process that had then progressed slowly because of the ability of the body to partly contain the progression. Eventually the clinical effects of the advancing pathology became apparent and laparotomy was performed.

CASE 2 EGK JOEY C

C was orphaned by a motor vehicle and subsequently hand reared. She was initially difficult to feed, but made adequate progress from 800 gm. The astute carer felt that her growth was not optimal. At a body weight of about 1.5 kg, her progress slowed and gradually became of greater concern as she was ‘failing to thrive’. She suffered from

malodorous diarrhoea, but there were no other abnormal clinical signs on examination. Faecal and urine testing revealed no abnormalities. Symptomatic treatment was given with minimal improvement. Eventually C became more unwell showing early signs of bowel obstruction. At that stage it was noted that she was uncomfortable during abdominal palpation and a vague, minimally tender mass was palpable in her abdomen. It was decided to perform an exploratory laparotomy.

C was anaesthetised using appropriate premedication, volatile anaesthetic induction, endotracheal intubation, intravenous cannulation and infusion, full monitoring and supplemental IPPV.

A midline incision was used to gain maximal access to the peritoneal cavity. A large dorsal mass of adhesions was found attached to the dorsal abdominal wall. The mass incorporated the common bile duct, pylorus of the stomach, small intestine and large intestine. On separation of some of the superficial adhesions a tear in the large intestine was noted. There was fibrosis surrounding the tear and the adjacent tissue was very fragile. There was a perinephric abscess contained within a mass of omental tissue. Attempts to divide further adhesions provoked areas of minor haemorrhage and were ceased. The bowel rupture was repaired, peritoneal cavity cleaned and irrigated and the abdominal wall closed in a standard manner.

C was transferred to intensive care where she was treated with intravenous fluids, antibiotics, analgesia, temperature control and general nursing. She later died. Post mortem examination revealed the extent of the above findings and indicated that the pathology had been developing for some time.

CASE 3 WOMBAT M

M was 10 kg body weight with a history of abdominal bloating for several weeks. She was losing weight and still attempting to eat and drink. Various medical treatments were given with no apparent improvement. Eventually her condition began to deteriorate rapidly and she presented in a fairly advanced state of illness.

On examination she was depressed, disinclined to move or lie down, dehydrated, afebrile, tachycardic with shallow rapid ventilation, pale mucous membranes, a tender bloated abdomen with depressed bowel sounds and unkempt. A diagnosis was made, of peritonitis with

toxaemia/septicaemia and a decision to perform a laparotomy in case there was repairable pathology.

Intravenous fluids, oxygen and analgesia were given as a priority and then M anaesthetised with a standard volatile induction, endotracheal intubation, IPPV supplemented spontaneous ventilation, temperature control and full monitoring.

A standard midline operative approach was used to open the abdomen. On opening the peritoneal cavity there was a rush of putrid gas and faecal content from the incision indicating that there was a ruptured bowel. Further investigating revealed a large tear in the bowel and severe and extensive peritonitis that had been developing for some time. It was decided that M could not recover from such severe and advanced pathology and treatment was suspended.

CASE 4 EGK JOEY BM

BM was orphaned by a motor vehicle. He was 2.5 kg body weight and appeared to be unscathed. His hand rearing began reasonably well but within a few weeks it was apparent that he was failing to thrive.

On examination he appeared bright and alert but less robust than expected. He was well hydrated but underweight. He was afebrile with a clear chest and normal sounding heart. His abdomen appeared asymmetrical and was mildly tender to palpation. Bowel sounds were present but depressed. A soft, vaguely defined, mobile mass was palpable on the right side of the abdomen. A plain radiograph confirmed the presence of loops of bowel in the mass.

General anaesthesia was performed with appropriate premedication, volatile induction, endotracheal intubation, intravenous infusion, IPPV supplemented spontaneous ventilation and full monitoring.

A midline incision was used to allow maximal surveillance of the abdominal wall. A 2cm tear was found on the right lateral wall of the abdomen with extrusion of several loops of small intestine into the extraperitoneal/ subcutaneous plane. After ensuring that the intestine was intact it was replaced into the peritoneal cavity and the defect repaired in a standard manner.

The postoperative course was uneventful and BM made a full recovery.

These case studies illustrate that ‘failure to thrive’ is sometimes caused by unusual, unexpected and difficult to diagnose pathology. The history is of prime importance and a thorough clinical examination necessary to be alerted to subtle signs indicating a more sinister underlying pathology than the common causes of the syndrome.

As wildlife carers we need to remain vigilant to the possibility of significant injury sustained in a motor vehicle accident. Fractured limbs, head injury, lacerations, chest injury and damage to eyes are common and detectable by early clinical examination. Deep injuries such as those described may remain concealed for a considerable time as the bodily defences mobilize to contain them. Wildlife patients appear, in general, to tolerate some injuries such that although they may feel discomfort or pain, they try to mask external signs of internal derangement until a late stage in the progression of the pathology.

Early detection of such problems, following motor vehicle trauma, may be enhanced by more routine and repeated clinical examination, having a high index of suspicion when there are only minimal clinical signs of trouble, keeping accurate records of routine care and body weights, more use of radiography and particularly ultrasound examination.

Obviously the pathology causing devastating problems in the patients discussed above, is much more easily and successfully treatable if discovered early rather than at a late stage.